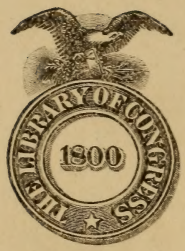

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MEDICAL DIAGNOSIS

GREENE

MEDICAL DIAGNOSIS

FOR THE

STUDENT AND PRACTITIONER

BY

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ST. PAUL

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PREFACE TO THE FIFTH EDITION

The author regrets that the obligations of military service in the Great War and the concentration upon private affairs necessarily following it have delayed the issuance of this volume.

He has made full use, however, of the opportunity afforded for a deliberate, painstaking and thorough revision.

Every section has been expanded by important additions and no effort spared to make the present volume a thoroughly practical and up to date treatise upon modern medical diagnosis. The rise of diseases of the heart and blood-vessels to first place on the list of causes of premature death makes imperatively necessary a better understanding of the now unrealized possibilities along the lines of their early recognition, prevention, and retardation.

The author has endeavored, therefore, to further emphasize in this volume the means of early and accurate diagnosis now available to the physician.

This has involved not only an amplification of those sections dealing specifically with etiology and clinical technic, but also a more extended reference to and discussion of the adoption of better standards and a more helpful view point.

In the preceding edition considerable space was devoted to the clinical use of instruments of precision in the diagnosis of heart disease, but the great and growing interest in these newer aids to the detection and efficient management of heart lesions has led the author to expand greatly the sections dealing with polygraphic and electrocardiographic technic and interpretation.

Among the many additions to the text, the author would call attention especially to the discussion of "influenza" and the interesting and unique radiograms of Dr. John Hunter Selby, illustrating the development and course of that "hemorrhagic pneumonitis" which gave to the epidemic of 1918 its terrific mortality.

The "Symptom Index" incorporated in this Edition must greatly increase the usefulness of the volume.

The author would express his grateful appreciation of the assistance rendered by his valued associate Dr. Harold E. Richardson, by Dr. Frank S. Bissell, Dr. Wm. Hunter Selby and many others.

He would acknowledge also the courtesy of his publishers and the generous cooperation which has made possible the addition of 105 pages to the text and the introduction of 74 new illustrations at a time when publishing costs have risen to hitherto undreamed of heights.

The author hopes sincerely that this new volume will merit a continuance of the kind reception accorded it through so many years by student and practitioner alike.

CHAS. LYMAN GREENE.

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MEDICAL DIAGNOSIS

CERTAIN FUNDAMENTAL PRINCIPLES AND SPECIAL PROBLEMS

Subclassification and individualization.

Scope of the Term Diagnosis.—Diagnosis means more than merely naming a disease. It demands such accurate subclassification and intelligent, painstaking individualization, as necessitates a knowledge of etiologic factors, the nature and sequence of pathologic changes, the effect of age, occupation, residence, habits, heredity, past ailments, and even of the constitutional peculiarities and personal characteristics of the individual.

Types and variants.

Text-book vs. Bedside.—Accurate diagnosis is prerequisite to accurate prognosis and effective treatment. From text-books and lectures the student learns the known types; at the bedside he studies variation from the type and the "personal equation."

Mental indigestion.

Pedagogic Spoon-feeding.—The older teaching of medicine represented, of necessity, an extreme and *remarkably effective* application of the dogmatic method combined with a sort of pedagogic forced feeding of the student with enormous conglomerates of predigested pap. This resulted in a tremendous overload for the memory, slight opportunity for thoughtful consideration and an exaggerated respect for *ex cathedra* statement.

A boon to the student.

Modern Methods.—The necessity for this system has passed in large measure and, with the lengthening of the courses in medicine, these have taken on the seminar form, and the objective type, to whatever degree the amount of available clinical material of the individual school permits.

With respect to the student, this change has resulted in the replacement of mere feats of memory and unquestioning acceptance of authority, by intelligent individualized study, personal observation and a well-developed critical faculty. Even during his college years or his period of internship he may commence the sifting, sorting, *and necessary forgetting*, which was formerly postponed until the graduate dealt with his own patients in actual practice, a form of post-graduate study too often fraught with initial damage and discomforture to both parties.

The critical faculty.

Honesty and an Open Mind.—*In medicine, even more than in surgery, diagnosis demands a sufficiency of facts, truthfully recorded, intelligently sifted, and viewed without bias or prejudgment.*

Bias and cowardice.

Self-deception, a narrowed mental vision which disregards new facts of later development in the individual case and resists blindly the introduction of new methods in practice, no less than cowardly adherence to an erroneous preconception, are deplorable.

Proper balance important.

Scientific Optimism and Scientific Skepticism.—We of the medical profession have not attained, as yet, that proper balance of scientific optimism and corrective scientific skepticism, no less necessary to the best interest of patient and physician than to medical progress itself.

Many tests proposed.

The civilized world is thronged with eager altruists seeking new medical truths; the result being a vast continuous outflow of honest, but more or less imperfectly established and unproven assertions, together with a small but persistent trickle of valuable facts, which tends constantly to raise the level of scientific fitness and practical achievement. To separate the wholesome grain of truth from the great volume of chaff is a never ending task of no small magnitude.

Few are chosen.

Bricks without Straw.—Within the past few years, for example, many tests have been advocated as of more or less *specific* value in the differential diagnosis of gastric carcinoma; yet up to the present time not one of these has demonstrated its right to any place higher than that of a member of the symptom group and some have fallen short of that.

Dangers of hasty acceptance.

To have accepted any of these tests as *specific* would have worked untold evil through errors of commission and omission alike; to have tried them out without bias would be most creditable to any student or practising physician possessing the necessary facilities and requisite technic.*

The Magic of a Name.—The glamor of a great name is well illustrated by the history of the rise and decline of tuberculin.

Sanguine promises.

Tuberculin.—It required years of disastrous experience to demonstrate the decided limitations with respect both to the diagnostic and therapeutic use of tuberculin, originally announced as a cure for tuberculosis, even in its advanced stages, under a most excessive dosage.†

The use of huge test doses for diagnostic purposes excited violent local and constitutional reactions even in the slumbering and innocuous foci carried by a large proportion of humanity and in many instances lesions, apparently obsolete, awoke to renewed activity.

Over-confidence and over-reaction.

No one who lived and practised medicine during that period can forget the furor which followed the first announcement of Koch; the wild rush of patients and physicians alike, to Berlin; the astounding self-deception of public and profession; the absurd statistics of incredibly rapid cures; and finally, the inevitable *over-reaction,* embodied in the dictum of Nicholas Senn, "away with tuberculin!"

Every medical man knows now that harm resulted from a wholly natural overconfidence in the honest, but mistaken, oversanguine and premature, announcement of a genuine discovery by one of the greatest and most sincere

* Sneyer: Absence of digestive leucocytosis. Watson: Increased alkalinity of the blood. Lang: Increased resistance of the red blood cells to hypo-isotonic salt solution. Brieger and Triebing: Heightened antitryptic index. Neubauer and Fisher: Glycyl-tryptophan test. Salkowski and Kojo: Estimation of colloidal urinary nitrogen. Grafe and Röhmer: Specific hemolysis. Salomon and Saxl: Urinary test, etc., etc., etc.

† The initial therapeutic dose, one thousand times that employed by users of tuberculin today, was carried rapidly upward, despite the stormy local and general reactions excited.

of medical investigators, who, despite this one error has placed the entire civilized world under a debt of gratitude.

Valuable qualities.

"The magic of a great name" is too often only magic in the end and there is no quality more valuable to or in the student, than a decent and respectful attitude of interrogation, inquiry, and independent reasoning.

A source of error.

Conflicting Evidence.—Extraordinary differences between the results of laboratory experiment and those attending their clinical application are of relatively frequent occurrence. What is true in relation to the guinea pig, dog or rabbit is not true *always and necessarily* for mankind and, furthermore, the reaction of the diseased human body may be wholly different from that of the sound one.

It follows that, despite the fact that through the laboratory and the experimental work there done, we have obtained the truths fundamental in the greater part of that new knowledge which is the glory of modern medicine, we cannot achieve results always by mere hasty translation of laboratory experiment into clinical usage.

The best of the many modern illustrations of this fact is found in the relationship of acidosis to periods of starvation.

Acidosis.

In the case of the normal body, starvation means inexorably an increase of ketone bodies in the blood and urine. Resting upon this proven fact, the clinician has carefully avoided starving his diabetic patients.

Now we learn through the brilliant work of Allen that what is true of the normal body may be wholly untrue of the patient suffering from *severe true diabetes mellitus* and that oftentimes the best way to remove diabetic acidosis is to starve the patient for a limited period.

Similarly until within the past few years the *diabetic diet* was made *rigidly* one of proteins and hydrocarbons because of the well established laboratory proof of the inability of the diabetic to burn and store carbohydrates in a normal manner.

By this method of feeding the output of sugar was reduced, but we actually invited the far greater threat of acidosis.

Now, one seeks to combine rationally all of the known facts with relation to protein, fat and carbohydrate metabolism in health and in diabetes alike and patients have a mixed dietary fitted to their caloric necessities and tolerance for a minimal carbohydrate intake.

Limitations Imposed by Extreme Delicacy.—Many genuine discoveries are unpractical or demand such delicacy of technic as renders them of limited use and sources of real danger in unskilled hands.

The opsonic index.

The determination of the *"opsonic index"* affords an example of a procedure so complex and delicate as to place it quite beyond the realm of the practical, yet its introduction was accompanied by an assumption of practical utility which the lapse of time and the test of experience have almost wholly destroyed.

The Wassermann test.

The Wassermann test is now undergoing its trial by ordeal and we are already learning its limitations and better estimating its very genuine value in its proper field; yet so many and so subtle are the sources of technical error,

as to make the test unsafe in the hands of anyone who is not a trained and up-to-date serologist.

The open mind.

The Desirable Attitude.—It behooves the student, physician and teacher alike to be alert and open in mind, prompt to try out new methods, yet slow to accept that which is *merely* new or to replace by it the older methods of known value and potency, until it shall have been thoroughly tested and proven.

The name of the originator may stand for capacity, past achievement, and honesty of purpose, but is not to be considered the hallmark of impeccability.

Mind blindness.

The Reactionary.—On the other hand lies that most abominable of all abominations, the stubborn mind-blindness represented by an extreme skepticism which denies even a hearing to anything representing a radical departure from established theory and practice.

Harvey.

The extent to which this was carried in an age which tolerated the most fantastic theories and lacked even the fundamentals of rational medicine is well illustrated by the antagonism which Harvey encountered, despite his beautiful demonstrations proving the circulation of the blood.

Lister.

A Modern Instance.—Even the epoch-making discovery of Lister failed to secure *general* acceptance and application for years after that great investigator himself and many followers had practically eliminated both "damnable" and "laudable" pus from their surgical wards.

To achieve a proper balance between these two extremes is the duty of every student and practitioner of medicine and to the greatest possible extent he should seek to add his mite to the great and ever increasing treasure of scientific truth.

A Common Source of Diagnostic Error.—To any one who has taught and practised medicine for many years it becomes clearly apparent that a certain incompleteness of conception with relation to pathologic sequence and its resultant symptomatic expressions is accountable for many of our diagnostic shortcomings.

We forget that the usual descriptions of chronic disease apply chiefly to its classical symptomatic expression *as an established and far-advanced pathologic process*, which has been steadily extending and progressing for years prior to its detection.

Possibilities of therapeutic initiative.

Early Diagnosis the Desideratum.—*The true end and aim of modern scientific diagnosis must be the earliest possible recognition of any disease, acute or chronic, and the promptest feasible institution of an effective therapy, whether this consists merely in wise instruction, in a conservative and retardant regimen, adequate supervision and control, or, the immediate and direct use of drugs.*

Fragmentary complexes.

Complete Clinical Picture Often Lacking.—The text-book description of a disease embodies necessarily an enumeration of *all* of the many symptoms which may be encountered in a large group of such diseases. The ailment as encountered in individuals may only in small part fulfill these apparent requirements.

In chronic interstitial nephritis or chronic myocardial degeneration the frank symptoms presented by the far-advanced lesion in a state of gross and

often terminal functional insufficiency are those that chiefly strike the eye and impress the mind of the reader, yet it is not at that stage but in the earlier and lesser decompensatory periods, such as may precede by years the fatal issue or the obvious breakdown, that the physician must find ultimately his greatest usefulness.

Though we cannot detect often their actual beginnings, we can measurably succeed in the diagnosis of relatively early stages of many chronic diseases, if we avail ourselves of the many direct tests now applicable under modern methods of examination and realize the value of imperfect *diagnostic mosaics* composed of fragmentary but sufficiently significant symptom complexes, subjective and objective, combined with an accurate and detailed case history, a thorough knowledge of etiologic factors and a proper understanding and appreciation of the known variants of disease.

Clinical mosaics.

The Distinctive Value of a Knowledge of Etiologic Factors.—A study of the causes of disease is valuable in relation to chronic, no less than acute ailments and certain constitutional peculiarities of structure and temperament, no less than actual antecedent illness are oftentimes of the utmost significance in relation to symptoms otherwise obscure.

Affect diagnosis, prognosis, and treatment.

"Chronic congenital asthenia" is of the utmost value, as a constitutional defect known to underlie many conditions of great importance. The history of certain past acute ailments is of extraordinary suggestiveness in many instances.

Example.—Knowing that at least 50 per cent. of even single attacks of acute rheumatism in childhood result in permanent damage to the heart, a history of such an attack becomes peculiarly suggestive in the presence of any suspicious but indeterminate cardiac symptoms or signs, and, in little children, may represent in such instances nothing more than a mother's account of "sore throats" and "growing pains," the peculiar significance of which must be known and appreciated.*

Masked rheumatism.

A knowledge of past luetic infection, the most protean and potent of common etiologic factors, at once throws special emphasis upon certain lines of investigation or offers a probable explanation of the presence of obscure and ill-coördinated nervous symptoms, a mysterious recurrent headache, or vague and indeterminate indications of pressure at the base of the brain.

Syphilis.

Poll Parrotry.—*The student should not memorize symptoms without proper consideration of their relation to the known specific and peculiar pathologic changes of the individual disease: rather let him learn primarily the* **general symptom groups,** *common to the many forms of "fever" "anemia," and the like, and then associate with the individual disease that which is "peculiar" and specific in its symptomatology and, above everything, the distinctive pathologic changes which underlie it.*

Useless overload.

Learn basic symptoms.

A multitude of diseases are "*febrile*" and there are hosts of "*anemic*" patients of various types, but all fevers and anemias have a basic general symptomatology. This last he should learn thoroughly as of broad appli-

* The younger children are peculiarly likely to show nothing save a little tenderness about one or more joints, following, after *several days*, "a sore throat." A vast number of such cases are wholly overlooked.

Differentiating symptoms.

cation, but stamp especially upon his memory the *peculiarities* or definite symptomatic variations that any specific ailment of that group presents.

Value of picture.

Mental Photography.—*Furthermore, every student should try to get a clear mental photograph of any ailment he is studying, and of the pathologic changes that underlie and explain its symptoms.*

In his mind's eye as he reads he should *see* the man with typhoid, the fever chart, his physiognomy, decubitus, rose spots, and more than that, the intestinal ulcers that underlie them. Such a method makes for quickness of perception and thoroughness alike.

Diagnosis by exclusion.

Direct vs. Indirect Methods.— Diagnosis by exclusion is a useful and valuable though roundabout method of arriving at conclusions by a process of negation; the object being to find in the signs or symptoms presented by a given case, one or more inconsistent with the diagnostic symptom group of all diseases save one. Modern advance has greatly reduced the necessity for the method and added enormously to its difficulty and laboriousness.

Usual method.

Diagnosis by deduction based upon our knowledge of the pathologic processes and sequences of disease together with the fullest obtainable symptomatic and contributory data, is the method in common use, and exclusion becomes merely a factor, not a formal procedure.

Therapeutic Diagnosis.—A therapeutic diagnosis is sometimes necessary and valuable, as, for example, in obscure syphilitic infection when the effect of mercury, the iodides, or salvarsan may go far to banish doubt.

Of great value.

In the recognition of the damaging, earlier, and lesser, cardiovascular insufficiencies, digitalis is invaluable.

"Snap" Diagnoses.—Certain single symptoms may name a disease and such are termed pathognomonic, but woe to the man who is betrayed into the *habit* of making *"snap" diagnoses* based upon some obvious or striking signs of apparent conclusive significance. His opportunity passed when pathology came to her estate and subjected brilliant but superficial opinions to the acid test of the autopsy.

Precipitate Decisions.—Many complex cases are encountered in which several ailments are concurrent and the obvious diagnosis may not represent the lesion of chief importance in the given case. Hasty conclusions are damaging in most instances and their frequency lends emphasis to the rule that all examinations must be thorough and complete and that the discovery of one important condition must not close the mind to other possibilities.*

Sources of humiliation.

* Finding the malarial organism had led to a hasty conclusive diagnosis of malaria in a case seen recently and, as an antecedent diagnosis, tuberculosis had been affirmed quite properly because of positive sputum findings. A blood culture revealed streptococcus viridans and *the dominant and progressively fatal* ailment proved to be chronic recurrent bacterial endocarditis.

Like instances are by no means infrequent and failures in diagnosis are extremely common in decompensated heart lesions of the silent murmurless type, because of the assumption that some obvious *secondary* morbid change is primary.

Dyspepsia, albuminuria, hepatic enlargement, congestion of the lungs, chronic bronchitis and paroxysmal dyspnea are some of the common secondary conditions leading to hasty and erroneous conclusions and midway diagnoses.

The Fundamental Source of Diagnostic Error in Chronic Ailments.—*This may be briefly expressed by the statement that we do not take or are not granted the time indispensable to good work.*

There is evident still, that same tendency to seize upon that which is obvious and guess at that which is obscure, which, in times past, found its sole justification in lack of available knowledge and instruments of diagnostic precision.

If one is to do good work he must be thorough, painstaking, and deliberate, and the *patient* must be taught that a look, or even a "look-over," a few questions, and a prescription, do not constitute a modern examination.

Two basic factors.

On the contrary an intelligent opinion, the unravelling of the tangled skein of symptoms in an obscure case, means more than the work of any one day and may demand a more or less prolonged period of direct observation and complete control.

Exactly the same statement holds good with respect to failures in treatment.

A tendency to patch rather than to repair is the cardinal sin in therapy today, as it was a hundred years ago.

Futile methods.

We cannot cure inveterate "nervous dyspepsia" with digestants or intestinal antiseptics, nor greatly prolong the life expectancy of a decompensating cardiopath with occasional doses of digitalis and a half-hearted attempt to restrict activity.

Important field.

We can do both in surprising measure, if opportunity, coöperation and time are given us to make possible genuine repair and relative rehabilitation.

With respect to tuberculosis the lesson has been learned. With respect to cardiovascular disease and other ailments quite as important and responsive, we have yet to realize the possibilities both of diagnosis and treatment.

Taking the Long Odds.—Many students and some practitioners habitually diagnose medical curiosities, failing to thresh out the variations of the prosaic and simple before assuming the long odds. On the other hand, it falls to the lot of every physician to encounter rarities, and their recognition means added credit and reputation.

Consider the prosaic first.

The physician often encounters some unusual case, only to have it duplicated within a short time, and one hears often of the triplication of such cases.

Duplication of rare cases.

Such occurrences are less mysterious than they seem for the detection of the primary case intensifies our interest and alertness. *Diagnosis is easy, usually, if the given disease of great rarity occurs to us as a possibility*, and we at once proceed to analyze and clarify the hitherto obscure history.

We also magnify the fact of accidental concurrence of like ailments, forgetting the many *individual* instances which lack the apparently mystic duplication.

A fallacy.

Prognosis.—Accurate diagnosis is indispensable to an intelligent prognosis and, unless the patient is actually *in articulo mortis*, the uncertainty of mere time forecasts makes them veritable traps for the unwary.

Traps for the unwary.

We often see patients at the point of death rally and live for months or years even when suffering from chronic disease, and to an even greater degree

the statement applies to those apparently overwhelmed by the toxemia of acute infections.

Rash prophecies.

The physician who rashly sets *exact* time limits to chronic diseases in response to the natural and inevitable interrogation of the relatives of a patient seldom gains much credit from his few successful guesses.

Indeed prognosis has little to do with *specific* forecasts as to life expectancy, which must usually prove futile, but in its broader sense it is the field in medicine which best exercises the knowledge, judgment and ripened experience of its master minds.

The man, not the disease alone.

Prognosis embraces almost every factor that can enter into an accurate, broad and highly individualized diagnosis and peculiarly exalts and emphasizes the value of a study of the "personal equation," the setting apart of the particular man, woman or child afflicted with this or that disease, from the mass.

Common variants.

We must neither treat, nor forecast the result of, pneumonia of itself, but study the individual who is suffering from it. We know that the flabby fat man is quite inferior to his lean wiry brother in resisting power, and that the total abstainer is a better "risk" in disease, as in health, than the tippler. The vigorous young adult with sound heart and blood vessels stands quite apart from the man whose heart muscle and arteries alike have suffered from the storms and stress of four score years of life. The man of unimpeachable family history, sound physique, good habits and moral courage offers a far better prognosis than his fellow who comes from tainted or short-lived stock, is defectively constructed, the victim of bad habits or possessed of little or no courage and determination to fight for his life.

Vital factors.

Previous illnesses and their effects, the particular virulence of the individual infection, the condition of the heart, the extent of pulmonary involvement, and the degree to which the circumstances and environment of the patient permit the use of the full resources of modern medicine to be exercised in his behalf, are some of the factors which would enter into the prognosis. In other diseases, acute and chronic, the last consideration is extremely important.

Lost opportunities.

Optimism vs. Pessimism.—*The confirmed optimist in prognosis, even when an extremist, does far less damage to any, save himself, than does the pessimist, for the latter not only frightens many a patient into an untimely grave, but, by his willingness to stop fighting the apparently victorious disease, loses a host of golden opportunities to wrest victory from seemingly irreparable disaster.*

Damaging revelations.

Pessimistic forecasts in relation to chronic disease, especially when *bluntly and tactlessly* communicated to the patient himself, are oftentimes fraught with disastrous results, and in acute disease the physician is seldom justified in exercising a *brutal frankness* which may serve no good purpose, but remove one of the strongest of the fighting reserves of the sick man.

In certain instances it is wholly proper and necessary to convey to the patient the advice that he take precautionary steps with relation to his affairs; but this may be so delicately imparted, and so guarded by hopeful and comforting assurances, as to cause little shock in most cases.

A Difficult Problem.—On many occasions the doctor is urged by family members to tell the failing but hopeful or unsuspecting patient flatly that he must die or is actually dying. Instances occur which make it necessary for the physician to assume this delegated function and of its legitimacy in the individual instance he must be the judge. The only deathbeds the author has attended in which peace was lacking at the end, have been those in which weak, high-strong patients were told that death was inevitable. On the other hand, he has witnessed the most beautiful resignation in the face of such knowledge.

Euthanasia.

The wonderful euthanasic calm which so generally precedes dissolution robs death of much of its pang and is not lightly to be broken, but, if it must be, the same mysterious, kindly influence ofttimes softens the blow. Nevertheless, the demands of religious belief, the known character and temperament or expressed wish of the patient, certain legal formalities, such as the "dying declaration," which demands that the statement be made under the firm conviction that death is impending, justify such a disclosure, *but only in rare instances need it be absolute, unqualified and unattended by some expression of hope.* In any event, bald disclosure is but seldom the duty of the physician and its damaging effect, as long as any atom of fighting hope remains, must be remembered.

Obligatory revelations.

Truthfulness Desirable.—*All this does not mean that a patient cannot be told the nature of his ailment even though the full and detailed knowledge of its gravity or severity be withheld.*

Securing intelligent coöperation.

This statement applies with peculiar force to cases of tuberculosis in that not only is the patient's coöperation absolutely essential to arrest or cure, but indispensable to the protection of others.

The questions of frankness, evasion or actual temporary deception, are determined by ordinary common sense which takes into consideration primarily, the fundamental fact that the **physician's function is that of securing for the patient the maximum of benefit.** *If the patient may know the truth without added danger to himself, that truth should be told and, in chronic cases especially, it is rarely wise to keep the patient in ignorance of his condition.*

The fundamental factor.

Tactful Disclosure.—Heart disease, Bright's disease, pernicious anemia and many other conditions can be dealt with effectively only when the full coöperation of the patient is secured, *for absolute control, full opportunity for supervision and abundance of time, are absolutely necessary to his welfare.*

Dread terms

In the minds of the laity the terms "heart disease" and "Bright's disease" have a meaning but little less dreadful than cancer and this is the inevitable result and reflection of the traditional general attitude of hopelessness and neglect held by the medical profession toward these conditions.

Fallibility of forecasts.

Knowing as we do the wide differences existing in the subdivisions of these ailments, the extraordinary frequency of overaccentuation and overestimation of the gravity of the individual case, the astonishingly long duration and slow progress often manifested, the curious and conservative transformations in type which are possible **and the wholly insuperable obstacles to diagnostic and prognostic impeccancy,** *we serve our patients best, usually, by avoiding the sinister*

concrete terms and telling them roughly and sketchily the pathologic causes or the general underlying basis of their symptoms.

Tactful honesty.

The patient who would translate into the terms of a rapidly fatal disease the words "Bright's disease," may be disturbed relatively little by the knowledge that his kidneys "are inadequate" to their daily task whether as to excretion, filtration or secretion, by reason of some changes in the filtration apparatus and the secreting cells. Thence, he is told, comes the tendency to retain at times body poisons, raise blood pressure and overtax the circulation and the necessity for his faithful and conscientious coöperation is manifest.

Revelation with reassurance.

The revelation of the fact that "the circulation is deficient," that the heart muscle "is deteriorated," "is a bit short on horsepower" or "lacking in tone," or even "that one of the valves is not wholly competent," oddly enough, may be told a patient with far less in the way of resulting shock than might be supposed. Furthermore, even though a fuller knowledge of a genuinely serious condition must be imparted, there is a world of comfort to the patient in the citation of actual instances of the same sort where the happiest results were obtained.

Usual response.

The intelligent patient at once sees that a logical reason underlies the demand for his coöperation and is the more likely to prove genuinely helpful and responsive, while retaining that hopefulness and undamaged *morale* which may bring added years of useful life.

In general, the physician whose only thought is the welfare of his patient and who is possessed of humanity, tact, and sound common sense will not go far astray.

The fool oftentimes must be frightened into obedience and some fools must be abandoned to their folly.

Firmness and decision.

Practitioner and Consultant.—The physician must work methodically, deliberately, and with open mind, but once his opinion is formed it should find tactful, clear and emphatic expression and every subsequent order and act must be characterized by firmness and decision. If to these qualities he adds that modest self-confidence, born of fulness of knowledge and resource, he will go far.

Consultation.—Such a man can deal with intelligent patients frankly and freely, will not be afraid to call counsel nor feel always that his after-conference with his consultant must be held in private, according to a custom more honored in the breach than in the observance.

Few physicians will use a visit of courtesy as a means of self-advantage or the humiliation of a fellow-practitioner, and one who cannot deal fairly with all parties concerned is out of place in this day and generation.

Lay Co-workers.—There are few intelligent families, moreover, in which the attending physician cannot find a confidant with whom he can talk freely and honestly, thereby increasing his usefulness and gaining coöperation and support.

Attitude in Sickroom.—Doubts, fears, and arguments, however, are not for the sickroom, where a confident, cheery bearing and helpful suggestion may mean far more than drugs.

Quack vs. Physician.—The quack never hesitates to make a diagnosis, but the physician of parts, knowledge, and honesty must often make none or at best a provisional one, and wait for more light. Honesty vs. ignorance and dishonesty.

The charlatan never acknowledges an error; the honest man, whatever his ability, must occasionally confess one.

The more superficial, ignorant or dishonest the man, the more dogmatic and hasty are his diagnoses, for with breadth and depth of knowledge comes its highest gift, a conception of its limitations.

CASE-TAKING

Significance and Importance of the Components of the Case History

The Value of the Case Book.—*It should be stated most emphatically that case-taking, recording, and reporting should be carried into every man's practice.*

Old case books well kept are mines of knowledge, and the science of medicine would be greatly enriched were the workers in city and hamlet alike to give to it reports of the unusual cases now for the most part allowed to pass without record. Unworked mines.

Tyro vs. Expert.—Ability to make a case history full, accurate, yet concise; to elicit the salient facts, and to each assign its proper value and perspective, demands that the observer be full of usable knowledge, quick of perception, and capable of avoiding both omission and verbosity. Essentials of case-taking.

Certain routine inquiries are indispensable and the student should be consistently painstaking and systematic in the days of his apprenticeship, though later, when a skilled clinician, he can elicit the cardinal points of a case and arrive at correct conclusions with less ink and fewer words.

The Subjective vs. the Objective.—Our knowledge of clinical symptoms in a given case depends upon either (*a*) *what we are told or* (*b*) *what we see or determine for ourselves; i.e., they are either* (*a*) **subjective** *or* (*b*) **objective,** the latter group including bacteriologic and chemic tests as well as physical signs.

"Hearsay Evidence."—In the patient we may encounter garrulity, stupidity, concealment, deceit or hypochrondriacal exaggeration. If he is comatose or possesses no common language, we are left dependent upon the testimony of outsiders. In any event we must exercise sound judgment, keen discrimination and facility in cross-examination or we cannot weigh properly the fallible, yet extremely valuable *subjective* data. Obstacles to case-taking.

THE OUTWARD SIGNS OF DISEASE

The First Impression.—The first glance or even the handshake may influence diagnosis and prognosis, and, while shunning the habit of making hasty physiognomonic diagnoses, the physician should train his observation and emulate the example of his predecessors of a darker medical era, who became adepts by force of necessity. Value and scope of inspection.

Diagnosis in disease of the nose, throat, eye, ear, and skin is essentially that of inspection, which throughout the whole range of medicine and surgery plays an important part.

Character and temperament, bad habits, disease past or present, may stand revealed at a glance.

Diathesis.*—"Temperament," which relates to the physical type and mental cast of the individual and was born of the old humoral pathology, is closely related to "diathesis," which applies to the group, but the latter implies a tendency to some special type of disease or diseases and the former has lost the prominent place it held in earlier times. One need give little thought to the elaborate older classification, though the "nervous," "bilious," "phlegmatic," "lymphatic," and "melancholic" temperaments are often clearly defined and aid in diagnosis and prognosis alike. Who, for example, has not encountered the "lymphatic" (congenitally asthenic) individual with a "prairie-fire tuberculosis" before which the physician stands helpless?

FACIAL EXPRESSION.—The expression helps us with those forgers of symptoms who seek ease, food and shelter under the hospital roof or wish to saddle damages for some spurious hurt upon the rich individual or corporation.

Malingerers.

Melancholia.

If melancholy has marked the patient for her own, "he carries on his face the impress of her signet," and incipient or established insanity of other types may manifest itself in a peculiar expression of the eyes, an "intensification of emotional expression" and oftentimes a deceptively merry one.

The stolid or even morose appearance of the immobile "mask-like" face of paralysis agitans or the fixed expression of surprise or fright which it may bear, and even the slighter facial palsies are often overlooked by the unobservant.

Facies, pupils, and speech.

General paresis even in its early stages often yields outward evidence. Lips, tongue, fingers and facial muscles may be tremulous; the pupils unequal and reacting to accommodation, but not to light, or wholly rigid. The speech may be slow, hesitant or explosive, consonantal words being difficult for the victim. In the "expansive" type excessive irritability, outbursts of causeless anger or delusions of grandeur accompanied by rapid changes of facial expression may be evident; everything of his being superlatively beautiful, his schemes grandiose yet obviously fallible, and he incomparable. The "depressive" form is characterized by a marked but somewhat unstable mental depression often associated with delusions of the hypochondriacal or even persecutory type.

Carcinoma of the stomach in its advanced stages is frequently associated with a curiously morose or saturnine expression well shown in a famous statue of Napoleon in his last days.† This, when associated with marked emaciation and a peculiar muddy pallor, is most suggestive.

Luetic babes.

Syphilis.—The syndrome formed by a peculiar eruption, coryza ("snuffles"), a hoarse cry, a weazened monkeyish face, fissured lips and raw nostrils and buttocks, if seen in a babe, is pathognomonic of congenital syphilis.

* Modern research has robbed the term of much of its meaning and ancient importance through the discovery of specific etiologic factors in relation to its manifestations.

It would be absurd, for example, to refer to the "furuncular" or "aneurysmal" diathesis in the light of our present knowledge of the infections underlying these conditions.

† In the Corcoran Gallery, Washington.

Hutchinsonian teeth.

The permanent upper central incisors of the older children are often peg-shaped, notched at their cutting edges, irregular and separated.

Keratitis and frog-face.

Fine linear scars may radiate from the angles of the mouth, *keratitis and chronic otitis media* often co-exist, and at any age a frog-face may result from syphilitic necrosis of the nasal arch. Syphilitic alopecia may be present also, or may have left its mark in a patchy grayness of the hair.

Putting the question.

Many cases of past syphilitic infection give no evidence, the examiner being forced to put the direct or indirect question. The former may oftentimes be definitely and clearly put in the case of men, but it is usually wiser to ask casually first as to antecedent "venereal" or "private" disease, or, in certain instances of especial delicacy, to quietly submit a blood specimen to the pathologist and await the report on a Wassermann test.*

FIG. 1.—Classical facies of untreated or inveterate *lues*. Areas of gray hair representing past alopecia. "Saddle nose." Patient and his father both showed perforated palate.

The therapeutic test is often invaluable though fallible and no mere negation on the part of the patient should outweigh a significant symptom group so far as the physician's attitude is concerned.

Outward acceptance, inward doubt, and appropriate action make a good tripod.

A pathognomonic syndrome.

One of the most easily recognized and pathognomonic (but in our day rare) syphilitic types combines alopecia or scattered areas of gray hair with saddle-nose and perforated palate.

A deeply fissured tongue *suggests* this disease, and many cases present themselves with significant body scars, an active and unmistakable eruption, or the "primary lesion itself."

Social station and occupation.

Dress.—The quality of the clothing and its condition may suggest *social station* and *occupation*. *Carelessly worn and disarranged garments, stained by food droppings*, if noted in a person formerly neat, suggest mental deterioration. *An ammoniacal odor suggests advanced prostatic disease with urinary incontinence or cystitis*, and in such cases white stains upon the shoes might sometimes give a hint of coexisting saccharine *diabetes*.

Ill-fitting clothing.

Recent Weight-Loss or Gain.—In public services especially, needy patients, formerly obese or dropsical, who have recently lost greatly in weight, may wear their old clothes, which may be suggestively loose and, on the other hand, a recently developed anasarca may make the patient appear to be bursting through his trousers.

Loose lacings.

Edema of the ankles is frequently suggested by loose lacing of the shoes, which may be cut and slashed for the same reason or the better to accommodate corns, bunions, or gouty toes.

Paralysis.—In the various forms of pathologic gait, and especially in spastic conditions, the wearing away of the shoe in a particular part may be suggestive.

* The student must realize that no individual, whatever his or her rank or calling can safely be held exempt from suspicion, yet tact and delicacy are essential. The therapeutic test and the Wassermann show how often a negative response must be discounted.

Phthisis.

Prostitutes.

VOICE AND SPEECH.—One should carefully note departures from the normal tone and enunciation. Even the layman recognizes the *soft, high-pitched* tones of the phthisical patient, and one notes oftentimes the *peculiar harshness in the voice of the prostitute* and the *thick muffled speech of* the victim of *tonsillitis or adenoid disease.*

Larynx.

Hoarseness and aphonia are at times symptoms of great importance, as pointing to acute or chronic laryngeal diseases, and the "*whispering voice*" may arise from tuberculosis or malignant laryngitis, extreme exhaustion, hysteric aphonia or a paralysis of the abductors, the last being due oftentimes to the pressure of an aneurysm or mediastinal growth upon a recurrent laryngeal nerve.

Slow, scanning, syllabic speech, combined with an intention tremor, suggests disseminated sclerosis or, very rarely, Friedreich's ataxia.

In advanced paralysis agitans **the crescendo speech** *may sometimes be encountered, the sentence being begun slowly and with hesitation, but increasing in rapidity and ending in a storm of words. Monotony of tone is the commoner vocal characteristic of this disease.*

In glosso-labio-pharyngeal paralysis, amyotrophic lateral sclerosis and pseudo-bulbar paralysis, one encounters a **mumbling speech,** associated with tremor and atrophy of the tongue and a curious immobility of the lips.

Essentials of speech.

Aphasia and Anarthria.—Normal speech demands that the centers and mechanism for the perception and recollection of spoken and written or printed words, of articulation, and of association, should be normal and harmoniously interacting.

It is evident that various disturbances may exist according to the character and location of the lesion; furthermore, that we must discriminate between "aphasia" and the cases of paralysis of the *muscles* of articulation ("*anarthria*"). True aphasia is usually of central origin, the peripheral mechanism being unaffected.

True aphasia vs. anarthria.

Motor Aphasia.—This must be recognized as distinct from sensory aphasia. It is characterized by inability to voluntarily express by speech ("*aphemia*"), by writing ("*agraphia*") or by gestures ("*amimia*") what may be a perfectly clear mental image.

Aphemia, agraphia, amimia.

Sensory aphasia, on the other hand, indicates a failure to understand or recognize spoken words (*auditory aphasia, word deafness*) or written words (*visual aphasia, word blindness*). A large number of subdivisions occur under both types, such as the loss of power to carry a tune or write music, or a failure to interpret or recognize musical sounds. Still another variety ("paraphasia") occurs in which wrong words or characters are spoken or written, or the same word repeated.

Apraxia.—By this is meant a loss of the faculty of recognizing or appreciating the identity, nature, and uses of objects or sensations, and here again one has various subdivisions, such as mind blindness (*visual amnesia*), mind deafness (*auditory amnesia*), as well as terms indicating an inability to recognize and interpret odors, tastes, etc.

THE COLOR OF THE SKIN.—The two conditions chiefly to be noted are *pallor* and *cyanosis*. *The pale face may or may not mean true anemia, for a high color may co-exist with a low hemoglobin percentage.* The color of the mucous membranes is more reliable and even "*rosy chlorotics*" usually show a pale conjunctiva. Ordinarily, pallor of the cheeks, *lips*, tongue, throat, ears, conjunctivæ and finger-nails indicate a reduced hemoglobin percentage with or without a reduction in cells, though it may only mean a reduced amount of normal blood at the surface due to contracted vessels or a weak circulation.

A misleading symptom.

Anemic Types.—*The color varies markedly in different types of severe anemia*, being greenish yellow in typical cases of chlorosis, almost waxy white in many simple anemias, a peculiar lemon yellow in most cases of Addisonian anemia, and a curious earthy tint in the profound secondary anemia of advanced malignant disease of the stomach.

Chlorosis, pernicious anemia, and cancer.

Mode of Onset.—Sudden pallor, as in minor shock, syncope, nausea or certain neuroses, is usually unimportant and fleeting, but, *in tubal pregnancy, cardiac disease, concealed hemorrhage, perforation of the stomach or bowel, and similar conditions, sudden pallor is a valuable and important sign.* The pallor of the anemias, aside from those due to rapid blood loss, is slowly progressive and persistent.

Sudden pallor.

Nephritis.—In *acute Bright's disease* the skin is white and associated edema may produce a peculiar and characteristic *pasty pallor*. In parenchymatous nephritis we meet with pallor, or in its late stages a sallow or brownish hue.

Acute vs. chronic nephritis.

Many authors speak of the peculiar "fawn-colored" skin as related to interstitial nephritis, but the author has found it oftenest in the "mixed" type of chronic kidney disease or in very late stages of the interstitial form,

Mixed and interstitial nephritis.

FIG. 2.—Bright's Disease. Facies occasionally encountered in chronic nephritis. Morning fulness of eyelids.

FIG. 3.—Appearance of lids in afternoon or evening.

the earlier period being often associated with the ruddy or high-colored ("clubman") countenance, and many of its victims appearing exuberantly healthy. *In most instances interstitial nephritis exists for long periods without the slightest physiognomonic sign.*

Baggy Eyelids.—A tendency to edema of the eyelids, especially of the lower, is often evident, and the pale, puffy and almost translucent lid of early morning may have become shrunken and wrinkled later in the day. The condition cannot be considered as pathognomonic, but should be regarded as suggestive. In chronic parenchymatous and "mixed" nephritis alike, the face may appear more or less puffy, sallow or even fawn colored.

Diurnal variations.

Heart Disease.—The color in certain forms of advanced heart disease is both interesting and important.

False pallor. False color.

Aortic regurgitation is usually associated with a pallor of the indoor-worker type, without true anemia, *but in endocarditic regurgitant and obstructive disease of the mitral valve*, even if compensation is present, *the color is often deceptively high*, producing, in girls especially, what the laity may consider an exquisite complexion, though in older women the pink is duskier and more patchy.*

Selective points for cyanosis.

The skilled eye detects the underlying cyanosis which shows still more plainly in the mucous membrane of the lips, the skin of the ears, nose and patella, or in the nails whose pink is replaced by a darker hue or, in extreme cases, by a purplish or blackish gray.

Venous obstruction and impaired oxidation.

Cyanosis.—This, whether general or local, with or without true dyspnea, ordinarily indicates obstructed venous return, deficient oxidation, or commonly both factors, such as may result from any of the following conditions: ordinary suffocation, congenital heart disease, asthma, emphysema, pulmonary fibrosis, obstructed glottis, trachea or bronchi, as from foreign bodies, croup, laryngeal diphtheria, capillary bronchitis or broncho-pneumonia proper, mediastinal tumors, or other heart and lung lesions.

Acute diseases.

It occurs also in acute diseases, such as pneumonia, pleurisy with either liquid or gaseous effusion, and, to a slight degree, in severe acute bronchitis. Paralysis and spasm, particularly of the diaphragm, may produce marked cyanosis, as may the inhibition of efficient respiration by severe pain.

The chief and most important physical factor in both local and general cyanosis is the slowing of the circulation, whether central, localized or due to increased viscosity and blood volume, as in erythremia, but the reduction of hemoglobin may result from the action of chemical poisons alone without any material slowing of the blood current.

In lobar pneumonia, profound cyanosis of the finger-nails is usually the forerunner of death.

Extreme cyanosis in ambulants.

No outward sign of disease exceeds cyanosis in importance, and in its extreme form it exists in but three classes of **walking patients,** *viz., severe emphysema or erythremia in the adult and congenital heart disease in the child.*

"Red cyanosis."

It is often associated with mere chilling of the body surface, hysteria, neuritis, etc. Erythremia is associated with a peculiar red cyanosis and in all cases of erythrocytosis cyanosis is a prominent feature.

Drug cyanosis.

Local vasomotor relaxation or paralysis is readily distinguished by the lack of turgidity of the venous trunks, and it should be remembered that certain forms of *drug poisoning*, especially that of acetanilid and its congeners, nitro-benzol, etc., may account for an otherwise inexplicable and extreme cyanosis.

Cold skin.

Through excessive heat loss cyanotic areas are cold save in acutely inflamed parts. The slighter degrees are often unnoticed by the student, *but pronounced cyanosis cannot be overlooked.*

*Such deceptive color is frequently present in splenic leukemia, even when quite advanced.

Other Abnormal Color Variations.—Of the many other departures from the normal tint one may mention the yellowish brown, dark brown, or brownish black of *Addison's disease*, the peculiar bluish or blackish gray of *argyria* (chronic silver poisoning), the sallow tint of *chronic malaria* and the varying yellows produced by *jaundice*.

Yellow, brown, and black skins.

Arsenical melanosis is quite common and may exactly simulate Addison's disease, but usually diminishes under drug discontinuance. Marked pigmentation is occasionally encountered in exophthalmic goiter. The yellowish-brown patches of pregnancy (*chloasma gravidarum*) and pelvic disorders are common and easily recognized. Certain abdominal tumors and peritoneal tuberculosis may cause extensive and misleading color changes.

Chloasma gravidarum.

Streaky pigmentation usually indicates scratching as the result of skin parasites or pruritic lesions and is often seen in public clinics. One occasionally encounters a pigmentation of like causation so diffuse as to closely simulate that of Addison's disease.

Vagabond's pigmentation.

Melano-sarcoma is associated with a grayish or blackish skin and the urine shows melanin. A brownish or dirty gray, spotted or patchy discoloration may occur in **hepatic cirrhosis** with or without jaundice, and *bronzing* may be associated with combined cirrhosis and diabetes (**bronze diabetes**) or, in rare instances, with extreme long-standing jaundice alone. Aside from its secondary color manifestation, **syphilis** in its tertiary and congenital form sometimes produces a peculiar sallow pallor not easily described, but readily observed. The author has encountered it chiefly in old cases of imperfectly treated syphilis, and it is likely to be associated with other luetic "reminders."

Drug Habitués.—Peculiar but indefinable forms of pallor and sallowness may mark the *cocain and opium* habitué, and occasionally the author has observed in such cases a physiognomy remarkably like that of chronic nephritis; but more often the habit may exist for years without distinctive outward signs.

Obscure pigmentation.

Certain cases of deep pigmentation occur lacking demonstrable etiology and without impairment of the general health.

As regards both marked color loss or its decided modification in chronic diseases, it should be borne in mind that the symptom is, as a rule, one indicative of the advanced stage of the disease.

High Color.—*A florid face* is often present in gouty subjects or in many of those suffering from early interstitial nephritis or hepatic cirrhosis, but may be due to idiosyncrasy, acne rosacea or daily exposure to harsh weather conditions. It often indicates an overluxurious and self-indulgent life or the abuse of alcoholics.

Not always high living.

Unilateral flushing is frequently observed as a neurosis, in lobar pneumonia, or as the result of mere *pillow pressure*, migraine, and, less commonly, of irritation of the fibers of the cervical sympathetic as in aneurysm of the aortic arch or mediastinal growths.

"Morbid blushing" is a troublesome and not uncommon vasomotor idiosyncrasy.

The flushed face of fever patients is too common to merit extended discussion.

General cutaneous hyperemia is seen in poisoning by belladonna, hyoscyamus, or coal-tar products, is common in the trivial fevers of infancy and early childhood, and may precede the specific exanthem in fevers of the eruptive type.

JAUNDICE.—(*Icterus*) **Hepatogenous vs. Hematogenous.**—Clinically, jaundice is either "*obstructive*," as in inflammation of, or direct or indirect obstruction of, the common duct, or, "*toxemic*," the latter form often being wrongly termed *hematogenous*. Although hemolysis is the primary change, both types are essentially hepatic as regards the direct source of bile pigment, and "obstruction" and "diffusion" jaundice cover nearly every type.

Hemolysis.

Toxic jaundice.

There are toxins which act directly upon the blood itself (within the hepatic tissues) or affect the hepatic cells. Poisoning by phosphorus, potassium chlorate, ether, chloroform, toluylendiamin, snake venom and arsenic are examples of one form; yellow fever, pyemia and malaria of another; while certain infectious diseases are *characterized* by marked associated jaundice. Such are "*Weil's disease*" and "*acute yellow atrophy*."

It occasionally complicates pneumonia, ulcerative endocarditis, syphilis, relapsing fever, and even influenza.

The typical mild jaundice is seen in simple obstructive catarrh of the bile ducts. In hepatic cirrhosis it is late and seldom extreme.

Blocked bile current.

Obstructive Jaundice.—The term "*obstructive*," as usually applied, covers a blocking of the passage of the bile from the liver to the intestines with resultant absorption into the general circulation.

The causes are severe gastro-duodenitis, catarrhal conditions, gall-stone, or parasites, involving the common duct, pressure closure by tumor, or, very rarely, by fecal accumulation, the pregnant uterus, or abdominal aneurysm.

*Strictly speaking, all cases of jaundice are obstructive, as even in the toxic form there is a high viscidity which favors absorption.**

Icterus nervosa.

Emotional Jaundice.—The remarkable jaundice associated with mental shock or profound emotion is probably obstructive and due to spasm and reversed peristalsis. In one such case coming under the author's notice profound jaundice followed a mere fleeting fit of anger.†

Dardanelles Jaundice.—This curious form, very prevalent amongst the British soldiers of the Dardanelles expedition, was non-obstructive, and proved to be due to a paratyphoid bacillus.

Hereditary Jaundice, of the Minkowski Type.—This interesting condition may affect succeeding generations and appears in several forms.

First, **hereditary icterus neonatorum,** in which an angiocholitis and common duct stenosis is commonly present and an extreme mortality ratio observed.

Second, a peculiar form associated with hepatic and splenic enlargement, anemia and infantilism.

† Carcinoma of the liver is accompanied by jaundice in more than one half the cases, but the cause is found to be pressure from the adjacent involved glands. In abscess of the liver, single or multiple, it may be absent or only slight.

* Jaundice is observed occasionally in cases of brain concussion.

Third, a congenital acholuric form with splenic enlargement, no bile in the urine, and little disturbance of the general health.

Symptoms of Jaundice.—*The skin, ocular conjunctiva, and the oral and pharyngeal mucous membrane yield the best evidence*, especially if the latter are blanched by the pressure of the finger or better by a microscope slide, *but even a marked discoloration is invisible by artificial light.* Importance of daylight.

By daylight the color of the skin varies from a faint or brilliant yellow to a deep greenish brown or bronze (melas-icterus), sometimes simulating Addison's disease, and not infrequently present in the last stages of ordinary cirrhosis of the liver as well as in that extremely rare form of combined interstitial hepatitis and pancreatitis known as *bronzed diabetes*. "Diabète bronze."

Obstructive Type.—The sweat and urine *are discolored* in clinically obstructive jaundice*, the latter often yielding the first evidence of the condition; **the pulse and respiration** are usually slow; *the stools are pale gray, pasty and fetid;* either constipation or diarrhea may be present; troublesome pruritus is common and urticaria or purpura occur in occasional instances. Clay-colored stools.

A marked hemorrhagic tendency is shown in severe cases and is of special interest with relation to surgical procedure.

There may be marked *mental depression* or extreme irritability and, *in certain grave cases, muscle cramps, convulsions, active delirium or a typhoid state may end in coma.* The urine often contains albumin and hyaline casts.

Acute Yellow Atrophy.—True malignant jaundice is a rare disease of unknown causation† chiefly affecting women, often related to pregnancy, sometimes to violent emotion or shock, but is probably invariably associated with some antecedent or concurrent infection. "Icterus gravis."

Icterus Neonatorum.—*There is a mild type*, benign and trivial, extremely common in new-born children, occurring during the first twenty-four or forty-eight hours and lasting for a week or two. The mild form.

The grave form of icterus of the new-born, when not hereditary, may be due to sepsis, usually of umbilical origin, to syphilitic disease of the liver, or to congenital absence of the ducts. It is frequently associated with hemorrhage from the navel and is a fatal disease. A fatal ailment.

Toxemic Jaundice.—In this condition jaundice is usually less intense, the stools are colored, the urine contains little or no bile, and the disease terminates promptly in recovery or death. It is associated with severe acute infections of varying character, certain forms of mineral or snake-venom poisoning, or the pernicious forms of anemia and malaria, and the constitutional symptoms may be profound.

In certain fatal cases observed by the writer actual obstructive jaundice of the ordinary clinical type has been closely or even exactly simulated but disproven by operation or autopsy. Jaundice may be extreme.

Urobilin Icterus.—Urobilin cannot cause icterus, but may be associated

* The milk of nursing women and even the sputum may be discolored.

† But 250 cases are on record since the first observation by Ballonius in 1616 (Osler).

with bilirubin in a jaundice due to partial biliary obstruction, and in slight degrees of obstruction may appear alone in the urine.*

DRY AND MOIST SKIN.—Hyperidrosis.—Sweating of the hands or feet may indicate idiosyncrasy, debility, congenital asthenia, or sexual neuroses.

Unilateral sweating, especially of the head or face, like pallor or cyanosis, occurs in pressure involvement of the sympathetic and in certain migraines and neuralgias. Sweating of the head is especially common in rickets and, of half the body, in rare instances of hemiplegia.

General sweating occurs as a critical phenomenon in certain acute diseases, as an associated symptom in malarial fever or phthisis and persistently in acute rheumatism, collapse, and severe pain.

Malnutrition and fluid loss.

Anidrosis.—A dry skin is associated with profound malnutrition and with most diseases causing extreme loss of fluid by the bowels or kidneys. It is a pronounced symptom in certain ailments such as myxedema, in which the skin may be both dry and harsh, and diabetes, chronic interstitial nephritis and carcinoma also furnish good examples.

Sudden checking of perspiration in those working under conditions of extreme high temperature is a well-known warning of impending heat stroke.

Qualitative Changes in the Perspiration.—The yellow sweat of jaundice, the extraordinary and rare, blue, brown, yellow, red, or even hemorrhagic perspiration of hysteria, the sour-smelling sweat of acute rheumatism, the disgusting odors of bromidrosis or the urinous taint of the sweat in diseases associated with impaired renal function may be encountered.

General terms.

EDEMA.—Three terms are used in this connection: (1) **edema proper,** *i. e.*, fluid confined to the actual connective tissues and usually localized; (2) **dropsy,** the accumulation of fluid in the serous cavities combined with edema; (3) **anasarca** (general edema). The last term, however, is often used synonymously with the one preceding.

Obstructive vs. Hydremic Edema.—Edema represents a capillary transudation exceeding the absorptive capacity of the lymphatics and may be obstructive (passive congestions), or hydremic (toxemias, infections, cachexias) and in many if not most instances represents a combination of both. Retained toxic substances and increased permeability of the systemic capillary walls are doubtless prominent factors in the latter, if not in both groups, and experiment fails to prove the older theory of pure hydremic plethora, *i. e.*, increased blood volume and water retention, while nevertheless indicating that the latter may play some part.

Obstruction and hydremia.

Salt retention.

In both the so-called static and hydremic forms, and especially in that associated with chronic parenchymatous nephritis, sodium chloride retention has been shown to assist in maintaining the edema.

Milroy's Edema.—This is a curious *hereditary, permanent* edema of the legs usually asymmetrical in its early stages.

Inflammatory Edema.—The clinical distinction between *inflammatory edema* and ordinary edema is largely dependent upon the physical constituents

* Such is the case in the two latter forms of congenital jaundice mentioned on the preceding page.

of the inflammatory exudate as compared with the simpler *transudate*, but, inasmuch as pronounced evidences of local inflammation and usually of acute infection accompany the former, no difficulty can often arise in differentiation.

Vagaries of Edema.—A thorough knowledge of the favorite locations and the vagaries of this condition is essential, inasmuch as it may shift its seat, obey or disobey the law of gravitation, and vary from swelling of the lids, slight puffiness of the ankles, or a localized *angioneurotic edema*, to general dropsy, in which the patient is drowning in his own transudate.

Angioneurotic edema is a localized* but *transient*, firm swelling, closely allied to urticaria and erythema nodosum, not ordinarily pitting readily on pressure, found in various regions of the body in certain ill-defined conditions and serious only when, as rarely happens, it involves the glottis. Usually transient and trivial.

Slight and Transient Edema of the Lower Extremities.—Aside from the lesser degrees and earlier stages of many of the recognized causes of edema this is found in cases of simple anemia, or after prolonged and exhausting tramps in those unaccustomed to physical exercise. An important and elusive sign.

In the earlier stages of gradually induced cardiac decompensation edema may appear late in the day, only to vanish during the night's rest, and its absence during the greater part of the waking hours causes many errors of observation.

Of certain cases of Bright's disease the reverse is true, slight puffiness of the eyelids being present in the early morning, but vanishing during the forenoon.

Cardinal Signs of Edema.—*The characteristic feature of all simple edemas is the appearance of a non-inflammatory swelling, often very slight, but usually tending to obscure the normal outline of the affected portion, readily receiving, and for a variable period retaining, indentations produced by the pressure of the examiner's finger or by the constriction due to clothing.*

Cavity Exudates and General Dropsy.—*In its extreme grades the same process tends to produce liquid transudates in such serous cavities (lymph spaces) as the pleura, pericardium, and peritoneum, producing what are known respectively as "hydrothorax," "hydropericardium" and "ascites."* Lymph-space transudates.

Such conditions accompanying general edema constitute "*general dropsy*," which in practice is limited to *Bright's disease, a failing heart, from whatever cause, or, both combined.*

In Bright's disease, especially, an hydrothorax or hydropericardium may occur suddenly and unexpectedly and escape observation until serious pressure symptoms are manifested even though no localized or general edema exists elsewhere in the body. Unheralded complications.

Cardiac Edema.—As between renal and cardiac conditions the attendant edema at times presents some distinguishing features.

In heart disease, edema (predominantly obstructive), even without general anasarca, is associated usually with cyanosis, often very slight, *and in its original seat and progress* usually follows the law of gravity, commencing in the feet (or over the sacrum in recumbent patients) and extending upward. Seeks the dependent part.

In all forms of non-inflammatory edema the slighter primary manifestations occur in loosely bound connective-tissue areas of maximum distensibility. Primarily in looser tissues

* Very rarely the condition is universal in distribution.

Primarily disregards law of gravity.

Renal Edema.—In acute and chronic parenchymatous nephritis any marked edema (hydremic) usually appears *first* in the face and eyelids and extends downward, being associated with marked pallor, *unmixed with any considerable degree of cyanosis*, unless there be obstructive effusion in the peritoneal cavity or thorax, or, as frequently happens in the established cases, there exists a complicating decompensation on the cardiac side.

Marble edema.

Furthermore, the edema of **acute Bright's disease** *as it appears on the body generally, or the extremities, is distinctly firmer than a recent cardiac edema*, and in persons with a delicate skin and more especially in children, the blue superficial venules contrast sharply with the dead white of the tense overstretched skin (marble edema).

Cardiac type of edema.

Interstitial Nephritis.—In typical cases edema of any severity is absent until the terminal stages are reached and then appears as a result of cardiac insufficiency and is of the cardiac type.

Diurnal vs. nocturnal edema.

Effect of Posture.—Edema of the cardiac or renal type, *when established*, is markedly affected by changes in the position of the patient; that of heart disease or parenchymatous nephritis, especially so, the attitude assumed by such a case during any night being often indicated in the morning by an increased swelling of the side upon which he has lain. As before stated, cardiac edema usually appears first in the most dependent portion of the body, as in the feet and ankles after a day's activity, or over the sacrum in the bedfast.*

Misleading or elusive edemas.

Leathery Edema.—In those curious, partially compensated heart cases with chronic gravity edema, in which the ambulant patient mistakenly or from necessity keeps on his feet for weeks, months, or years, one may see a peculiarly hard edema of the legs and lower thighs.

The skin becomes leathery and pigmented in such instances and in consequence can be indented only by sustained firm pressure.

One should not be misled by certain edemas of the lower extremities due to *varicose veins*, usually, but not always, unilateral, *though seldom equal in degree* if bilateral.

Edema of the calves without edema of the ankle or other regions has been observed by the author in several victims of heart disease possessed of unusually tight, trim ankles. Such an edema might easily be overlooked if, as is usually the case, only the ankles or lower tibial shaft are tested.

Collateral Localized Edema.—*Over purulent exudates, areas of suppurative inflammation and severe neuritis, such edema may, but does not always, occur;* as, for example, in empyema, mastoiditis, parotitis, pericarditis, hepatic abscess, perinephritic abscess, superficial lymphangitis and multiple neuritis.

Urticaria and angioneurotic edemas.

Non-inflammatory circumscribed edemas are either angioneurotic, purpuric, giant urticarial, or, of the ordinary obstructive type due to the mechanical blocking of veins or lymphatics.

Unilateral vs. bilateral edemas.

Edemas are almost invariably bilateral, if of the cardiac or renal type; unilateral, if due to the blocking of the local circulation; but bilateral edema may also result from blocking of a main venous trunk.

* Tenderness over the tibia, if bilateral, should always suggest persistently recurring edema even if none is detected at the time of examination.

Edema of the arm may be due to the pressure of tumors or enlarged glands upon the venous drainage channel, to thrombosis, and even to massive pleuritic effusion or mediastinal tumors, including aneurysm. Venous block.

Obstructed flow in the femoral veins causes edema of one leg or *unequal* involvement of both.* The whole lower half of the body may be affected if the inferior vena cava be blocked. The head, neck, arms and thorax are edematous if the superior vena cava is obstructed below the azygos veins; the head and neck alone, if the obstruction be above these veins.

Other specific causes of edema are trichinosis, beri-beri, and ordinary multiple neuritis. The blue edema of hysteria is a rare condition allied to or identical with the angioneurotic form.

The cachectic edemas, as seen in leukemia, scurvy and pernicious anemia, are usually of the mild cardiac type and, as previously stated, in terminal chronic Bright's disease of the interstitial type the edema is distinctly or predominatingly cardiac. Secondary cardiac edema.

LYMPHANGITIS AND PHLEBITIS.—Acute Septic Forms.—Both acute lymphangitis and acute phlebitis, if severe, may be associated with edema and occasionally great difficulty arises in the differentiation of the two conditions. Confusion possible.

For that matter the two ailments may, and often do, co-exist and both depend upon the same etiologic factors in the form of pyogenic organisms.

Acute Septic Lymphangitis.—This is extremely common as compared with phlebitis and especially so in relation to infected wounds of the upper extremities.

Its cardinal features are: *first, red streaks running proximally in the direction of the tributary glands, associated with tenderness on pressure over these lines and tenderness and swelling of the glands draining the infected area.* The red lines.

In severe cases the edema is progressive, the swollen lymphatics *may* be felt as thin raised cords and irregular areas of redness occur at intervals over the lymphatic lines. Palpable lymphatics.

Acute Phlebitis.—*In acute phlebitis the glandular tenderness and swelling are usually absent or much less marked. The redness is less decided or altogether lacking, the vein if palpable is much larger than the lymphatic cords and the superficial venules are markedly dilated.*

If the resulting thrombus undergoes purulent transformation, embolic phenomena initiate a pyemic state.

The red areas of a lymphangitis may suggest erysipelas but lack the well-defined, sharply circumscribed, advancing, raised margin of that disease.

A migratory form of septic phlebitis is occasionally encountered which may be most baffling. Migratory phlebitis.

ASCITES.—Fluid in the peritoneal cavity may be part of a general dropsy as in heart disease, Bright's disease, or obstructive pulmonary conditions such Common causes.

* Such edema may coexist with that of the cardiac or renal type and persist after the heart or kidneys cease to be factors in its production.

In walking patients especially the unequal involvement of the two sides always suggest it.

as emphysema and fibroid lung, which have produced right heart insufficiency, or it may result from any form of chronic peritonitis, whether simple, tuberculous, or malignant. Furthermore, it may be caused by portal obstruction, whether due to disease of the liver itself, to thrombosis of, or to pressure upon, the vein.

Small and Large Effusions.—In tuberculosis, malignant disease and most of the cases associated with new growths, the effusion though always considerable is relatively small.

Massive effusions.

Occurring as a part of general cardiac or renal dropsy, or of portal obstruction, the exudate is ultimately large and produces a tense and markedly protuberant abdomen.

Shifting dulness and tympany.

This tends to broaden in the flanks if the patient assumes a dorsal recumbent position. The umbilicus is prominent and percussion reveals a dulness obeying the law of gravity, and hence, in the absence of limiting adhesions, shifting its seat as the position of the patient is changed.

Whenever and from whatever cause the peritoneal cavity contains any considerable free exudate, the fluid seeks the most dependent portion, the intestines float upward, and yield a tympanitic note which shifts posturally exactly as does the dulness, but to diametrically opposite locations.

Ascitic wave.

The patient being in a sitting or standing posture, a *fluctuation wave* may usually be felt as a shock to the receiving finger if the abdomen is sharply tapped upon one side just below the line of percussion dulness, while the finger-tips of the other hand are placed opposite. The ulnar surface of the hand of an assistant should be *lightly* applied in the median line between the

False wave.

percussing and receiving fingers to interrupt a *false vibration* in the wall otherwise indistinguishable from the true ascitic wave.

Effect of adhesions.

Sources of Error.—*Shifting dulness may be interfered with by adhesions which prevent the flow of exudate or hold intestinal coils in a fixed relation to the wall;* moreover, some time may be required in certain cases for the fluid to change its site, and several moments should be allowed to elapse before negative findings are reported.

Technic important.

Dipping.—Such effusion makes palpation of the spleen, liver or underlying tumors difficult and often demands that the palpating finger shall be *suddenly and sharply* depressed, preferably both during forced inspiration and at the end of forced expiration, the temporary displacement of the fluid often making the underlying structure palpable.

Small exudates.

The knee-elbow position is necessary for the detection of small exudates, the dulness then appearing in the very region most resonant in the recumbent patient.

In massive effusions with excessive tension the ascitic thrill or wave may be, but rarely is, absent. It may be difficult to obtain in small effusions, in the presence of adhesions or through an excessively fat or edematous abdominal wall.

Differentiation.—*Elaborate tables of differential diagnosis in relation to ascites seem to the author almost futile.*

Movable percussion dulness in the flanks and *bilateral dulness with central resonance* at once rules out *pregnancy* and *cysts*, whether *pancreatic*, *massive*

ovarian or hydatid, the signs being diametrically opposite. Encysted ascites may give rise to insuperable pre-exploratory diagnostic difficulties. *Meteorism, i.e.*, excessive tympanites yields a universally tympanitic note.

Hepatic Cirrhosis.—The ascites of portal obstruction, usually due to hepatic cirrhosis, lacks the associated edema of the face usually encountered in acute and chronic parenchymatous nephritis, though a variable degree of edema of the cardiac type may be present in the lower extremities in long-standing cases. Ascites without general edema often co-exists with emaciation, giving rise to the "*poached-egg belly*" which in the adult is usually associated with cirrhosis of the liver, portal thrombosis or a tuberculous or malignant peritonitis.

Edema slight.

"Poached-egg belly."

A big belly in a thin man invariably indicates a pathologic condition.

Character of Ascitic Fluid.—In hepatic cirrhosis, cardiac or renal disease, it is usually clear, straw colored, and of low specific gravity (1010 to 1015). It is but moderately albuminous (2 per cent. or less) and may spontaneously coagulate on standing. The color is slightly darker in cirrhosis than in the ordinary secondary forms. In tuberculosis and malignant disease it is of higher specific gravity, contains 4 per cent. or more of albumin, may be hemorrhagic and, rarely, is milky or turbid from fat or true chyle.

Characteristic of transudate

Exudate.

Chylous vs. Fatty Ascites.—The two latter conditions may oftentimes be readily distinguished by the size of the fat globules which in chyle resemble a closely packed field of cocci as shown on microscopic examination. Mere blocking of the thoracic duct may or may not cause chylous ascites. Rupture of the duct or of a chyle-bearing lymphatic may occur, or *filaria sanguinis hominis*, or malignant or tuberculous ulceration be present.

Milky fluid.

Chyliform Ascites.—In malignant or tuberculous disease especially, a milky ascitic fluid may be present which is not a true chylous ascites but rather an exudate attending a chronic inflammatory process and containing fat as a product of cell degeneration and disintegration. In such cases the exudate contains large numbers of such degenerating cells.

The false and commoner form.

Lactescent Ascites.—A curious milky, but not fatty, ascites is occasionally met with, under the same conditions as accompany chyliform ascites.

In such cases there is no separation into layers and the peculiar appearance is due supposedly to lecithin or some protein.

Occasionally an exudate occurs which cannot be distinguished from that of a true chylous ascites save by its relatively *very* slow reaccumulation after tapping and emptying the peritoneal cavity.

LYMPHEDEMA.—The chronic forms of edema due primarily or chiefly to lymph stasis involve also a persistent and more or less progressive proliferation of the skin and subcutaneous connective tissue of the affected members.

Its best known form is that known as **"elephantiasis."** This ailment is endemic and due to the presence of the *filaria sanguinis hominis*, and indeed is frequently encountered in countries infested with this organism (*filaria Bancrofti*).

Endemic form.

On the other hand, sporadic cases are not infrequently encountered which present the same clinical appearance but lack any specific etiologic factors.

Sporadic type.

Prominent symptoms.

In each variety the persistent and slowly progressive enlargement of the leg, the leathery, furrowed, uneven, varicose surface and the slowly increasing difficulty in locomotion are unattended by the cardinal sign of cardiac or renal edema, viz.—pitting on pressure.

FIG. 4.—Sporadic lymphedema. (*Courtesy of the late Dr. Burnside Foster.*)

FIG. 5.—Sporadic lymphedema. (*Courtesy of the late Dr. Burnside Foster.*)

One or both legs may be affected and in the endemic form a similar enlargement of the scrotum, penis, labia, or clitoris may occur or the same process may attack the arm, lip, or ear. (See under "Elephantiasis").

Only slight pain or discomfort is experienced in the non-filarial cases, but in those of the endemic type a considerable amount of suffering may arise.

Onset and exacerbations.

The endemic (filarial) form may come on suddenly with fever, redness, and swelling and such patients may be subject to recurrent attacks of the same type, each resulting in greater residual permanent enlargement of the affected part.

Palpatory crackling.

SUBCUTANEOUS EMPHYSEMA.—Save in malignant edema and glanders, this condition indicates the entrance of air into the subcutaneous tissue through a wound or rupture of an air-containing viscus, as in tracheotomy, cough, rupture of pulmonary alveoli, malignant ulceration of the esophagus, etc. It offers no difficulty in diagnosis because of the pathognomonic *tissue-paper-like crepitation* of the distended tissue under finger pressure.

SUBCUTANEOUS HEMORRHAGES.—These will be fully considered under purpura, and it need only be said that the discoloration of the effused

blood is persistent under pressure, that the spots undergo the same changes in color as an ordinary bruise, are not elevated usually, and vary greatly in size.

Petechiæ and Ecchymoses.—The term ecchymosis is applied to the subcutaneous hemorrhage *one-half inch or more in size* as in the ordinary bruise and, like the *petechia*, it may be regular or irregular in outline.

The petechia is less than one-half inch in area and often is pin-point or pin-head sized. This term is often incorrectly used to describe a point of redness which blanches under pressure such as the "rose spots" of typhoid fever. When complicating an exanthem, as in measles or smallpox, true petechiæ invariably indicate a severe type of disease. In chronic diseases they are ordinarily associated with a definite cachexia or a hemorrhagic tendency (see "purpura"), and in certain obscure infections they indicate sepsis and, especially, *malignant* or "chronic infective endocarditis." They should not be confused with the nodular swelling of erythema nodosum.

Incorrect usage of term.

In acute and chronic disease.

In ulcerative endocarditis the occurrence of petechiæ may be of great diagnostic value and in the "recurrent infective endocarditis" the presence of ephemeral, red, raised, tender cutaneous nodules appearing upon the extremities is of interest and of almost pathognomonic value.

Cutaneous nodules.

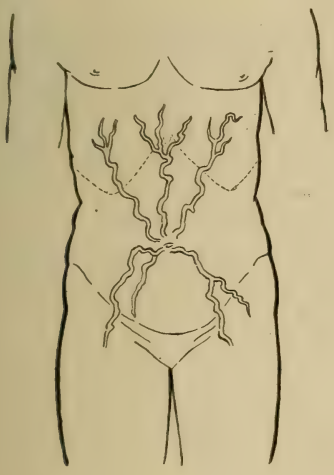


FIG. 6.—Collateral veins in a typical case of portal obstruction (*After Krause.*)

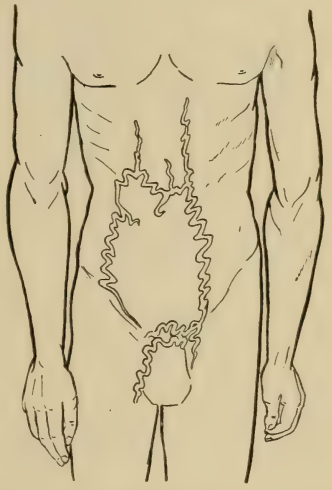


FIG. 7.—Collateral veins in a typical case of obstruction of the inferior vena cava. (*After Krause.*)

COLLATERAL VENOUS CIRCULATION.—Portal vs. Caval Obstruction.—Hepatic cirrhosis or thrombosis of the portal veins may produce a marked and evident enlargement of the superficial abdominal veins, and a similar condition occurs in thrombosis of the inferior vena cava. The former chiefly affects the median region of the abdomen; the relation of its veins to the navel suggesting the term "*caput medusæ*," and though the lower thoracic veins are involved, the group lies *chiefly* within lines dropped from the middle of the clavicle to the groin.

"Caput medusæ."

In the latter (obstruction of the inferior vena cava) the enlargement is predominantly lateral and usually less complicated in pattern. Unfortunately, admixture of the two may occur as in the case of a massive ascites of cirrhosis producing caval obstruction leading to enlargement of both groups of superficial veins, and furthermore the distinctions laid down above do not always hold true in other instances.

Visible dilatation.

Visible Internal Mammary and Intercostal Veins.—In any form of intrathoracic tumor, especially of the mediastinum, visible enlargement of the internal mammary and intercostal veins may be evident when the main venous trunks are blocked. If the superior vena cava is obstructed the blood flows downward to reach the ascending current in the inferior vena cava through the azygos veins.

Visible arterial pulsation.

Congenital Obliteration of the Aorta.—Obliteration of the descending thoracic aorta below the *ductus arteriosus Botalli* is a clinical curiosity, the patient usually, but not always showing a visible, pulsating, *arterial*, collateral circulation between the subclavian above and the vessels of the lower extremity below.

The pulse in the lower extremities is small and delayed, the aorta dilated proximally to the point of obstruction, and a diastolic aortic bruit and left ventricular enlargement, are usually manifest.

Lower Thoracic Veining.—The arborescent branchings, indicating more or less closely the peripheral diaphragmatic attachment and frequently observed on the walls of the chest, seem to be without definite clinical significance. The author has seen them most often in cases showing pleural adhesions, right ventricular dilatation or overstrain, cirrhosis of the liver, obstructed pulmonary circulation of the chronic type, chronic cough, and, not infrequently general arterial or myocardial degeneration.

Clinical relationships.

DRUG ERUPTIONS.—The iodides may produce acne-like or even varioliform or erythematous rashes; the bromides, an acne-like eruption; whereas phenacetin and its congeners, the balsams (such as copaiba), sodium salicylate, diphtheria antitoxin and various other sera, are capable of producing urticaria, or, other rashes, the more misleadingly simulating scarlatina or measles, in that a *febrile ailment may be coincident.* Many drugs produce rashes in certain individuals only, and these efflorescences are so varied as to preclude description.

Simulate many diseases.

Idiosyncrasy.

DESQUAMATIONS.—The chief diseases followed by desquamation are: scarlet fever (*lamellæ*), measles (*bran-like scales*), smallpox (*crusts*), erysipelas (*flakes*), dermatitis (*exfoliation*).

Important inferences.

SCARS.—*Study the history of scars.* The pits of previous *smallpox* may exclude that disease in some doubtful exanthem in a comatose patient. The scar of a carbuncle at the back of the neck may suggest *glycosuria*, and multiple linear scars at the angles of the mouth often follow *congenital syphilis.* Linear knife-cut scars on the left hand usually indicate *right-handedness* and *vice versa.* Small, bright, shining, slightly depressed scars on the chest may indicate the past use of *croton oil*, and grouped, finely linear, parallel scars over the lungs, heart, liver, or spleen, suggest *wet cupping* for

Wet cupping and the electric belt.

the relief of some pain or acute inflammation in the past, the nature of which may prove an important link in the case history. Scars at the waist line are common in those who have worn *electric belts*, and in the supraorbital region or intercostal zones may suggest respectively *supraorbital* or *pectoral zoster*.

Zoster, acne, and syphilis.

The tiny depressed scars of acne affect the chest, shoulders or face, and similar ones when generally distributed suggest *syphilis*, whereas cicatrices over the heel, sacrum and scapula suggest *bed sores* and hence past severe ailments such as typhoid. In elderly men an old time "seton" may have left its double scar at the back of the neck.

Bed sores.

Scars of Suicidal Attempts and Past Operations.—*Bullet and stab wounds* especially, may be suggestive in relation to past suicidal attempts, character, past associations, occupation and present disease.*

Neck scars.

Cervical scars, if anterior, long and linear, particularly if left-sided in origin and *transverse* in direction, suggest abortive *suicidal attempts*. Such are usually made by a razor and run obliquely downward from the angle of the jaw *across the median line*, or almost transversely across the throat, seldom following the line of operative incision of the anterior cervical gland chain or observing the limitations in extent and direction of the thyroidectomy incision, as would be the case with the somewhat similar scar of a past operation.

Glandular tuberculosis.

Puckered, depressed irregular scars in the cervical triangles, especially the anterior, suggest the slow healing of once broken down, discharging, *tuberculous glands;* while fine, long, linear cicatrices suggest radical operation for their removal, when they follow the known direction of the glandular chain.

Groin scars.

Irregular depressed scars in the groin do not suggest past syphilis but rather chancroid.

Common operative sites.

The scars of operations for appendicitis, ovariotomy, gall-stones, gastric ulcer, and the like are but too common nowadays and too characteristic to merit special description. All abdominal scars, however, demand an examination of that part of the abdominal wall incised in order that any hernial tendency may be detected.

Valuable when distinctive.

Luetic Scars.—There is not much of positive differential or diagnostic value in syphilitic scars save in certain situations or in certain forms of eruption, but when present and sufficiently characteristic they are of great assistance.

In general, one may say that syphilitic scars may be round, reniform, oval or horseshoe-shaped, and are smooth and seldom traversed by fibrous bands except at joints.

Lupus scarring may closely simulate syphilis, but is not multiform. Frequently there are multiple punctate depressions, and some old luetic scars are pliable and have a brownish-red areola (see also "Syphilis"). Of special

* In a person of a misleadingly clerical appearance and pretensions, such a wound proved to have been the result of a drunken brawl in a bawdy house of which he was the proprietor, and led to an investigation of past and present habits that proved illuminating with relation to his obscure ailment.

significance are the scars indicating a primary lesion (usually, of course, upon the genitals), and those of a destructive ulceration of the soft palate.

Drug habit.

Epithelioma and Hypodermic Marks.—The scar of operation for *epithelioma* of the lips or nose is especially suggestive, and *bluish marks* from an old needle coated with oxide of iron, or similar marks together with evidences of repeated superficial infection, may suggest confirmed morphinism or cocainism. Many victims of drug addiction use a dirty needle and in its introduction disregard clothing when circumstances render its removal inconvenient. In right-handed persons of this type the opposite arm and the right thigh are often characteristically scarred.

THE HEAD.—Sutures and Fontanelles.—*Open sutures persisting after the ninth month suggest hydrocephalus, cretinism or rickets. The posterior fontanelle should be closed at the end of the second month; the anterior at the end of the second year.*

Delayed closure with a gap of unusual size suggests hereditary syphilis, rickets or cretinism. **Bulging fontanelles** occur in chronic hydrocephalus and in meningeal hemorrhage or inflammation, but slight prominence and pulsation may occur in any child suffering from febrile ailments and is negligible. A sunken fontanelle is present in wasting diseases and spurious hydrocephalus.

Cranio-tabes, indicated by a soft "egg-shell" crackle on pressure over circumscribed occipital areas or over the posterior portion of the parietal bones may occur in rickets, congenital syphilis or hydrocephalus.*

The pulsating congenital tumors of variable size bulging from the sutures are classified as hydrencephalocele, meningocele or encephalocele.

Tumors and nodes.

Wens or cysts are common in the scalp and should not be confused with the deeper seated *immovable* syphilitic nodes representing a gummatous periostitis. In the latter case any co-existing brain symptoms suggest similar growths on the inner surface, their consistence being soft and doughy and nocturnal exacerbations of any attendant pain a prominent feature.

Caput quadratum.

Rickets.—A square or oblong head with flattened elongated vertex; a high, square forehead, often with frontal and parietal protuberances, when it surmounts a small face, indicates severe, classical rickets. It is usually associated with delayed closure but no bulging of the fontanelles, together with a sweating forehead, muscular weakness, mental retardation, delayed growth and beading of the costo-chondral articulations especially the fifth and sixth (*"rickety rosary"*). Epiphyseal swelling and thinning and palpable crackling of the postero-inferior aspect of the parietal and occipital bones (*cranio-tabes*) are often demonstrable.

The rickety rosary.

Cranio-tabes.

Deformities of the chest, legs and spine are common and the restless little sleeper may have rubbed away the hair at the occiput.

Hydrocephalus.—If the child's head be large but *globular or pyramidal* rather than square, the face relatively small, the space between the eyebrows prominent, the fontanelle large and *bulging*, the sutures separated and the external veins visibly distended, a history of excessive cranial growth or congenital deformity will be obtained and hydrocephalus is evident.

* The same "egg-shell" crackling is encountered in osteosarcoma of the long bones.

Oxycephaly. (*Tower Head*).—The head of excessive height above the ears and running to a narrow or pointed vertex is occasionally encountered. The supra-orbital ridges are poorly developed and exophthalmos and optic neuritis may result from increased intracerebral pressure.

The ailment is due to premature closure of certain of the sutures.

Congenital Syphilis.—Cranial asymmetry, exaggerated frontal eminences, keratitis, a saddle-nose, or a perforated palate may co-exist with "sore bottom" in the luetic child, and, with the exception of the last, even as isolated symptoms are of great diagnostic significance.

Typical syndrome.

Osteitis Deformans (*Paget's Disease*).—A marked increase of the head circumference without facial involvement may be associated with the *curvature* of the *enlarged* shafts of the long bones characterizing the disease.

Deformity of long bones.

Leontiasis Ossea.—This frequently produces marked deformity of the skull by osteophytic deposit particularly in the frontal regions and is associated with massive orbital rims and exaggerated malar prominences.

Leonine brow.

Facial Hemiatrophy.—Slight unilateral differences may be merely degenerative stigmata, but in true hemiatrophy the face is mesially divided into distinctly different halves, the extreme and apparent emaciation of the one side contrasting sharply with its better-nourished fellow.

A curious contrast.

In microcephalic idiocy the skull is extremely small and narrow.

THE EYELIDS.—Dark circles or duskiness under the eyes most marked in brunettes are commonly but often mistakenly construed as indicating pelvic disease. Though frequently an indication of such conditions, and often present as a transient condition in young women during or at the onset of a menstrual period, they may be constantly present in certain normal women and especially in olive-skinned brunettes. In both sexes and at any age they may accompany extreme prolonged pain, insomnia, overexertion, weak heart, or exhausting diseases.

Puffiness or edema of the lids may indicate mere recent weeping, trichinosis, disease of the accessory nasal sinuses, Bright's disease, arsenical overdose or chronic poisoning, anemia, or pertussis. Unilateral swelling may be due to angioneurotic edema or to actual inflammations, such as boils, insect bites, erysipelas or glanders. Associated with exophthalmos, it may indicate cavernous sinus thrombosis or tumor pressure upon the ophthalmic veins.

Trivial or important.

Seek simple causes first.

Inflammation of the lids may be due to conjunctivitis, simple or specific, and is a common manifestation in *measles*, coryza, iodism, hay fever and eye strain. Styes, chalazion, warts, epitheliomatous or syphilitic ulcers, gouty tophi, lachrymal cyst, or obstruction with tear overflow, and *blepharitis marginalis* need no extended description. *The injected conjunctivæ of measles is an early and suggestive symptom.*

Red eyes of measles.

THE EYES.—Many valuable inferences may be drawn from careful inspection of the eyes, and the expression may indicate bad habits concealed, or give a valuable clue to the temperament and mental status of the patient. Study of the background is of course a matter for special investigation.

Ocular Palsies.—Paralysis of the ocular muscles alone as indicated by ptosis or strabismus at once suggests *syphilis*, brain tumor, locomotor ataxia, meningitis or profound toxemia.

Nystagmus is an involuntary lateral, rotary or vertical movement of the eyeball, and in the absence of *hysteria, extreme refractive errors, albinism, cataract or corneal opacities*, suggests disseminated sclerosis, Friedreich's ataxia, tumors of the cerebellum or pons, terminal locomotor ataxia, basal meningitis, chronic hydrocephalus, or epilepsy.

FIG. 8.—Exophthalmic goiter. Typical facies.

Sign of slight value.

The Arcus Senilis.—This is indicated by a white line surrounding wholly or in part the corneal margin and is a symptom of slight specific significance when seen in persons beyond middle age, as it is merely a local indication of a general senile degenerative process. In younger persons it is of much more significance and may be associated with definite cardiovascular symptoms.

Bilateral vs. Unilateral.

Exophthalmos.—In most cases this is bilateral and associated with exophthalmic goiter, both eyes being unduly prominent and producing a peculiar staring expression, striking and easily recognized.

Unilateral exophthalmos usually indicates a new growth, abscess, or tumor of some kind in close relation to the affected eye, thrombosis of the cavernous sinus or hemorrhage into the orbital tissue.

Wasting diseases or atrophy.

Enophthalmos.—This is the opposite of exophthalmos and ordinarily accompanies collapse, wasting diseases, severe hemorrhage or persistent profuse diarrheas. Unilaterally it is found in hemiatrophy or lesions of the sympathetic nerve.

FIG. 9.—Thrombosis of right cavernous sinus. Edema of right side of face and right unilateral exophthalmos. (*Gordon.*)

Corneal Opacities.—These indicate, as a rule, syphilis or tuberculosis, but they may result from direct corneal injuries of any nature.

Important associations.

Cataract.—In many cases this disease of the crystalline lens is associated with diabetic manifestations or seems to reflect a general degenerative process, and should suggest a painstaking investigation of the heart, blood vessels, and urine. Juvenile cataract is frequently associated with struma or congenital syphilis.

The Dry and the Moist Eye.—If the eye be permanently or for long periods uncovered by the lids, as in profound collapse, it becomes glazed and dry, or even seriously inflamed as in rare cases of exophthalmic goiter with

extreme protrusion of the eyeballs. On the other hand, profuse lachrymation may accompany irritation of the conjunctiva or result from a mere photophobia. Actual persisting overflow (epiphora) may be present if the lachrymal duct is blocked or markedly displaced from any cause. Epiphora.

THE NOSE.—Saddle-nose is discussed elsewhere. A coarse, broad organ is seen in certain strumous types and in pituitary disorders, myxedema and cretinism, or may be purely racial or familial. Its base may be broad and shapeless in adenoid disease and nasal polypi, and it may be the seat of distressing and humiliating vascularity even in the temperate individual. Such innocent redness is most frequently due to exposure to the elements, gastric and pelvic derangements, cardiovascular disturbances, or chronic obstructive nasal catarrh. The alæ may carry a tuberculous or epitheliomatous ulcer and are frequently involved in the herpetic manifestations of certain acute diseases. Misleading redness

False Saddle-nose.—In achondroplasic dwarfs (see under "hands") the broad-based pug nose often suggests the true saddle-nose of syphilis.

No confusion should arise and the condition as seen in *achondroplasia* is due to precocious calcification.

Offensive nasal discharge suggests atrophic rhinitis, syphilis, necrosis or impacted foreign bodies.

Sneezing needs no special discussion though occasionally a troublesome phenomenon.*

Working Alæ.—In neurotic individuals this is commonly observed and it also constitutes one of the signs of marked dyspnea.

THE EAR.—Earache.—In infants the condition may be indicated only by crying, restless movement of the head or rubbing of the ear, and in them, as in the case of the profoundly toxemic adult, who may give no evidence of pain, an acute suppurative otitis media may be entirely overlooked until the discharge appears. Otitis often overlooked.

The condition is a common one in acute infections, such as in tonsillitis, pharyngitis, scarlet fever, influenza, typhoid, diphtheria and measles, and its complications are so serious (mastoiditis, meningitis, brain abscess, etc.) as to demand careful watching in such cases.

Earache and deafness may also be due to impacted cerumen, abscess or ordinary furuncle of the meatus externus, simple catarrhal inflammation and blocking of the Eustachian tube, decayed teeth and alveolar abscess, foreign bodies, neuralgia, and rarely cancer of the tongue.

Hematoma Auris.—Bruising and swelling of the pinna is frequently encountered in the insane.

Cyanosis and anemia may be reflected in the color of the external ear. It is the most frequent situation for frost-bite and may be the seat of ochronosis (blue-black cartilages), persistent localized gangrene† and gouty tophi (gritty sodium urate nodules near the margin of the pinna). Ochronosis, gangrene and tophi.

* In one instance known to the author violent and persistent sneezing was excited by normal sexual stimuli.

† In a case of drug habituation a recurrent superficial gangrene of years' duration affected the ear alone and ceased permanently only when the victim, who refused all treatment directed to a cure, stopped the use of the hypodermic and took his morphin by the mouth.

Significance of discharge.

Discharges of various kinds may be noted, slight and serous if due to meatal eczema; purulent if from suppurative otitis media or meatal abscess; clear and serous or primarily bloody in fractures at the base of the skull. Hemorrhage from the lobe is not uncommon in hemophilia even without wound or demonstrable abrasion.

Edema over the mastoid process and pressure tenderness are important signs of mastoid disease though occasionally due to local periostitis.

Herpes and mucous patches.

THE LIPS.—The grouped vesicles of herpes common in coryza, pneumonia and malarial fever, the mucous patches or even *the initial lesion of syphilis*, epithelioma, fissures, and, in badly nursed profoundly prostrated patients, *crusts and sordes*, are some of the important conditions to be noted. The color of the lips is a valuable sign of cyanosis and a less constant and specific one of anemia.

Sordes.

Epithelioma usually occurs at or beyond middle age, ordinarily affects the lower lip, grows indolently and exists for a long time without marked involvement of the neighboring glands.

Syphilis.—A labial lesion of syphilis is *promptly* followed by glandular swelling. Mucous patches and syphilitic fissures tend to involve the angles of the mouth and leave linear radiating scars, but ordinary "cold cracks" or fissures are of no significance.

Acute swelling of the lips commonly indicates trauma, insect bite, angioneurotic edema, simple abscess, or, more rarely, corrosive poison, cancrum oris, erysipelas or phlegmon.

The Open Mouth.—Aside from paralysis or primary mental defects, the dry lips and open mouth are seen in coma, chronic hypertrophy of the tonsils, or nasal obstruction (usually due to adenoids), inflammation of the buccal cavity, the profound exhaustion of certain acute adynamic febrile conditions and the terminal stages of chronic disease.

The Odor of the Breath.—*In acute poisoning* this may yield information of the first importance and the odor of carbolic acid, the aromatic, bitter-almond aroma of hydrocyanic acid and the peculiar smell of ether, chloroform, laudanum and alcohol are readily detected. *In the terminal stages of diabetes mellitus* the fragrant fruity breath is most suggestive of an excess of acetone and diacetic and oxybutyric acids in the blood and hence of impending coma. *Phosphorus* yields an unmistakable odor.

The "fruity" breath.

Foul breath in itself most often indicates improper care of the teeth, constipation, chronic naso-pharyngeal catarrh or local disease of the buccal cavity and pharynx, or, more often, the naso-pharynx. The bad breath of chronic drinkers is usually associated with a furred tongue and chronic gastritis.

Uremia.—A peculiar heavy, aromatic odor is noticeable in the so-called "uremia" of acute and chronic nephritis or in the terminal stages of failing heart, and a peculiar cadaveric emanation is sometimes noticeable at the time of death from exhausting disease.

Cadaveric emanation.

THE BUCCAL CAVITY.—Pigmentation.—The pigmented areas of Addison's disease appear here and upon the lips and tongue, as do the tints of jaundice and cyanosis.

Exanthems.—In acute exanthematous diseases the buccal cavity may be the site of the earliest eruption, as in measles where small red spots with a tiny bluish-white center appear before the development of the cutaneous rash (**Koplik's spots**).

The papules and vesicles of varicella and variola may often be seen and the vivid redness of scarlet fever is somewhat characteristic.

Petechiæ.—In scurvy (scorbutus), purpura, terminal leukemia, hemophilia and other ailments associated with hemorrhage, both submucous and superficial bleeding may occur and the latter may assume a most intractable and even fatal form.

Moisture and Dryness.—As regards secretion, there may be either *dryness*, such as occurs in the administration of atropin, in mumps, or as the result of mouth-breathing from whatever cause, or, the opposite condition, *salivation*, such as accompanies certain of the acute inflammations of the buccal cavity or results from the overuse of mercury. Either secretory abnormality may be associated with hysteria. "Drooling," such as occurs in bulbar palsy, facial paralysis, diphtheritic paralysis, and idiocy, is not necessarily attended by actual increase of secretion.

Salivation and drooling.

STOMATITIS.—Inflammation of the buccal mucous membrane may be encountered in any one of six chief forms:

1. **Simple catarrh,** as seen in teething children, acute infections, gastro-intestinal disturbances or direct irritation.

This condition is characterized by redness, dryness and heat of the mucous surface with subsequent increase of mucous secretion, local discomfort upon taking food, and moderate fever. True salivary flow is diminished.

2. **Follicular or Aphthous Stomatitis.**—This indicates impaired general health and digestion, is common in children and not infrequent in adults, and is *characterized by small vesicles soon transformed into superficial small ulcers with an inflamed areola, often appearing in crops and involving chiefly the edges and the tip of the tongue, the deeper folds, or the inner aspect of the cheeks. Constitutional symptoms are identical with those of the catarrhal form but the process may be persistent or spreading and painful.*

3. **Parasitic Stomatitis** (*"thrush," "muguet," "soor," "mycotic stomatitis"*) is due to the *öidium albicans* seen readily under the microscope and indicates usually poor nutrition and uncleanly artificial feeding in infants.

It is characterized by small curd-like deposits appearing first upon the tongue, but tending to coalesce, spread, and, in extreme cases, cover the whole aural and pharyngeal surface. The patches are readily removed and leave no ulcerated or excoriated surface.

4. **Mercurial Stomatitis** (*Salivation*).—Either through occupational poisoning, idiosyncrasy or overdosing, mercury may cause profuse salivation associated with spongy swelling and even ulceration of the gums, loosening or loss of the teeth, or even necrosis of the jaw.

A metallic taste and fetid breath with tenderness of the teeth are usually the first symptoms, and patients under mercurial treatment should be care-

A useful procedure.

fully watched for increased flow of saliva and made to bring the teeth sharply together at each visit in order that tenderness may be detected.

5. Gangrenous Stomatitis (*Noma*).—This fatal ailment is rare, being seldom encountered save in the badly nourished and environed children of the slums, in whom it follows convalescence from acute fevers, chiefly measles.

A deadly disease.

It is characterized by the appearance of a sloughing, spreading ulcer usually on the cheek, rarely on the gum. The breath is fetid, there is high fever, profound prostration, delirium and diarrhea. Gangrene rapidly develops in the cheek and may involve adjacent structures to a frightful degree or appear in other portions of the body.

6. Ulcerative or Fetid Stomatitis.—This condition stands midway between the aphthous form and cancrum oris, occurring chiefly in children during dentition, rarely in adults, and at times seeming to be epidemic.

The gums are swollen and spongy, bleed readily and show linear sloughing ulceration. The process occasionally involves the tongue and inner surface of the cheeks and lips; the submaxillary glands enlarge and the constitutional symptoms are often severe and associated with extreme prostration and marked febrile reaction. A recurrent form and a membranous form occur.

Rarely, and chiefly in marasmic or scorbutic children, the process assumes a type suggesting noma, but ordinarily recovery occurs in a week or ten days.

The interesting stomatitis of "*sprue*" and "Trench Mouth" is fully described under the proper headings.

THE TONGUE.—The Coated and the Clean Tongue.—In many diseases, acute or chronic, whether affecting proximate or remote structures, the condition of the tongue may assist, slightly, diagnosis, prognosis and treatment.

Helpful but not dependable.

A lightly coated tongue may be found in health or may indicate fever, disease of the naso-pharynx, gastrointestinal disturbances, mere smoking, drinking, or the possession of bad teeth.

A heavy, pasty, yellow coating is usually indicative of naso-pharyngeal catarrh, catarrhal gastritis, or other disturbances of the digestive tract and is encountered in rheumatism, influenza, and certain other acute infections.

Abnormally clean tongue *may be, and often is, associated with gastric hyperacidity or even ulcer.*

The indented tongue.

In chronic dyspepsias of the asthenic type the tongue is flabby, often enlarged and laterally indented by the teeth.

In the typhoid state the heavily coated tongue is sometimes bright red along its margin and at its tip, which oftentimes shows a triangular red area.

The Brown Tongue and the Beefy Tongue.—In all conditions of *profound toxemia and exhaustion* such as septic conditions, severe typhoid fever and the typhoid state in general, the tongue tends to become *brown, dry and fissured,* is *tremulous* and is *protruded or retracted slowly* or only on command. Such a tongue or its congener, the *red, shiny, dry, "beefy" tongue* indicates a bad prognosis.

The tongue of exhaustion.

The "Strawberry Tongue" with its bright red swollen papillæ emphasized by a white pasty background is often an early sign of scarlet fever. A *unilateral coating* is sometimes present in hemiplegia, but may be due to bad teeth on the affected side.

Scarlatina tongue.

Ulceration and Fissures.—*These may be a part of the buccal inflammations elsewhere described and always suggests a careful examination for syphilis, gastrointestinal disturbances and disease of the teeth and gums.*

Tuberculous ulcers are surrounded usually by a zone of pallor and are themselves relatively pale, shallow and sharply defined. Like syphilitic ulcers they are rarely primary.

Tuberculosis and syphilis.

The luetic ulcers are usually like serpiginous fissures if secondary, but in the gummatous tertiary form may be deep and rapidly progressive. Suspected malignant ulceration always demands the preliminary therapeutic test for the exclusion of syphilis as, otherwise, differentiation may be extremely difficult.

Deep fissures along the edge or deep scarring of the surface suggest past syphilis as does a *smooth atrophy of the posterior surface.*

The tongue reflects more or less definitely the general pallor of anemia and the cyanosis of cardiac or pulmonary disease, and one may detect sometimes scars suggestive of the tongue-biting accompanying *multiple* past epileptic seizures. Such scars may also result from an existing glosso-labio-laryngeal paralysis. *In general, tongue scars are especially suggestive of syphilis.*

Important indications.

Unilateral atrophy, the tremor and jerky protrusion of marked alcoholism or actual delirium tremens, and the intermittent fibrillary muscle spasm associated with paresis, must not be forgotten.

Marked enlargement may be due to acute glossitis or to acromegaly and myxedema. **Tumors** must be thought of as well as the various acute conditions affecting the mucous membrane of the mouth.

Various pigment deposits may occur here as in other portions of the oral mucous membrane, the most marked being the deep brown, purple, or black deposits of Addison's disease, and the yellow tint of the inferior lingual surface in jaundice.

Miscellaneous Discolorations.—*A black tongue* usually indicates the use of iron, bismuth, or charcoal, and *brown discolorations* are most commonly due to the use of tobacco, licorice or chocolate, less often to laudanum and rhubarb.

The term *Nigrities linguæ* is applied to a peculiar form of black tongue due to the development of a yeast-like fungus *Cryptococcus linguæ pilosæ* upon its dorsum.

"Black" or "hairy" tongue.

The pigmentation of the prominent elongated, filiform papillæ has given it also the name "hairy tongue."

The corrosive acids either whiten the surface (oxalic, carbolic, sulphuric acids as well as ammonia and corrosive sublimate) or yellow it (chromic, hydrochloric and nitric acids). Caustic soda or potash and the nitrate of mercury redden it. The tongue may be the source of persistent hemorrhage in purpura or hemophilia.

Mineral acids and caustic alkalies.

Geographic tongue.

The term **"geographic tongue"** is derived from the annular red patches on the dorsum and edges, due to a painless desquamative process. These spread peripherally and, by coalescence, form eccentric areas with prominent margins.

Leucoplakia buccalis (*"buccal psoriasis," "leucoma," "ichthyosis"*) is characterized by non-ulcerative, smooth, white, firm, raised patches. **The smoker's patch** occupies the dorsal surface of the tip as a pearly yellow, or perhaps reddish plaque.

Movements.—Slow, hesitant protrusion and withdrawal are observed in the typhoid state. Tremor may be marked in the same condition as well as in paralysis agitans and, combined with fibrillary twitchings, in bulbar palsy, general paralysis of the insane and disseminated sclerosis.

Etiologic importance great.

THE GUMS.—Relation of Diseases of the Teeth and Gums to General Ailments.—The relation of diseases of the teeth, jaws, gums, and of mouth infections in general, to other ailments both local and constitutional, is of late receiving deserved attention and it would appear that these tissues are second only to the tonsils and accessory nasal sinuses in etiologic importance.

Certain cases of heart infection, chronic arthritis, nephritis, dyspepsia, general malnutrition and the like have been repeatedly traced back to mouth and jaw infections in the author's clinic and private practice.

Marginal redness is usually of little significance, being attributable to uncleanliness in most instances, or to dental caries. It is of some importance in young persons as an indication of tuberculosis and is present in some cachectic states.

Lead poisoning.

A grayish or bluish-gray line, composed actually of a series of dots and lines, about 1 mm. from the actual margin may indicate lead poisoning. Rarely a similar *greenish-blue* line is present in copper poisoning.

Swollen, spongy or bleeding gums commonly indicate mercurial stomatitis or conditions of great debility and exhaustion as in scurvy and various cachexias. In neglected cases of the typhoid type accumulation of oral

Sordes.

débris form the so-called *sordes*. *Dental abscesses, pyorrhea,* and various growths such as the malignant *epulis* also occur.

Around the base of the gums as well as in the more readily visible portions of the buccal mucous membrane may be seen the sharply defined, flattened,

Mucous patches.

moist, grayish, *"mucous patches"* in cases of *syphilis*.

THE TEETH.—As regards the period of eruption of the first and second sets of teeth, a wide margin must be allowed in both directions, an unusually early or distinctly delayed appearance being of no significance.

Early vs. Late dentition.

A greatly delayed dentition is usually indicative of malnutrition and often associated with distinct developmental defects.

The disturbances of "teething," while doubtless greatly exaggerated and usually negligible in well-nourished, breast-fed children, are nevertheless

Not negligible.

important and are accountable, directly or indirectly, for many "upsets" of infancy. Any doubting physician would be convinced of this fact if he himself should for a time suffer from similar inflammations from whatever cause.

FIG. 10.—First dentition. Illustrating the sequence of eruption of the temporary teeth. *A*, sixth to ninth month; *B*, eighth to twelfth month; *C*, twelfth to fifteenth month; *D*, sixteenth to twenty-fourth month; *E*, twenty-fourth month to end of third year.

A On the other hand, we should not adopt dentition as a blanket term to cover obscure ailments lest we overlook more concrete and serious conditions.*†

As in the case of the finger-nails, *transverse grooves or furrows on the teeth* indicate past severe illness; *dentated cutting margins* are common in malnutrition; and pitted teeth may be due to past stomatitis.

B **Early decay** is common in pregnancy, phosphorus poisoning, rickets, diabetes and other forms of severe malnutrition and the importance of good teeth and especially, *sound roots*, in connection with chronic dyspepsia and *many other conditions* is too little appreciated. Many obstinate and persistent gastric ailments are promptly cured by proper attention to the remaining teeth.

Often important.

C

In "status lymphaticus" the two central upper incisors may unduly exceed their lateral neighbors which are actually subnormal in size.

D

Pyorrhea alveolaris, associated with the presence of *entameba buccalis*, and such blind abscesses and peridental infections as are revealed only by the X-ray, are extremely important as hidden sources of chronic infection.

Foci of infection.

E

Hutchinsonian Teeth.—If the upper central incisors of the *second dentition* are peg-like, short, narrow, separated, with a single notch at the cutting edge and rounded corners, they constitute an almost pathognomonic sign of congenital syphilis.

True type.

Mere serration of the cutting edge, or even notching, is in itself insufficient for diagnosis, being encountered frequently in badly nourished, non-syphilitic children.

* The normal periods are, *in months*: *First dentition:* two lower central incisors 6 to 9. Four upper incisors 8 to 12. Four anterior molars and lower *lateral* incisors 12 to 15. Canines 16 to 24. Posterior molars 24 to 36. *Permanent set* (*in years*): *Molars*, first set 6; second set 12 to 15; third set 17 to 25; *Incisors* 7 to 8. *Bicuspids* 9 to 10. Canines 12 to 14. It follows that at the end of the first year there are 6 teeth; at the end of the second 16, and that the first dentition should be completed in two and one-half years.

† The late Frederic Forchheimer taught that "teething produces teeth and nothing more," and such statements, though exaggerated, have gone far to correct the tendency to ascribe specific, serious ailments to this common and usually trivial condition.

Caution

A false diagnosis of syphilis is a very serious matter and every case should be fully proven before any opinion is expressed to family members.

One may base tentative treatment upon his own assumption and the therapeutic test is still invaluable but extreme reticence is wise and necessary.

Progressive Separation of the Teeth.—This is encountered in cases of acromegaly associated with enlargement of the upper or lower jaw and constitutes an important sign of this disease.

FIG. 11.—Hutchinsonian teeth. Congenital syphilis.

Commonly misinterpreted.

Teeth grinding is a common symptom of adenoids, reflex irritation or gastric disturbance in children and is rarely due to the commonly accepted cause, worms.

Arched palate.

THE JAWS.—The high-arched palate, due usually to neglected chronic naso-pharyngeal obstruction, and so often associated with the neurotic temperament in the adult, is readily noted, as is the massive lower, or less often, *upper*, jaw of an acromegaly. The prominent square jaw and the receding chin are universally associated in the lay mind with firmness or the lack of it respectively, but such assumptions are subject to many exceptions.

Jaw spasm suggests tetanus, spasmophilia, hysteria, strychnin poisoning or irritation of cerebral origin. *Paralysis* may be noted in pontine hemorrhage, neuritis, brain tumor, or meningitis, and the jaw movement may be more or less mechanically impeded or pain-inhibited in such diseases as quinsy mumps, and trichiniasis.

Pain inhibition.

THE SOFT PALATE.—*Perforations* have been referred to previously as essentially pathognomonic of syphilis. *Paralysis* is usually due to diphtheria, less commonly to cerebral tumor, meningitis, caries and bulbar palsy.

The condition of the fauces and tonsillar areas is of great importance in relation to the exanthemata, but will be considered under diphtheria, tonsillitis and other headings.

Anesthesia of the soft palate and its uvula is a common and valuable indication of hysteria.

Lack of vigor.

THE HANDS.—The Handshake.—Aside from the stigmata of occupation, the hands afford much suggestive information. The flabby handshake itself often indicates a congenital lack of vigor or acquired weakness.

Tremor may often be felt as well as seen, and thus what seems like a mere formality may contribute to diagnosis and prognosis.

Persons of the asthenic or tubercular type often have hands of a peculiarly delicate texture, easily compressed and weak in grasp and the flexible slender hand of many such individuals sharply contrasts with the clumsier resistant one of the person of phlegmatic type and lacking such congenital stigmata.

The broad spade-like hand of myxedema, the bulbous terminal phalanges and incurved nails of chronic pulmonary disease (**pulmonary osteoarthropathy**), the clumsy hand of acromegaly with its bony hypertrophy, great

size and myxedema-like outline, as well as hands showing Heberden's nodes, gouty tophi, the deformities of arthritis deformans, or those of so-called chronic rheumatism may be readily recognized. Myxedema and acromegaly.

Fig. 12.—1. Heberden's nodes. 2. Syphilitic dactylitis. 3. Clubbed fingers. 4. Arthritis deformans. 5. Spade hand of myxedema.

The atrophy of neuritis and deforming arthritis is associated with a peculiar glossiness of the skin especially marked in the fingers. **In Raynaud's disease** the so-called "cold" or "dead" fingers are encountered and actual gangrene of the skin may result from that ailment, or from direct injury, severe frost-bite, neuritis, leprosy or senile endarteritis. "Dead fingers."

In achondroplasic dwarfs one may encounter excellent examples of the "*trident hand*" (see "*Achondroplasia*"). "Trident hand."

Wasting of the Hand.—Aside from neuritis, arthritis deformans, and mere disuse, a most significant and important wasting occurs in progressive muscular atrophy, syringomyelia, poliomyelitis, amyotrophic lateral sclerosis, and the cervical forms of chronic pachymeningitis.

Tender, Superficial, Transient Nodules.—As stated previously superficial, small, tender nodules constitute one of the most important signs of "chronic recurrent infective endocarditis" and appear both upon the hands and feet, affecting especially the dorsal, but occasionally both surfaces.

These transient tender nodules should not be confused with the relatively or absolutely insensitive ones so commonly seen after an attack of acute rheumatism, especially in children. These persist for weeks or months. Rheumatic nodules.

Wrist-drop.—This common symptom is due to paralysis of the musculospiral nerve as the result of direct injury or poisoning by lead or alcohol.

THE FINGER-NAILS.—*Chronic indolent ulceration* (onychia) surrounding the nail suggests syphilis or marked malnutrition but occurs also in tuberculosis, leprosy, syringomyelia, neuritis and chloral habit.

Brittle, striated, or split nails, with or without marked deformity, most frequently occur in connection with gout, peripheral neuritis, syringomyelia, and prolonged repeated exposures to the X-ray.

Fig. 13.—Clubbed fingers. Chronic pulmonary disease.

Most important to the case-taker at times are the transverse ridges indicating a past severe illness. These are easily noted, last for a period of six or eight months, and by the proximity to the matrix indicate approximately the date of the causative ailment. Register past illnesses.

Color indicators.

The nails are also color indicators for cyanosis and anemia and will show the ebb and flow of the capillary pulse in aortic regurgitation.

Nail-shedding occurs after injury to the matrix or may be the result of neuritis, locomotor ataxia, or syphilis.

THE ARM AND LEG.—Many of the conditions affecting these extremities, such as edema, pain and tenderness, are discussed under their appropriate headings.

Luetic nodes.

Hard, circumscribed, immovable, bilateral, non-inflammatory swellings, upon either the ulna or tibia, are usually syphilitic nodes, and the more diffuse, *bilateral*, painful enlargements of the tibia often associated with nocturnal increase of pain usually prove to be luetic when the modern tests are applied or antisyphilitic treatment is instituted. The smaller rheumatic nodules are described fully elsewhere.

Localized edema, heat, redness, and severe deep-seated pain over long bones and especially the tibia, suggests *acute periostitis or osteomyelitis* which, if neglected, may lead to death from acute sepsis or to chronic fistulous openings indicating secondary necrosis.

Rachitis and scorbutus.

Enlargement of the lower end of the radius suggests rachitis, and an exquisitely tender, non-inflammatory, *brawny induration over the shaft of the femur or leg bones in infants or young children* is an almost pathognomonic sign of infantile scurvy, an ailment often obscure in its other manifestations (see "Scorbutus").

Barlow's disease.

Varicose veins are common in the lower extremities and may be bilateral or unilateral. They are often associated in middle-aged or elderly persons with chronic indolent ulcers and may produce a misleading edema like that of cardiac weakness. *Such swelling, however, is, as stated previously, usually either wholly or predominantly unilateral.*

Unilateral edema with congestion or pallor, pain and localized tenderness, suggests phlebitis. *A perforating ulcer*, usually under the ball of the great toe, may be present in advanced locomotor ataxia or, more rarely, in diabetes.

Deep cyanosis or cold whiteness of the foot or toes suggests Raynaud's disease, frost-bite or the early stage of an *actual gangrene* which may be associated with injury, arteriosclerosis, massive embolism, diabetes, frost-bite, ergotism, Raynaud's disease and rarely, it is claimed, with exophthalmic goiter. Mottled, dusky redness and pain over the sole of the foot may indicate *erythromelalgia.*

Intermittent lameness.

The presence or absence of normal pulsation in the accessible peripheral arteries, such as the dorsalis pedis and posterior tibial, should be carefully noted, in connection with painful diseases of the lower extremities especially, as indicating the dysbasia arteriosclerotica of Erb (*intermittierende Hinken*),* obliterative endarteritis or incipient gangrene.

This condition, much more common than is generally believed, is fre-

* This interesting ailment is also known as "intermittent claudication" and "crural angina" and is associated with an arteriosclerotic process localized or predominant in the affected extremities.

quently overlooked in its earlier stages and the patient is treated for chronic sciatica or rheumatism without avail and to the neglect of the cardio-vascular system. Often overlooked.

It may never lead to gangrene but always suggests the presence of degeneration of the myocardium and thus demands that special attention be paid to the heart itself.

Enlarged glands in the groin or femoral triangle suggestive of syphilis, chancroid, Hodgkin's disease, leukemia, malignant disease, etc., should be noted, as well as the enlarged epitrochlear gland above the inner condyle of the humerus which *may* indicate a past or present syphilis.*

Transient nodular swellings usually painless and subsiding with bruise-like color changes are common in erythema nodosum and as stated previously, small, painful, tender areas occur in chronic recurrent septic endocarditis.

The BACK.—The chief deformities of the spine are essentially four in number:

1. **Lateral Curvature** (*"Scoliosis"*).—This is most frequent in young girls of asthenic structure and habitus who suffer from faulty position in standing and sitting, and from general muscular weakness. It also results from rickets, chronic diseases of the lung and pleura, paralyses affecting posture, and *mollities ossium*. In its ordinary form this deformity is seldom extreme and is usually remediable. Asthenic girls.

2. **Lordosis** or exaggerated normal curve is exemplified by the later stages of pregnancy and suggests that condition, abdominal tumors, ascites and pseudo-muscular hypertrophy.

3. **Kyphosis.**—If the sharply angled posterior projection of the spine is present, Pott's disease (spinal caries) or *mollities ossium* are suggested, though a simple dorsal *curve* occurs in rickets, debility and chronic emphysema.

4. **Immobility of the Spine.**—Temporary stiffness may be due to muscular strain or rheumatism, and chronic rigidity indicates arthritis deformans, paralysis agitans or Pott's disease. "Poker spine."

The curious **spondylitis deformans** is a rare ailment chiefly affecting middle-aged men and is a peculiarly localized rheumatoid arthritis. One form affects the spine alone; another commences in the hip-joints and extends upward to the spine and shoulder, the result being a rigid kyphotic spine alone or with ankylosis of other affected joints. Certain cases are associated with marked muscular atrophy and nerve root pains.

Tumors and Swellings.—A great variety are discussed in surgical and orthopedic works, and congenital *spina bifida*, abscesses due to caries, fatty tumors, bed sores and sacral edema are more or less frequently encountered.

THE JOINTS.—The following points should be determined: (*a*) *Presence, extent and character of any redness, swelling and deformity.* (*b*) *The position assumed.* (*c*) *Heat.* (*d*) *Tenderness.* (*e*) *Fluctuation or edema.* (*f*) *Degree of mobility in relation to pain excitation.* (*g*) *Presence of crepitus.* (*h*) *Outline of bony structures.* (*i*) *Ankylosis.* (*j*) *Atrophy.* (*k*) *Contractures.* (*l*) *Associated or antecedent injury.* (*m*) *Acute or chronic*

*A most inconstant sign in old syphilitic cases.

infection, local or remote (tonsils, accessory sinuses, teeth, etc.). (n) Excessive exhaustion, exposure, fatigue or other recognized causative or predisposing factors.

General Comment.—*It should be remembered that in* **acute rheumatism** *the larger joints are chiefly and primarily affected, usually bilaterally, successively, and in a definite order, and that the skin is moist, the sweat acid, and fever and anemia marked.*

In acute gout a single joint, usually the great toe, is primarily affected, a preference is shown for the small joints, the actual pain and throbbing are greater and more definitely paroxysmal than in rheumatism in which they are chiefly excited by movement even of the slightest degree.

In chronic rheumatism deformity is, as a rule, not extreme, fever absent, changes of the weather markedly affect it and exacerbation and subsidence are the rule.

In arthritis deformans an acute primary attack cannot at first be distinguished from acute rheumatism, but it is more persistent, yields less readily to specific treatment (salicylates, etc.) and tends ultimately to become chronic, the joints grating on movement, deforming and finally becoming fixed. Both large and small joints are involved and the disease may become almost universal.* Many cases of arthritis deformans develop insidiously and exhibit no acute or subacute stage.

The Floating Patella.—In the knee-joint, fluid is most readily detected by fully extending and supporting the extremity and making downward pressure upon the patella which may be felt to tap against the articular surfaces. Ordinarily the outline of the distended synovial membrane in such a case is characteristic.

Sign of effusion.

Septic Arthritis.—This occurs chiefly in the course of, or immediately following, acute infectious diseases, especially scarlet fever and dengue, and is common in pyemia and gonorrhea. The infantile form of septic arthritis may occur without an assignable cause and progress rapidly to suppuration. This term is specifically applied to such cases as show a purulent joint exudate. The various *tuberculous lesions* are too numerous to permit of discussion in this volume.

Exudate is purulent.

Common Errors.—One of the most regrettable of diagnostic errors is involved in the failure to recognize *osteomyelitis* which all too frequently is treated primarily as rheumatism. Radical surgery is urgently needed in these cases and one should remember that the epiphyses are chiefly involved rather than the joint itself and that the disease is distinctly septic in type, the pain extreme, deep-seated and boring, and, further, that in osteomyelitis as in acute rheumatism several points *may* be involved, and furthermore, that a *secondary* pyemic arthritis may occur.

Fatal mistakes.

* In all cases of chronic affections of the joints, of a rheumatic or rheumatoid nature, one must consider the probability of their etiologic unity as now clearly indicated by recent investigations relative to the causative effect of chronic infections of the tonsils, accessory nasal sinuses, the teeth and gums and the prostate. Acute rheumatism itself is really a streptococcic arthritis, though the joint exudate is usually sterile, the peculiar streptococci being found in the subserous connective tissue, as first shown by Poynton of London.

In hemophilia, scurvy or purpura, both hemorrhagic and pseudo-rheumatic joint affections may occur. Hemorrhagic diathesis.

In locomotor ataxia and in syringomyelia, one of the large joints, most frequently the knee, ankle, or hip, may become suddenly swollen, with or without pain, and go on to rapid disintegration (*Charcot joints*). Charcot joints.

In any chronic joint affection, and as a result of prolonged disuse, there may be marked muscular atrophy simulating that of certain diseases of the spinal cord, but the electrical reactions are preserved. Atrophy of disuse.

Hysterical joints may create much confusion, being excessively tender and frequently contractured. *Chorea* is often associated with what is probably a true rheumatic arthritis. A confusing type.

Pain Referred to Joints.—Examples of misleading pain of this character are found in the reference of the pain of hip-joint disease to the corresponding knee; the shoulder-joint pain of *angina pectoris* or a circumflex neuritis; the misleading radiating pains of spinal caries, of aneurysms and of locomotor ataxia. Diaphysitis, caries of the ends of the long bones and especially osteomyelitis are most misleading. Misleading localization.

TREMOR.—Modes of Testing Tremor.—To distinguish between *passive tremor* and *intention tremor* the patient is asked to make some movement such as is involved in taking up and fastening a collar button, buttoning the vest, or lifting a glass of water to the lips. In the latter variety a tremor is increased, or indeed initiated, by the coördinate movements, and may be wholly absent when the patient is at rest. Effect of coördinated movement.

All tremors of the extremities are increased by extension; therefore the patient should stretch the arm or leg in front of him, keeping the fingers or toes separated as widely as possible in hyperextension. Again if the tips of the fingers are allowed to rest upon the physician's palm a vibration otherwise imperceptible may be readily detected. Extension increases tremor.

Facial Tremor.—Any latent or existent tremors of the muscles of the face may be emphasized by the firm closure of the eyes, elevation of the eyebrows, or by drawing the corners of the mouth down or outward, and tremor of the tongue is markedly increased by full protrusion. Action vs. Repose.

In the case of individual muscles or muscle groups in various portions of the body, putting these in action or under continued strain usually increases the tremor.

One should note whether the tremor in question is localized or general, passive or intention, coarse or fine, jerky, rapid or slow, regular or irregular, temporary or persistent.

Conditions with Which Tremor is Associated.—The tremor of *advancing age* and the extremely rare condition of congenital or inherited tremor are unimportant, as are those due to temporary *nervousness, muscular or mental overstrain* and the excessive use of tea, coffee or tobacco. Trivial tremor.

Tremor in acute disease usually indicates a profound toxemia, being one of the unfavorable symptoms, for example, in typhoid fever, and it is often encountered in the typhoid state whatever be the underlying original disease.

In alcoholism it is not only a prominent feature of delirium tremens, but may often enable the physician to detect a recent debauch or persistent excessive potations, and, if marked, it suggests the possible imminence of an acute attack. Such a tremor is ordinarily fine, regular and persistent, and very similar to that of *drug habituation.*

Rapid, fine tremor.

Exophthalmic Goiter.—In this disease the tremor is fine and rapid, and if associated with a large thyroid and rapid pulse with or without exophthalmos, the diagnosis is made. Every suspected case should be tested for tremor.

Coarse jerky tremor may be associated with cerebral lesions or disseminated sclerosis.

"Pill-rolling" and "bread-crumbling."

Paralysis Agitans.—This is ordinarily a relatively slow tremor, first affecting the thumb and forefinger and causing a rolling movement of the thumb over the forefinger known as the "pill-rolling" or "bread-crumbling" tremor. *It disappears during sleep* and *unlike intention tremor is checked by volitional movement.* In its more advanced form it may involve the arms, legs, and head.

Mineral Poisoning.—*Any case of otherwise unexplained tremor should suggest the possibility of chronic poisoning by some metal or drug, such as cocain, opium, arsenic, lead or mercury.*

Disseminated sclerosis.

Intention Tremor.—This condition suggests always disseminated sclerosis, but may be met with in a marked form as a senile tremor, occasionally in hysteria, and rarely in cerebral lesions, or even lead poisoning.

Tongue and lip tremor.

Tremor of Muscles of the Face.—*Fibrillary twitching or flickering of the facial muscles* strongly suggests profound asthenia, general paresis or chronic alcoholism. In progressive muscular atrophy, juvenile or spinal, it may be observed in other regions as well.

Tremor limited to the tongue and lips occurs in some cases of profoundly asthenic fevers and in general paresis.

SPASMS, CRAMPS, AND CONVULSIVE SEIZURES.—The term **cramp** should be reserved for a *painful*, spasmodic, muscular contraction, temporary or intermittent and localized. In the skeletal muscles it may be associated with alcoholism, gout, diabetes, Bright's disease, pyelitis, pyelonephritis, hysteria, or excessive muscular fatigue and most commonly involves the calf of the leg.

Scrivener's palsy.

Occupational Cramps.—The constant overuse of one set of muscles such as is met with chiefly in the occupational neurosis, is typified by the "scrivener's palsy" and may take the form of cramp, though more frequently it is a simple spasm, mere weakness or an actual paralysis.

Cramp Colic.—The term is quite correctly applied to painful muscular spasm of the viscera such as is involved in renal, intestinal, or biliary colic.

Wry Neck.—This may be congenital, traumatic or purely spasmodic and save for cases associated with actual traumatism at or subsequent to birth, represents a disturbance affecting the spinal accessory nerve. In true congenital wry neck there is atrophy of the sterno-mastoid (usually the right) and facial asymmetry, as opposed to mere muscle callus due to

rupture at the time of delivery. Spasmodic wry neck may be tonic, clonic, or more rarely combined, and may follow injury or exposure or be a true neurosis. In any event the muscle group of spinal accessory innervation is chiefly involved and the spasm of the rotators elevates the chin and swings it toward the unaffected side. The shoulder may be raised and the head drawn decidedly backward if the trapezius be much involved. Bilateral spasm drawing the head back and the face upward is rare. The clonic spasms are particularly distressing, and unfortunately the disease usually tends to become fixed.

Varieties.

Trapezius involvement.

One must remember the possibility that wry neck of the acquired type may be the result of an associated cervical caries, or a part of the phenomena of "spasmophilia."

Important warning.

Nodding Spasm.—(*Spasmus nutans*).—This condition is usually a neurosis, particularly, as seen in the adult. In children it may be mimetic, reflex (teething), or associated with rickets and, probably, with spasmophilia.

Ocular spasm, especially nystagmus and, less often, strabismus, may be associated with this condition. The oscillations cease during sleep, are increased by excitement or when under observation and associated mental defects or an epilepsy are occasionally noted. The reflex cases may be purely transient.

Nocturnal intermissions.

Tetany.—*This condition is probably one expression of the spasmophilic diathesis and is entirely distinct from tetanus.**

It is merely a painful bilateral and symmetrical tonic spasm of the *extremities*, of variable duration, tending to exhibit periods of exacerbation and occasionally intermit. It usually involves the feet and hands, less often the face, neck and jaw (*trismus*) and rarely affects the muscles of the trunk.

The disease is most frequent in infancy and the tendency diminishes with added years save for a disposition to intensification occurring at puberty.

Excessive muscular irritability is evidenced by the active contraction not only of the one, but sometimes of all, the extremities following pressure upon the main nerve trunks (*Trousseau's sign*), and even the lightest tap over the facial nerve causes contraction of the facial muscles (*Chvostek's sign*). The spasms may be intermittent, rhythmically recurrent, or persistent over considerable periods.

"Trousseau's" sign.

Chvostek's sign.

The attacks vary greatly in severity and duration and in infants and young children may last but a few hours or even minutes.

Duration of attacks.

Paresthesia may follow the lightest pressure upon a sensory nerve and *the response to the galvanic current is greatly intensified, both cathodal and anodal closure contractions being induced by 5 milliamperes of current.* Fever is sometimes present, and the disease is usually paroxysmal in type and associated with poor nutrition, *deficient calcium salts*, rickets, and marked gastro-intestinal disturbances.

Electric reactions of spasmophilia.

Calcium deficit.

Among the rare exciting causes of tetany are gastric lavage (in cases of

* A deficiency of calcium has been noted in this condition and oral administration is said to relieve it.

dilated stomach), pregnancy and lactation, and removal of the thyroid gland, or, more probably, as the result of coincident *parathyroid* resection.

Epidemic tetany.

An epidemic, non-fatal, form of tetany in adults has been quite prevalent at times on the continent of Europe, but is extremely rare in America.

Transitory cramp.

Paramyoclonus Multiplex.—This *syndrome* covers those peculiar cases occurring almost always in males and of unknown causation, in which *lightning-like clonic, with occasional tonic, spasms* of the larger muscles recur either persistently or paroxysmally, unaccompanied by sensory disturbances.

Often familial.

These are usually symmetrical and rhythmical and cease during sleep. It is often distinctly familial and in the type described by Unverricht either succeeds or accompanies epilepsy.

Myokymia.—This condition is characterized by continuous *fibrillary* contractions chiefly affecting the extremities. The affected muscles show a diminished response to both faradic and galvanic currents.

Myotonia (*Thomsen's Disease*).—This markedly but not exclusively familial disease is characterized by tonic muscle cramp induced by voluntary movement but passing off as these are repeated.

Vermicular muscular contraction.

The myotonic reaction observed in these cases is a characteristically slow contraction and relaxation upon stimulation by either the faradic or galvanic current; a deliberate vermicular muscle-contraction wave passing from cathode to anode.

Athetoid Movements.—These curiously deliberate, writhing, twisting movements of the fingers and hands, more rarely of the feet and toes, may result from infantile palsy or follow hemiplegia in adults. They are slower and less jerky than the movements of chorea, with which they are sometimes confounded.

Myoidema.—One frequently notes a marked fleeting, localized contraction if a muscle be sharply tapped with the finger. This phenomenon is especially common in the muscles of the chest in connection with tuberculosis but is not a sign of diagnostic importance.

Of slight importance.

Catalepsy (*Auto-hypnotism*).—This extraordinary symptom usually indicates hysteria, but may be encountered in melancholia, dementia, brain tumor, meningitis and tetanus, or be produced by hypnosis of which it constitutes the second stage.

The patient appears asleep and the limbs show *a peculiar plastic rigidity* and will remain for minutes or hours in the position in which they are placed. It is often auto-hypnotism, the eyes are closed, rolled upward, the pupils contracted and sensibility to pain and special general sensation is abolished.

Journalistic standbys.

The duration of such attacks varies from a few minutes to several hours, but may endure for months in dementia precox or melancholia. In such cases the patients are kept alive by gavage.

Hysterical Seizures.—The actual study of cases is necessary to an understanding of the physiognomy and external manifestations of this extraordinary condition, but once a large number have been carefully observed the difficulties, in great degree, are removed. The topic is dealt with entire in its proper section.

INFANTILE CONVULSIONS.—*A tendency to easily induced convulsive seizures like laryngismus stridulus and tetany seems to be one expression of the spasmophilic diathesis at present believed to be largely dependent upon a deficit of calcium salts.*

Spasmophilic diathesis.

Attacks precisely similar to those of epilepsy or differing only in some particulars may be observed in both children and adults. In the former they may be associated with indigestion, general malnutrition (whether primary or secondary to disease), rickets, fever, as is so frequently seen at the onset of acute infectious diseases, rarely in congestion of the brain and quite commonly as reflex phenomena due to peripheral causes.

Malnutrition and infections

Cerebral disease.

Disease or irritation, such as dentition with its associated digestive disturbances, phimosis, diseases of the eye and ear, or possibly intestinal worms may be exciting causes. Convulsions accompany the larger number of cases of cerebral disease in children, and one observes occasionally in young infants from the time of their birth a decided though gradually diminishing tendency to convulsive seizures.

Reflex type.

Symptoms.—Aside from the usually premonitory symptoms, such as slight twitching, teeth grinding, restlessness or irritability the attack is precisely similar to that of epilepsy. The diagnosis in repeated attacks may depend upon their subsidence or disappearance after a cause has been found and removed, but the spasmophilic electric reactions are clarifying diagnostic elements.* The infant mortality from this source is considerable, and, unfortunately, a large number of true epilepsies commence during the first three years of life. Over a third of those investigated by Osler occurred in the first year.

Comment.—*The onset of an acute infectious disease, simple overloading of the stomach, or indigestion, are accountable for the excitation of the greater number of such attacks in predisposed children. If the condition is persistent we have to think not only of epilepsy but of the various diseases affecting the brain and its covering, of renal disease and of adenoids.*

Cause usually simple.

STATION, ATTITUDE, GAIT

Test for Static Ataxia.—Static ataxia is tested by having a patient stand with heels and toes together. Marked swaying (more than an inch or two of excursion) *increased upon closing the eyes* strongly suggest locomotor ataxia and is, in fact, usually associated with other evidences of incoördination, lost knee-jerks and a pupil reacting to accommodation but not to light.†

Locomotor ataxia.

In Ménière's disease or a lesion of the mid-cerebellar lobe the patient may sway violently and fall unless closely watched.

Ménière's disease.

In cerebellar lesions the ataxia of all types is extremely coarse but is much lessened in recumbency, nor is Rombergism so greatly increased

Cerebellar ataxia.

Rombergism.

* See "Tetany."

† One sufferer from locomotor ataxia aptly described his sensation as that of a "chicken on a clothes line."

upon closing the eyes as is the case in locomotor ataxia. *In ordinary vertigo and the pseudo-ataxia of hysteria, muscular incoördination usually disappears in recumbency.*

Decubitus (*Active or Passive*).—*In the bedfast patient, its chief divisions are the active and passive, dorsal and lateral, and it varies with the condition of the patient and the nature and localization of his disease.*

Sthenic vs. asthenic ailments.

Even a severe *pneumonia* does not always rob the patient of his active decubitus, but in severe *typhoid fever* the patient lies relaxed and helpless, must be moved from side to side, and occasionally lifted and straightened out to relieve a cramped posture or prevent his gravitating to the foot of the bed.

Voluntary shifts.

The terms dorsal and lateral explain themselves, but it is important to note whether the patient changes voluntarily from one to the other decubitus or, through weakness or because of pain excitation or increase, maintains a fixed posture.*

Abdominal lesions.

In appendicitis the patient usually lies on the back, often with the right knee drawn up, or, as in general peritonitis, with both knees so placed. *In severe abdominal colics* (intestinal, hepatic, renal, etc.) the knees are often drawn close to the belly and the *lateral coiled position* is common.

Variable decubitus.

In acute pleurisy the attitude is variable, the patient often lying on the affected side to limit movement and favor the vicarious respiration of the sound side. In others this attitude is primarily painful, but with the coming of an effusion and subsidence of inflammation it is the favored position.

Rigid neck.

Acute meningitis, if established, is characterized by cervical rigidity, retraction of the head and neck giving a distinctive appearance.

Fear of jarring or handling.

In severe rheumatic fever *and in certain cases of* **infantile scurvy and rickets** *as well as in early* **acute peritonitis,** *the patient lies in a dorsal position, and his whole expression may evince the liveliest apprehension, when his bed is approached or any effort made to examine or move him.*

ORTHOPNEA.—*In cases of heart disease in which the right heart has failed; in asthma whether spasmodic, cardiac or renal, and in severe emphysema, massive pericardial effusions, mediastinal tumors, or laryngeal obstruction, one is likely to find that peculiar combination of dyspnea and an obligatory partial or complete sitting posture which constitutes orthopnea.*

High diaphragm.

In cases where the diaphragm is pushed upward, as in massive ascites or severe meteorism, orthopnea may be present, though usually absent. *Consciousness and a certain amount of physical strength are necessary to the voluntary maintenance of this position.*

Acute form.

An **acute** *orthopnea is seen not only in asthmatic seizures, but with major angina pectoris, sudden cardiac weakness and infarction or edema of the lungs.*

Important Variant of Orthopnea.—In certain cardiovascular cases it may be impossible for the patient to resist the impulse to assume *suddenly* a sitting or even a standing posture, and, inasmuch as sudden death has

A double risk.

* In acute self-limited ailments like pneumonia we may sometimes observe a profoundly toxic, apparently comatose, patient change his decubitus from passive to active and feel that the ebbing tide has turned.

frequently followed the natural attempt on the part of the attendant to restrain the patient, both judgment and discrimination are required.

This condition is most frequently encountered by the author in coronary sclerosis, anginas and advanced aortic lesions. *In the terminal incompensatory stage of mitral lesions* there is often a peculiar listless rolling of the head from side to side, which, with the associated anasarca, blurred features, cyanosis and orthopnea, makes a characteristic picture. Modifications of this syndrome may persist for weeks and are ominous.

Herald of death.

In some cases of aneurysm, acute aortitis, asthma, extreme cardiac incompensation, or a malignant growth involving the mediastinum, the patient not only sits up, but must lean forward so as to rest the head upon the knees or upon some special support.

In several cases of Hodgkin's disease coming under the author's observation, rapidly enlarging glands within the mediastinum made this attitude obligatory for weeks before the patient's death, a special bed-table or arrangement of pillows affording support for the head and arms. *In diaphragmatic pleurisy* the patient usually sits inclined toward the affected side. Rarely, in dyspnea or dysphagia, due to the pressure of aneurysm or mediastinal growths, and, in some cases of vertebral caries as well, the prone position is that of election.

Prone position.

Most careful attention should be given to the attitude of the semi-unconscious patient with particular reference to paralysis, for the appearance and passivity of an affected member or side is oftentimes characteristic.

Common Conditions.—The curvatures of kyphosis, scoliosis, lordosis, and the compensatory spinal arching of emphysema are everyday matters. The wide "stance" of advanced locomotor ataxia and the similar attitude of osteitis deformans are also more or less characteristic.

Spinal curvature and "stance."

Emprosthotonus and Opisthotonus.—Spasmodic flexion of the body which makes its supports the forehead and feet (emprosthotonus) is less frequent than the backward flexion which makes the head and heels the sustaining points (opisthotonus). Either may occur in hystero-epilepsy, uremic convulsions, tetanus, cerebro-spinal meningitis and strychnin poisoning.

Associated conditions.

GAIT.—Necessary Maneuvers.—When a special examination is necessary the legs should be uncovered from the hips down and in women the night-dress or chemise may be brought forward between the thighs and fastened above.

The patient should walk briskly, then slowly, with the eyes open, then shut, stop abruptly, and also turn sharply at command and follow a rug border or crack at right angles to the previous line of vision.

Due allowance must be made for nervousness and careful watch be kept lest a serious fall occur in some ataxic patient.

Caution.

The limping gait suggests acute or chronic joint ailments, flat-foot, corns, etc.

A tilting forward of the body is noticeable in those merely "round shouldered," in paralysis agitans, extreme emphysema, kyphosis due to vertebral caries, and certain painful abdominal affections both acute and chronic.

Leaning backward or an actual lordosis may be due to spinal disease, advanced pregnancy, abdominal tumors, ascites or extreme obesity, and is a striking symptom of pseudo-hypertrophic paralysis and cretinism.

Locomotor ataxia.

Ataxic Gait.—*The gait of the advanced ataxic stage of locomotor ataxia* is striking, the feet being raised suddenly, jerked uncertainly forward beyond the ordinary limit and brought down heel first with a stamp. The patient keeps them well apart and must keep his eyes upon the ground, lest he sway or even fall. A sudden turning movement or an abrupt rise from the sitting posture is to him difficult or impossible. A patient may have the disease for years, however, before this *typical* gait appears.

Other associations.

An ataxic gait occurs in many conditions other than locomotor ataxia; and among these are Friedreich's ataxia, cerebellar tumor, syringomyelia, hereditary cerebellar ataxia, some cases of general paresis, and various intoxications affecting the cerebrospinal system, *i.e.*, lead, arsenic, alcohol, etc.

"Mowing gait."

The Spastic Gait.—*In this gait, characteristic of hemiplegia if unilateral, or some form of lateral sclerosis if bilateral*, the rigid leg moves stiffly, the foot drags, necessitating a tilting of the pelvis at each step, and the toes describe an arc ("mowing gait"), thus wearing down the sole of the shoe on its inner side.

Hysteric simulation.

In hysteria a simulated unilateral spastic condition may be observed but in most instances the foot is merely dragged and not swung inward.

Cross-legged Gait.—*Spastic contraction of the adductors sometimes produces a cross-legged "progression" also occasionally simulated in hysteria.*

Fig. 14.—Tabes. Station: legs widely separated, feet everted.

May be evident even when the ataxic gait is not typical or even very marked. (*Gordon.*)

The Steppage or Prancing Gait.—This is most often seen in multiple neuritis or peroneal muscular atrophy as a result of foot-drop, because of which the patient must lift the foot high with each step in order to raise the toe clear of the ground and avoid tripping. The result is a peculiar "*high action*," the patient appearing to step over constantly recurring, though non-existent, obstacles.

Foot-drop.

The Shuffling Gait of Senility.—This is a matter of daily observation and is associated with slowly progressive loss of strength and mentality.

The Choreic Gait.—When present, it is sometimes peculiarly like that of the school boy, who in the schoolroom clownishly stumbles or trips over his heel to attract the attention of his fellows, and it is seldom or never unassociated with other characteristic symptoms. Spasmodic adduction, extension, and outward rotation of the legs form a sequence which soon makes progression impossible if unrelieved.

A late symptom.

The Reeling Ataxic Gait.—This is especially characteristic of cerebellar (vermis) lesions, and differs in no essential respect from that of a drunken man. *A similar gait may occur in any condition accompanied by vertigo.* "Drunken" gait.

The Waddling Gait.—In this form lordosis is marked, the feet are widely separated, the pelvis and head of the femur are jerked forward at each step, the flexed knee is advanced, extended only after the foot is placed flat upon the ground, and the patient cannot stand on tiptoe. It indicates bilateral hip disease, congenital dislocation of the hip-joints or pseudo-hypertrophic paralysis.

Dromedary Gait.—In the so-called "progressive torsion spasm" of children (Flatau-Sterling) the peculiar movements in walking have given rise to the terms "camel's walk" or "dromedary gait."

The Festinant Gait.—When present this is pathognomonic of advanced paralysis agitans, and, in typical instances, the patient seems as if trying to recover from a thrust from behind. He goes slowly at first; then trots or shuffles at increasing speed with the body bent forward and finally pitches helplessly forward, if unsupported. Retropulsion and lateropulsion of the same type may be present also in such extreme cases.

Intermittent Claudication.—(*"Dysbasia angiosclerotica," "intermittierende Hinken"*). This justifies further special reference, for though it is associated definitely with arteriosclerosis and deficient circulation in the lower limbs, it is frequently overlooked or misinterpreted.*

It is characterized by numbness, pain or muscle cramps on standing and walking, associated with a temporary rigidity, paresthesia or disability, all of which symptoms are often entirely absent when at rest. Not rare if sought.

The condition is far more frequent than is generally believed and in several cases observed by the author is has been associated with serious myocardial degeneration and general circulatory embarrassment. A significant finding.

The pulse is usually absent or extremely weak below the knee during the seizure and is usually deficient in one or both of the main arteries of the foot even when the patient is at rest.†

Thomsen's Disease.—*This, a family disease, is characterized by recurrent, transient muscular stiffness or painless contraction on attempting any muscular action.*

This, in walking, at first checks or delays each step, but gradually wears away and permits normal progression, only to re-appear when any new muscle group is called into play or the same action repeated after a rest. A curious complex.

Astasia-abasia.—This curious condition must be classed as a functional neurosis inasmuch as all other nervous functions are normal and the picture Effect of posture.

* The same condition may be present in the arm in rare instances.

† This condition need not be confounded with the **intermittent spinal claudication** of Dejerine, which represents what, at first, is a mere sense of *fatigue* in one limb under exercise later in both and still later the development of a spastic paraplegia. Disturbances of the deep reflexes and the sphincters appear early, pain is absent, the pulse in the arteries of the foot is unaffected. The disease is, nevertheless, due to the same cause as intermittierende hinken, but the changes are localized in the arteries supplying the lumbo-sacral segments of the spinal cord.

strongly suggests hysteria. *Walking may be impossible yet all of its movements may be normally performed when the patient is lying in bed.* The inability may be absolute or partial, giving rise to what simulates spastic paralysis on the one hand or flaccidity upon the other.

"Jumpers."

Saltatory spasm is sometimes observed, the abrupt leaps being due to violent involuntary muscular contractions induced by the assumption on the erect posture.

DESCRIPTION OF NYLIC STANDARD TABLE OF HEIGHTS AND WEIGHTS

Dr. O. H. Rogers, of the New York Life, has constructed from the same figures, the graphic table shown on the opposite page.

"The short vertical lines marked 5′6″, 5′7″, 5′8″, and so on are the height lines. The diagonal curved lines, marked 20, 30, 40 and 55, are the age lines for men; the lines designated by the sign o- are the age lines for women. The intermediate ages fall at proportionate distances between these curved lines. The intersections of the age lines with the vertical height lines fix the normal weight-points. The weights at intervals of 5 pounds are recorded on the horizontal lines to the right of the diagonal curved lines.

"Thus, a man 5′6″ in height should weigh, at age twenty, 135½ pounds; at age thirty, 143½ pounds; at age forty, 149½ pounds; at age fifty-five, 153½ pounds. The weights for intermediate ages are found at corresponding intermediate points.

"A woman 5′6″ in height should weigh, at age twenty, 130½ pounds; at age thirty, 137¼ pounds; at age forty, 143½ pounds (the same as a man at age thirty). At fifty-five the weights of men and women are the same. In this way are found the normal weights for any height at any age.

"Now, let us suppose that a man 5′6″ in height and twenty years of age weighs 225 pounds; how much is he overweight? His normal weight is 135½ pounds. Passing from the normal weight-point horizontally to the right until we reach the vertical line marked 225, we find ourselves just outside the 65 per cent. line; such a person is thus found to be 66 per cent. overweight. Again, supposing the weight to be 100 pounds, we pass horizontally to the left to the 100 pounds vertical line; the intersection of our weight line with this vertical line shows him to be 26 per cent. underweight. In the same way the percentage of any given weight above or below the normal may be obtained.

"The vertical weight lines are 25 pounds apart, but for greater accuracy the intervening spaces are divided by broken lines into 5-pound subdivisions.

"Mistakes in the use of the chart will be avoided if the following steps are taken in their order:

"1. *Fix upon the height line.*

"2. *Locate the age-point on that line. The point thus found gives the normal weight for the height and age.*

"3. *If the case is very much over- or underweight—pass horizontally to the right in case of overweight, and to the left in case of underweight, to the intersection with the vertical weight line. The position of the point thus found fixes the per cent. over- or underweight.*"

Importance of weight.

WEIGHT AND HEIGHT.—*Every doctor's office should contain a pair of accurate scales, and the past, recent, present, and best weights are oftentimes of great importance in diagnosis, prognosis and treatment.*

Obesity.—*In general, one encounters a tendency to increase in weight after the age of forty and excessive corpulence is ordinarily associated with a weak musculature, poor resistance to acute disease and a decided tendency to degeneration of the heart, blood vessels and kidneys in middle age.*

Its many causes.

Emaciation.—This may arise from exophthalmic goiter, hysteria, worry and loss of sleep, the anxieties and strain of a love affair, improper or insuffi-

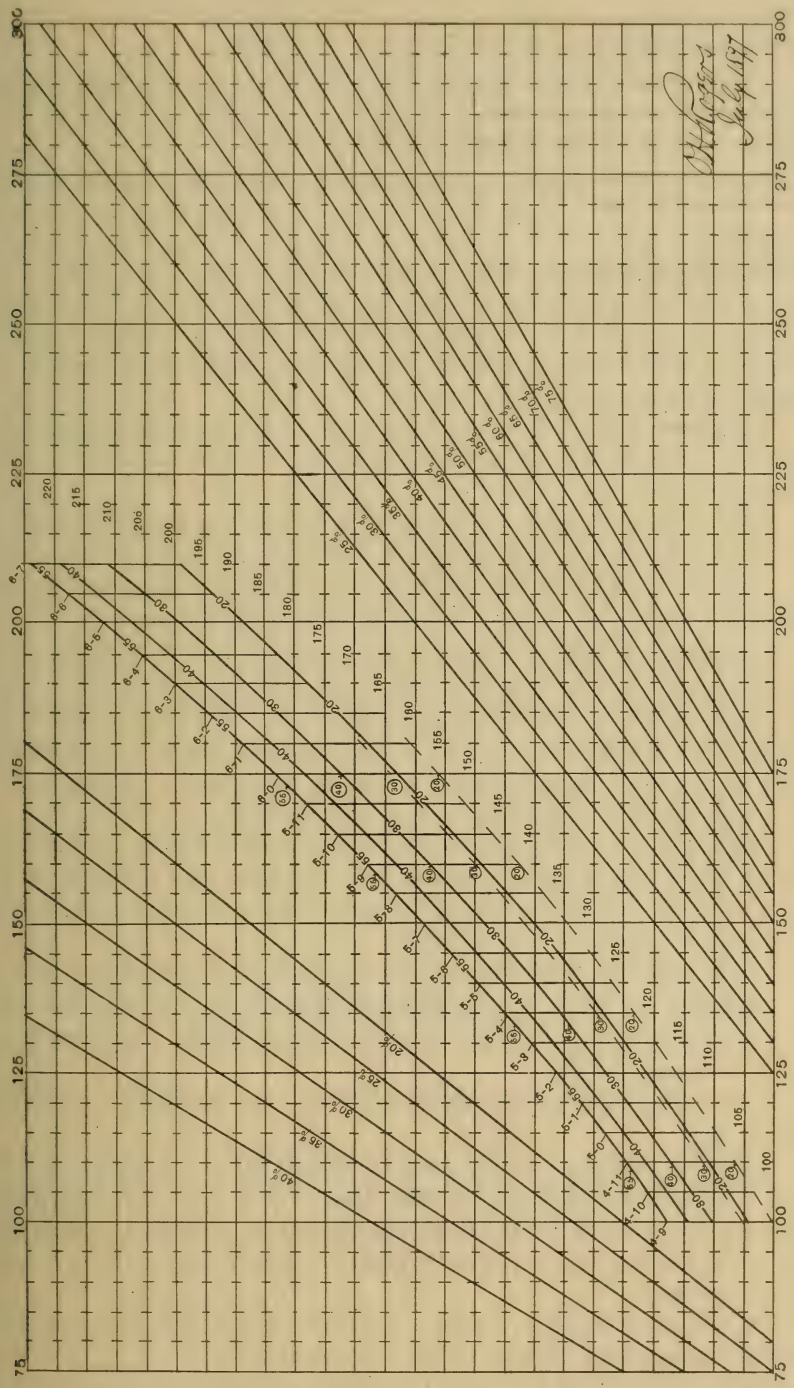


FIG. 15.—Nylic Standard Table. Heights and weights.

cient food, diabetes, chronic vomiting, profuse diuresis, sweating, purging and many other exciting causes.

It is pronounced in certain digestive disorders, in symptomatically active *universal congenital asthenia*, in all severe or prolonged fevers, true diabetes mellitus and the various wasting diseases of children.

Self-starvation.

One of the commonest factors is unconscious self-starvation through whimsical or mistaken limitation of the diet as is so often seen in the asthenic forms of indigestion.

Chronic starvation, unconscious, deliberate or imposed, is one of the commonest and least recognized causes of persistent dyspepsia of the functional type as encountered in asthenic individuals and some very serious cases arise from the vicious, dangerous, and not infrequently fatal, fasting cures conducted by quacks, or self-imposed.

A cause of "nervous indigestion."

Progressive Loss of Weight.—This is one of the most significant signs of incipient tuberculosis, carcinoma of the stomach and many other chronic ailments.

Wasting.

A fair estimate of weight loss can be made by pinching up a fold of skin over the triceps when the elbow is semiflexed and noting the degree of the misfit.

In chronic exhausting diseases and in the emaciation of advanced age the skin is not only loose, but also relatively inelastic and relaxed.

Increase of Weight.—*Following illness, this ordinarily indicates an improved general condition and the arrest of any local disorder, but large tumors, myxedema, edema and exudates, also increase it, and in heart disease and Bright's disease especially, the variation in body weight may reveal a transudate not shown by outward signs. Loss of weight in the renal and cardiac types of edema may at times, therefore, be a favorable symptom.*

Misleading gains in weight.

Weight loss may mean little or no loss of strength and in those who have become obese from physical inaction and overeating and drinking, may actually promote muscular activity, *but progressive emaciation and increasing weakness are usually of serious import.*

Weighing infants.

Infants should be weighed weekly and should show an increase of from 200 to 250 grams per week for the first four months, from 120 to 200 grams weekly for the five succeeding, and somewhat less for the remaining months of the first year of life.

During the first days after birth they may show normally a considerable weight loss.

Accurate Weighing.—When accurate results are necessary in individual cases, weighing should be done at the same hour in relation to meals, on the same scales, and with the same amount of clothing, if possible, inasmuch as the estimate of 5 to 7 pounds usually made for the clothing of adults is subject to grave error.

Height and weight.

The relation of height to weight is important and is well shown in the ingenious and accurate "*Nylic*" table. (Page 55).

The Attitude of Life Insurance Companies.—Insurance companies regard with suspicion men whose weight runs more than 20 to 25 per cent. above or below the standard as indicated by the table, and that this prejudice is well

Standard relationship.

founded has been shown by the results of the "combined actuarial investigation" which has proven an unduly high mortality in this class of lives.

Light Weight.—In connection with *underweight* a family history of tuberculosis or a personal history of doubtful environment, past infection, a narrow chest, small heart and the evidence of poor circulation are extremely important. The many etiologic factors, acute and chronic, naturally receive close scrutiny.

Demands careful scrutiny.

Overweight.—So also in *overweight* one must distinguish between those who have big bones, firm well-developed muscles and moderate abdominal girth and the flabby, big-bellied, sedentary livers and heavy eaters, particularly if these latter have a family history indicating an hereditary tendency to apoplexy and diseases of the heart and kidneys.

Necessary discrimination.

Comment.—*Very tall slender men, giants, professional athletes, and big-bellied heavyweights are not as a rule long-lived.*

AGE—RACE—SEX—HABITS—SOCIAL STATE AND RESIDENCE

AGE.—Age Estimation.—By practice and observation we can usually estimate closely the age of patients, but hair dye and modern beauty parlors may deceive the very elect among case-takers. On the other hand, manly chivalry may be a stumbling block and, in the case of women at least, like the tactful census enumerator, we may at times be obliged to accept statements with a mental reservation which should find a place in the case history.*

Stumbling blocks.

Certain ages represent a special predisposition to certain diseases, acute and chronic.

Diseases of infancy and youth.

In infancy and childhood we meet especially with acute digestive disturbances, spasmophilia, rickets, the exanthemata, affections of the lymph glands, meningitis, infantile palsies, epilepsy, chorea, croup, cretinism and congenital heart disease.

FIG. 16.—*Rapid aging.* An active energetic man aged forty-nine. (Compare with Figure 17, page 59.)

Tuberculosis attacks the young with an especial frequency and a resultant higher mortality, but in those under the age of puberty it is peculiarly liable to affect the lymph glands, particularly those of the cervical triangles, the bones, or the abdomen.

During adolescence, chlorosis, various forms of hysteria, epilepsy, acute rheumatism, gastric ulcer and tuberculosis are extremely common.

* In certain very rare instances the appearance of youth is maintained without the use of artificial beautifiers. The author recalls one woman, thirty-five years of age, who had the face of a girl of sixteen.

The degenerative period.

As middle age approaches, the tendency to the exanthemata and to acute infections is diminished and a predisposition to degenerative diseases of a chronic type appears. Such are arteriosclerosis, myocarditis, prostatic disease, gout, gall-stones, diabetes, the insanities, various forms of paralysis, profound blood disturbances such as leukemia and pernicious anemia, carcinoma, and chronic Bright's disease. Cardiovascular syphilis tends to become clinically prominent in middle age, even though the primary infection and insidious, initial degenerative changes may date back several decades.

In old age the predisposition to cardiovascular and malignant disease is intensified, and, in addition, we find chronic bronchitis and emphysema, passive pulmonary congestion, and a return to the childish tendency to broncho-pneumonia.

FIG. 17.—*Rapid aging.* Same man six years later.

In some instances diseases are wholly limited to certain age periods, but usually, in weighing diagnostic probabilities, *the question is one of relative frequency only.* For example, cancer of the stomach does occur in young people, but is as rare in a person under twenty, as is mumps in one over seventy.

Apparent vs. Actual Age.—*Two very essential points are (a) apparent as compared with actual age; and (b) unduly rapid aging.*

Rapid aging.

Rapid aging may bring the man of thirty to the same status as the one of seventy, and on the other hand, the latter, by reason of inherited vigor of constitution and right living may be physically twenty years under his actual age.

Extended youth.

Every clinician repeatedly encounters cases of marked arteriosclerosis in men under thirty and the inheritance of poor structural material, lues and dissipation or mental strain are the chief factors in early aging.

Age and Prognosis.—*Finally, one must remember the effect of age upon prognosis.* The acute exanthemata, though more readily acquired, are for the most part much more lightly borne by the child than by the adult and, on the other hand, resistance to tuberculosis is much more marked in persons above the age of thirty. Pneumonias are peculiarly fatal at the extremes of life, but "young old people" strongly resist the advance of chronic disease.

SEX.—In general, men and women suffer about equally and resist in about the same measure the larger number of diseases: nevertheless, there are certain striking differences both as to incidence and severity. As compared with men, women, through the disabilities peculiar to their sex, appear to develop a superior philosophy and endurance particularly marked in chronic disease.

Woman's predisposition and immunity.

The woman is especially liable to the multiform expressions of congenital asthenia, hysteria, myxedema, arthritis deformans, chlorosis and chronic secondary anemias, gall-stones, goiter, cancer of the breast or uterus, and gastric ulcer.

On the other hand, she is relatively free from hemophilia, aneurysm and locomotor ataxia, and less liable to acute infections, gout, appendicitis, typhoid fever, certain diseases of the heart, progressive muscular atrophy, diabetes, carcinoma of the stomach and bowels, leukemia, pernicious anemia and Addison's disease.

With respect to aortic disease, her so-called "immunity," in the light of recent research, appears to represent a less degree of exposure to that syphilitic infection now justly held accountable for the larger proportion of such cases as first become manifest in middle age.

RACE.—The special predisposition of the Hebrew to functional nervous ailments and diabetes mellitus; of the Irish to tuberculosis; of the English to gout; of the continental races to suicide, etc., etc., is well known. Moreover, certain races show markedly greater resistance to disease and surgical procedure than do others.

Scope of inquiry.

HABITS AND ENVIRONMENT.—*The term "habits" should often cover a much larger field than the patient's indulgence in tobacco, drugs, or alcoholics and include his environment and mode of life, his hours for meals and for sleep, his manner of eating and the nature of his meals, no less than the extent of his drinking.*

Specific inquiry.

In dealing with dyspeptics, one should ask specifically what was taken for breakfast or lunch preceding the time of the examination, or just what is usually taken at such a time, how much water is drunk and when, how much time is taken for the meal, whether the food is properly masticated, and, indeed, ascertain the condition of the teeth and gums by direct inspection when examining the tongue and throat.*

* As a matter of routine practice, the examination for infected tonsils, pyorrhea alveolaris, peridental abscess, antrum disease and other foci of infection, has become a prime necessity, because of the important part played by cryptogenic infections in the causation of disease.

Tea, Coffee and Tobacco.—*Too much stress is laid upon the mere use of tea, coffee and tobacco, and too little upon the idiosyncrasy of the patient in regard to these articles.*

Individualization necessary.

That which constitutes excess for one may be but moderation in another, and the slight amount representing the average consumption is usually of little consequence; hence it is a positive hardship to arbitrarily shut off, rather than to sensibly cut down, the tea, coffee or tobacco which for years has been the solace of some elderly patient.

Excess a relative term.

As regards tobacco, especially, one may say that the signs of *overindulgence* are found in unrefreshing sleep, furred tongue and bad taste in the mouth, nervousness which takes the form of mental irritation and perhaps tremor, and at times a distinct disturbance of digestion and irritability or palpitation of the heart. In the man under middle age these symptoms mean little; in the elderly man much, and for the latter, persistence in excess is fraught with danger.

Tobacco poisoning.

Its dangers.

Snuff Habituation.—The very extensive and steadily increasing use of "*Copenhagen*" snuff now is known to be attended in many instances by distinctly toxic symptoms.

It is used not only by "snuffing," but also, and perhaps more generally as a "quid" held persistently in the mouth in the gingival buccal angle.

"Snuff- or Copenhagen-heart" often differs in no essential respect from the irritable, labile and arrhythmic "tobacco-heart."

"Snuff-heart."

In addition one must consider snuff as a possible cause of obscure chronic indigestion, chronic headache, psychasthenic states, and actual exertion dyspnea due to cardiac insufficiency, usually of minor grades.

The Use of Alcohol.—The common mistake of the novice is a failure to bring out tactfully yet fully the facts bearing upon a patient's alcoholic indulgence.

Misuse of terms.

He is too easily satisfied when the patient says he is "not a drinking man," or he "takes an occasional drink," or "not enough to hurt him," "a drop now and then," "just a social glass," etc., etc. An absolute denial of overindulgence is usually proudly made by the man who has been a hard drinker for years but has stopped, it may be only a few days before examination. The "*now and then*" kind, or those who say "*often not for a year*," frequently represent the worst type of periodic spree drinkers.

Euphemisms of the bibulous.

One should, therefore, inquire tactfully about the present and the past, the extent, the hour, the kind, in what relation to meals and with what apparent effect upon the health.

Specific inquiries.

It is impossible to lay down an absolute rule as to what constitutes *excess* and it is probable that the man who takes a little liquor, even two or three times a day well diluted and on a full stomach, may suffer little or no bad effects, but the American style of drinking is peculiarly harmful as taking the form of "cocktails" before meals and of a multiplicity engendered by the pernicious custom of "treating."

What constitutes excess

A Genuine Risk.—*The danger in "moderate" drinking lies doubtless much more in the risk of forming a habit than in the physical damage wrought by the*

intoxicant itself, but the reality and insidious nature of this risk is but too well known to every practising physician.

Relation to disease.

Relation to Disease.—The three diseases most often due to such *over-indulgence* are: delirium tremens, alcoholic neuritis, and cirrhosis of the liver, but it is a powerful contributory factor in an enormous number of chronic diseases. It depresses vitality, diminishes resistance and thus invites and promotes their development and accelerates their progress. In severe acute diseases, such as pneumonia, the confirmed drunkard stands little show.

DRUG HABIT.—Opium and Cocain.—As regards the use of drugs, we have chiefly to deal with the various forms of opium and with cocain. *Neither produces in every individual a clear and definite syndrome, but nervous instability and a peculiarly baffling and bastard symptom-complex are suggestive.* A markedly dilated pupil in cocain users or a markedly contracted one in the victim of the opium habit may attract attention, but in either at the time of examination the pupil may be normal.

Bastard complexes.

Hypodermic marks.

If one examines such patients thoroughly, he will often find recent *hypodermic punctures*, black dots representing old ones, or evidence of recent or old multiple abscesses due to the use of dirty needles. Such are usually on the right thigh or left arm of a right-handed person, and the needle is usually introduced directly through the clothing.

Mental state.

The peculiar pallor sometimes present has been referred to elsewhere, the skin is likely to be dry in morphin users, the appetite poor and bowels constipated and the victims of either drug are subject to fits of profound depression succeeded by periods of buoyancy. "Nose rubbing" may suggest opium, and in the case of cocain users especially, formication is not an infrequent symptom. The excessive use of cocain may be honestly denied by one who has unwittingly contracted the habit through the use of the drug in the nasal passage or throat, the patient not realizing that absorption takes place in such instances as readily as if the drug were actually taken into the stomach. The overuse of such drugs as acetanilid may become a fixed habit and lead to serious or even fatal illness.

Innocent victims.

Clouded or Misty Case Histories and Physical Findings.—*Baffling and indeterminate histories and findings should particularly suggest the possibility of larval syphilis, drug habit, some abnormality of the internal secretions, mineral poisoning, hysteria, or actual malingering.*

MARRIAGE.—The question of marriage is important chiefly in relation to the matter of pregnancy or child-bearing in women and its after-effects. Many obscure ailments in the female are traceable to unrecognized or neglected lacerations, while endometritis, uterine displacements, pelvic abscess and epithelioma are especially common in multiparæ.

Pregnancy, childbirth and miscarriages.

The number of pregnancies, the duration and severity of labor, miscarriages, the stage of pregnancy at which they occurred, and the circumstances attending them, the number of children living and dead, the health of survivors and the causes of death of those diseased, are important.

Marks of syphilis in a child at once direct attention to the parents and

vice versa, and tuberculosis and other diseases may be suggested in a similar manner.

Bachelor vs. Benedict.—Insurance experience seems to show that the benedict is a superior risk and, on the average, outlives his bachelor fellow.

OCCUPATION.—Sedentary vs. Active.—Occupation and environment are important factors in diagnosis and prognosis and may throw light upon degenerative processes, functional and organic nervous disease and accidents. City dwellers and indoor workers in sedentary occupations suffer especially from dyspepsia, tuberculosis, and similar disorders. On the other hand, even country dwellers and out-of-door workers *may* suffer from the poor ventilation and deficient sanitation to be noted in many farm houses, and among them the transmission of tuberculosis from one family member to others is equalled only in the city slums.

City vs. Country.

Tuberculosis.

Exposure.—So also must one consider the effect of unusual exposure to cold, wet, and to such poisons as malaria.

The best conditions are to be found in camp life or in the homes of the better class of agriculturists, the worst in the slums, sweat shops or improperly ventilated factories of great cities. On the one hand, there is the maximum of fresh air, sunshine, and healthful exercise; on the other, foul air in the shop and home, constant use of the same muscles and in cramped positions, and, as a rule, deficient or improper food.

Best and worst conditions.

The Fatigue Neuroses.—Certain occupations involving the continuous use of one neuro-muscular unit produce specific ailments as do those involving pressure, or irritation of a particular portion of the body. Cases of *nystagmus* have been reported as due to the cramped position of miners working with eyes fixed upon a particular point.

Miner's nystagmus.

"Penman's cramp" or "scrivener's palsy" is a familiar example, and the *chronic laryngitis* of military men, clergymen, auctioneers and public speakers falls under the same head.

Among the commonest examples of these localized impairments of co-ordination are: "telegrapher's cramp," "dancer's cramp," "piano-player's cramp," "tennis-player's cramp," and "seamstress's cramp," but the list might be extended indefinitely.

Another example of occupational disease is the inflammation of the patellar bursa especially common among floor scrubbers and known as "*housemaid's knee.*" This condition is encountered also in roof shinglers, tile layers and others working under similar conditions.

Housemaid's knee.

"Charlie Horse."—This is a peculiar occupational disease affecting professional athletes, especially base-ball players, and to some extent the amateur as well.

It is characterized by a localized myositis causing induration of the muscles of the thigh and a peculiar limp.

"Going to boarding school" is one of the worst of occupations if the school work is overhard, the heating and ventilation insufficient, the food poorly prepared, lacking in quantity and variety and out-of-door exercise scant.

Potent for good or evil.

Nothing breeds more chlorotics than a poor boarding school or makes healthier women than a good one.

Barkeepers.

Habits in Relation to Occupation.—The excessive use of intoxicants is often definitely related to certain callings. The manager of a large hotel syndicate has said that he has yet to see one barkeeper who failed to develop the drinking habit, and this statement undoubtedly expresses a rule subject to few exceptions.

A traveling man who sells bar supplies, cigars, or even mineral waters is constantly subjected to a pressure which too often results in the formation of the liquor habit.

Combined factors.

Occupation Involving Continuous Mental Strain.—In this day of great enterprises a not inconsiderable proportion of the chronic ailments encountered by the physician may be traced in part to continuous mental overstrain, usually combined with lack of exercise, improper diet and too often with overindulgence in liquor or tobacco.

Value of vacation.

No one who has seen the remarkable improvement in chronic disorders of the heart, stomach, kidneys, or various diseases of the nervous system that follows a period of complete rest and freedom from worry can doubt the potency of mental overstrain as a factor in the etiology and prognosis of disease. Railroad men, financiers, and especially board of trade operators suffer greatly from this cause.

The Physician.—The life of a practising physician is of such a nature as to readily explain the high early mortality encountered in this class, for in the physician's calling are combined constant exposure to infections, deficient and interrupted sleep, lack of exercise and recreation, excessive and continuous mental strain, and an exhausting demand upon his kindly emotions.

A neglected topic.

MINERAL POISONING.—A complete volume would be required to do justice to this aspect of our topic, and there can be no doubt that too little attention is paid to the possibility of occupational poisoning by physicians other than those practising in large manufacturing centers, where the conditions are exceptionally favorable to the development of occupational disease.

Arsenic.—Aside from poisoning due to the inhalation of fumes or dust during the processes of milling, grinding and smelting, one encounters cases among workers in anilin dyes, toy or artificial flower makers, dyers of woolen or cotton goods, playing-card makers, taxidermists, lithographers and shot makers.

Anilin.—This derives its harmful effect from three sources, viz.: itself, the arsenic often combined with it, and the nitro-benzol used in its manufacture.

Bromin and Iodin.—The fumes of this substance cause bronchitis and predispose to tuberculosis.

Carbon Bisulphid.—This highly poisonous substance is used as a solvent for sulphur, iodin and oils, and is largely used in the manufacture of rubber goods.

Chlorin.—In the bleaching of linen, cotton, bones, ivory and rags the fumes are irritating to the bronchial mucous membranes and predispose to tuberculosis.

Chromium.—This enters into chrome yellow and chrome green, and is used in staining glass or porcelain, printing bank notes, and dyeing linen, wool and silk, and, as potassium bichromate, is used in photography.

Copper.—Workers in brass, nickel platers, bronzers, copper-sheet scrappers, pin makers, bell-metal workers, stone workers, engine wipers, and others handling, copper, bronze, brass or nickel may suffer from chronic poisoning. It should be remembered that brass is composed of copper and zinc, with or without tin and lead; bronze, of all four metals, and that nickel plating is sometimes done with an alloy composed of copper, nickel, iron and tin. It is further combined oftentimes with lead and arsenic. One of the most severe cases of lead poisoning ever encountered by the author was caused by polishing an alloy of this kind. Alloys.

Lead.—The possibility of lead poisoning in obscure gastrointestinal affections as in connection with its well-known nervous manifestations should never be forgotten. Not only is it found in lead smelters, refiners, sheet-lead rollers, lead-pipe makers, shot makers, typesetters, plumbers, toy makers, and painters, but also in lacquer polishers, gilders, bronzers, enamel workers, glaziers, pot, pan, card, cardboard or brick makers, makers of brass instruments, file cutters, flint glass workers, workers in white or red lead and litharge, calico printers and those engaged in the manufacture of lace, artificial flowers and wall papers.

Mercury.—Chronic mercurial poisoning may result from idiosyncrasy, long-continued overdosage, or employment in smelters, quicksilver mines and felting rooms.

Phosphorus.—This is now comparatively rare, and is almost wholly limited to those engaged in the manufacture of the parlor match.

Turpentine.—Some persons are peculiarly subject to turpentine poisoning, and painters working in poorly ventilated rooms may become chronically affected.

Occupations Involving Excessive Heat.—Extreme *dry* heat can be borne without serious results in persons habituated; nevertheless, firemen on ocean steamers or naval vessels, and bakers suffer from heat, moisture, foul air, and the effects of chill and exposure due to extreme temperature variations. Moist vs. dry heat.

Miscellaneous Diseases of Occupation.—Millers, potters, file cutters, grinders of edged tools, wool and cotton spinners, marble and stone cutters are peculiarly susceptible to tuberculosis, particularly in its fibroid form. Handlers of rags and skins occasionally acquire anthrax, internal or external. Female domestics are peculiarly liable to anemia, gastric ulcer and tuberculosis. Butchers or slaughterhouse men suffer from septic infections, stablemen from tetanus and glanders, brewers, saloonkeepers, bartenders and others of the same class show an enormous mortality from alcoholism, tuberculosis, diseases of the nervous system, pneumonia, diseases of the liver and kidneys, and suicide. Tuberculosis. Anthrax. Sepsis, tetanus, glanders.

OCCUPATIONAL STIGMATA.—Those who follow certain occupations often present somewhat characteristic deformities: the lowered shoulder of The lowered shoulder.

the desk worker, pack peddler and tailor are familiar to everyone. Few railroad trainmen escape the loss of one or more fingers.

Furthermore, even the study of the callosities associated with certain callings prove helpful occasionally in indentification, and a few of the occupations thus suggested are given briefly below. Callosities are shown at the points indicated:

Banjo, Guitar, and Harp Players.—Finger-tips both hands.

Zither Player.—Finger-tips of left hand, under surface and tips of index, middle and ring finger of right.

Violoncello or Violin Players.—Tips of fingers, left hand only.

Hand Compositors.—Palmar surface of thumb and index finger, right hand.

Fencing Masters.—Ulnar border, right palm.

Hand-organ Man (carrying own organ).—Outer side of right hip and thigh.

Seamstresses.—Roughened radial border of terminal phalanges of left thumb and index.

Tailors.—Ring-shaped callus on right thumb and index finger, left thumb and index roughened, enlarged bursæ over external malleoli.

Turners.—Outer border of right little finger.

Clerk, Using Pen or Pencil.—Outer surface of terminal joint, right little finger.

The rough callous hand of the day laborer contrasts sharply with the soft hand of the sedentary worker, as does that of the seamstress or domestic with those representing the lighter occupations of her sex.

Sailor and soldier.

The tan of the sailor contrasts sharply with that of the soldier, the one wearing his shirt open at the front, the other a high collar and close-buttoned coat.

The domestic parasite.

Idleness a Disease Breeder.—*Want of occupation, mental and physical, is often deleterious and the psychasthenia, hysteria, morbid irritability and ennui so commonly seen in spoiled, pampered, pleasure-loving, and responsibility-free women, are seen rarely in the hard-working housewife.*

Value of a "hobby."

The man who for years has given his attention to active business or professional work may find complete idleness both boresome and dangerous, and few men or women, can be well mentally or physically without some definite occupation, however trivial.

Every man who in later years forsakes his life's work should cultivate a hobby.

Postal address.

RESIDENCE.—The novice frequently fails to secure the full name and postal address of a public service patient under examination, forgetting that future communication is sometimes most important, in that some of the most valuable information results from following unusual cases.

With respect to the medical history proper, both present and past residence should be known, and in general, information upon this subject is of value in relation to the following points:

(a) The possible introduction of epidemic disease by persons coming from an infected area.

(*b*) *In tracing its distribution*, as in an epidemic of cholera or typhoid affecting certain portions of a city or larger district. Tracing epidemics.

(*c*) *In tracing its individual sources* as illustrated by the detection of careless dairymen, who spread infection along their route; and finally,

(*d*) *The special liability to certain diseases in definite districts or countries*, as illustrated by diseases of the tropics, such as malarial fever, dengue, yellow fever, and plague, the hydatid disease of the Icelander, or the leprosy of the Sandwich Islander. Climate an etiologic factor.

The Home.—Specific inquiry concerning the exact situation of the patient's house, its elevation, exposure, the character of the surrounding soil, and the location of the sleeping chamber, is sometimes important.

FAMILY HISTORY.—*The transmission of special structural vulnerability and predisposition is just as marked and as well-proven as is inherited likeness in form or feature.* Inherited predisposition.

In taking a family history, one should secure full information as to the terminal illness of family members, often going back two generations and including collateral branches if significant facts develop. It is often of the utmost importance that the state of health or cause of death of the husband, wife and the children of the patient be established. Extension of inquiry.

It is seldom sufficient to ask whether any members of the family, immediate or remote, have suffered from hereditary disease, or even to put the question in the somewhat more specific form of an inquiry concerning tuberculosis or cancer. Specific inquiry.

Apoplexy, heart disease or Bright's disease may be readily admitted, but insanity, epilepsy, tuberculosis and malignant disease are often concealed, either intentionally or quite as often, innocently. Deliberate concealment or actual lying are far from infrequent in ordinary case histories and are especially so in life insurance applications. Concealment common.

The questions must bring out if necessary, the leading *symptoms* of the illnesses of deceased family members. One may ask if there was chronic cough, spitting of blood, emaciation, night sweats, or fever, the vomiting of blood, or the presence of a tumor, associated with emaciation and failing strength, in the given case.

Certain Euphemisms of Case-taking.—Deaths from "*childbirth*," "*exhaustion*," "*grief*," "*broken heart*," "*general decline*," etc., must never be accepted without careful and tactful cross-examination. "*Senility*" and "*old age*" are much abused terms and in the lay mind cover death at any age above fifty.

No man ever passed his novitiate as a casetaker without saying hard things of "*malaria*," "*chills*," "*fever*," "*decline*," and especially "*marasmus*" and "*don't know*," and no student can give better evidence of his skill and tact than is furnished by an adequate, unambiguous family history.

Alternatives in Heredity.—*Few diseases are directly transmitted as such, hereditary influence usually taking the form of more or less marked predisposition.* Direct transmission rare.

Thus the disease of the descendant may be but the congener of that of the forebear and is then termed an alternative, or the inheritance may be that of marked vulnerability or its opposite, relative invulnerability.

Resistance and non-resistance.

Short- vs. Long-lived Families.—To the former class belongs the family whose members show a low average lifetime, the ready acquisition of, and feeble resistance to, acute or chronic ailments. To the latter class appertains a vigor of constitution and strength and harmony of structure which ensures resistance to disease and long life.

Congenital asthenia.

Congenital weakness of structure, imperfect or unstable function, and a peculiar nutritional instability, characterize large groups of people in all civilized countries, and the condition has been well named by Berthold Stiller "*Asthenia Universalis Congenita.*" This is now widely accepted by European clinicians as the condition underlying many bastard syndromes which, in the past, have been considered and treated as clinical entities.

Nearly all cases of so-called "nervous dyspepsia" ("gastric neuroses") fall under this head and an understanding of this fact supplies the means of their successful treatment.*

Alternatives.

The Nervous System in Heredity.—*A strong alternative relationship exists between alcoholism, insanity, epilepsy, hysteria, and criminal impulse.*

Specific inheritance.

In some families, however, there is little variation, insane grandchildren follow insane grandparents and epilepsy in the father means the same disease in the child. Some remarkable cases of hereditary alcoholism have come under the author's observation. In one, especially, it seems impossible for any male family member, or even collateral branches, to escape the curse.

Cardiovascular alternatives.

Apoplexy.—The hereditary tendency to cerebral hemorrhage is one of the most striking facts in medicine but is of course inseparable from its etiologic causes and *apparent* alternatives, arteriosclerosis, *chronic Bright's disease*, aneurysm and the degenerative diseases of the heart.†

Age and sex incidence.

Cancer.—Few physicians of experience will be found who deny an hereditary *predisposition* to cancer, yet it is well to minimize it and to avoid too radical statements in regard to it, as tending to create undue apprehension in the minds of members of tainted families. Like apoplexy, the tendency in this disease is toward death in middle age, the tendency to transmission seeming to be more marked from mother to daughter than from father to son.‡

Diabetes.—Few diseases are more distinctly hereditary than diabetes, if we regard it as one of the alternatives of inheritance in nervous and mental diseases. According to recent observers it is possible to separate from other types a distinctly hereditary form which is characterized by an equality in predisposition as between male and female, or even the predominance in the

* The author has been especially interested in these dyspepsias and in the narrow, atonic, dilatable, symptom-producing heart which is associated with this condition, and deals with both, in their appropriate sections.

† The author has reported a case in which, of nine family members, five died of apoplexy, two of chronic Bright's disease, and one of heart disease, every death occurring between the ages of forty and fifty-five. The then surviving brother has since died of the dominant disease. In each case, doubtless, chronic interstitial nephritis, and associated arteriosclerosis, myocardial degeneration, and arterial hypertension coexisted.

‡ Undoubtedly the hereditary element has been exaggerated in the past, but quite as certainly the present tendency on the part of some writers to deny it represents an over-reaction.

female. The best examples of inherited disease are found in the race showing the greatest vulnerability, namely, the Hebrew.* Race and heredity.

Gout.—This distinctly hereditary ailment is potent in the production of arteriosclerosis, apoplexy, Bright's disease, gastrointestinal ailments and certain forms of diabetes as well as all forms of gout. Potent and protean.

Hemophilia.—This extraordinary disease is atavistic in its transmission, the deadly tendency to excessive bleeding, spontaneous or induced, being transmitted through the females of a family to certain of their male issue. In certain families it has been traced back for hundreds of years in an unbroken sequence. Atavism

Syphilis.—In relation to family history direct inquiry is usually impossible and the physician must ordinarily depend upon the disclosures of parents or the recognition of inherited syphilis in the patient.

TUBERCULOSIS.—Direct Transmission vs. Environment.—That, in excessively rare instances, tuberculosis may be transmitted from mother to child *in utero* cannot be doubted, and it is certain that the incidence of tuberculosis is greater in those of tainted family record. Direct transmission rare.

Inheritance is due chiefly to a congenitally asthenic structure and a constitutional functional inadequacy, which render the tissues of the child of tuberculous parents more vulnerable and less resistant to the tubercle bacillus than is the case in one of sound, vigorous and untainted stock. Inherited physical deficiencies.

Even predisposed children, if favored in climate, nutrition, and environment, need seldom or never develop the disease under conditions eliminating the possibility of contact with the living germ, the exception being found in a latent prenatal glandular or bone infection. Importance of environment.

Unfortunately, however, these child victims of predisposition born in tuberculous households are in every way exposed, during the earlier years of life, through their creeping over dirty floors, their tendency to carry dirty hands and infected articles to the mouth, and especially through the embraces and caresses of the infected parent, brother, or sister. Direct exposure.

The peculiar shape of the chest associated with tuberculosis and found most frequently in those of tainted stock is merely one of those many structural peculiarities and deficiencies of *chronic congenital asthenia* which become factors in infection and favor the progress of the disease. Thoracic conformation.

Naso-pharyngeal Obstruction.—Of great importance are the obstructions in the naso-pharyngeal passages, especially adenoid growths and hypertrophied tonsils. These, in themselves favorable soil for the tubercle bacillus and other microörganisms, embarrass respiration, hamper the development of the lungs and permit collapse of the lower chest, while further contributing to infection through frequent recurring colds and chronic catarrh. Invites infection and lowers vitality.

Life Insurance.—One has only to consult insurance manuals to understand the importance of physique and family taint as factors in tuberculosis, yet, admitting certain exceptions, it is generally true that, the attainment of

* M. Loeb: The Hereditary Form of Diabetes. Zentralblatt für innere Medizin, Aug 12, 1905, No. 32, p. 786.

Physique vs. Heredity.

full manhood, an exceptional physique, and freedom from direct exposure, largely overbalance a bad family history.

The victims of congenital universal asthenia possess every characteristic of tuberculous predisposition and an astonishingly large proportion of them show positive tuberculin reactions or skiagraphic signs suggesting or proving past infection of the lungs even though at the time free from active symptoms.

It must be remembered that the greater number of men and women have at some time become infected and will yield signs under tuberculin tests or the X-ray, even *though they may never afterward show any evidence of an active lesion.*

Special Conditions Affecting Heredity in Tuberculosis.—With respect to the individual of tainted stock, the significance of tuberculosis in the parents is *apparently* but slightly greater than that in brothers or sisters, but probably this is because arrested tuberculosis in the mother or father is seldom to be traced in case-taking, such past events being usually either forgotten or concealed. Anyone who has practised in a region frequented by cured consumptives will appreciate the truth of this statement.

Degree of relationship.

Physical Resemblance.—Mere facial resemblance is of little importance, but general structural likeness is of decided import.

Direct Exposure to Infection.—*The health of living family members and especially of the children of parents under examination, or vice versa, as well as the condition of parents affected by, or dying of, tuberculosis at or near the time of the birth of any individual under examination are vitally important factors.*

Vitally important data.

The member of a family or household in which active tuberculosis exists, and the individual who associates with infected persons outside the family, is exposed to great risks from infection if proper precautions are not faithfully and intelligently observed.

"White plague" households.

PREVIOUS ILLNESSES.—Cardinal Importance.—*A knowledge of the general state of health and past illnesses of a patient may assist the diagnosis, either by excluding some immunity-conferring disease, or by fitting a symptom group to the known after-effects of some previous illness. The exanthemata and yellow fever illustrate the one, syphilis both.*

Ground ailments.

A patient reporting "stomach trouble" may present a clear history of previous gastric ulcer, appendicitis, gall-stone colic, or the morning vomiting of advanced interstitial hepatitis.

Another may have repeated attacks of acute rheumatism or gout and our attention is thus specially directed to the heart, blood vessels and kidneys which may reveal changes wholly adequate to explain his symptoms. A history of severe nocturnal headache may be explained by an ancient syphilis, an intractable periodic neuralgia, by past malaria, or oftener by a history of accessory nasal sinus infection.

Use plain language.

*In such interrogation the simplest and most homely phraseology should be used in dealing with the ignorant, and leading questions cannot wholly be avoided.**

* An ignorant patient of the author dying of perforating but previously unrecognized duodenal ulcer readily admitted having formerly "puked" blood and suffered much "misery in his guts," though on repeated questioning he had denied "pain," "nausea" and "vomiting."

HISTORY AND ANALYSIS OF THE "PRESENT AILMENT"

Tactful guidance.

Interrogating the Witness.—*The patient should be led to tell, almost of his volition, but in as few words as possible, the symptoms of his disease and their duration.*

Checking garrulity.

Any attempt to theorize or drag in immaterial facts should be tactfully checked. To guide and direct the disclosures, check garrulity, verbosity and triviality, to systematize the disclosures and yet use few leading questions, constitutes an art both valuable and rare.

General vs. Specific Symptoms.—*One should discriminate between symptoms that are general in their nature, common to a large number of diseases and subject to various interpretations and those that are local, peculiar or specific.*

Some of the principal general symptoms will be described in this connection and only their special features referred to in other sections or under the symptomatology of specific ailments.

FEVER.—Thermometry.—Fever as a clinical manifestation is merely an elevation of the body temperature, formerly determined roughly by the hand, but nowadays accurately registered by the clinical thermometer. In such diseases as pneumonia and typhoid the skin is dry and hot, yet on the other hand, a high rectal temperature may exist in cholera even when the surface temperature is as low as 70°.

Misleading surface temperatures.

Selective points.

The thermometer is ordinarily applied to one of four points, those being in the order of frequency, the *mouth*, the *rectum*, the *axilla*, and *vagina*.

Rectal readings will exceed mouth temperatures by about one degree Fahrenheit and constitute the only accurate registration in the aged and in cases of profound weakness.

Worthless thermometers.

There is no poorer investment than a cheap thermometer and the test scales supplied with some of those of the lower grades are worthless by reason of improper calibration and imperfect seasoning of the instrument and misrepresentation on the part of irresponsible manufacturers.

Precautions to be Observed.—*In the use of a thermometer, certain precautions must be observed:*

(*a*) *It should be cleaned before and after use.* (*b*) *The scale should invariably be inspected, as the thermometers are self-registering and require to be shaken down after use.* (*c*) *If used in the mouth, it should be placed well under the tongue and held by the tightly closed lips (not teeth) of the patient.*

If for any reason the lips cannot be closed, if the patient be unconscious or delirious or if an acute stomatitis or tonsillitis be present, the temperature should be taken per rectum.

(*d*) *Axillary temperatures should be avoided whenever possible, as being subject to maximum error variation.*

If so taken, the thermometer should be placed deeply in the previously dried axilla and the corresponding elbow should be kept close to the body and carried well forward.

Axillary readings are particularly misleading in incipient or but slightly febrile tuberculosis and their use is seldom necessary or justified.

Classification.

Temperature Range in the Human Body.—*Subfebrile* temperatures are those running from 99° to 101.5°F.; *moderate fever*, 101.5° to 103°F.; *high fever*, 103° to 105°F.; *hyperpyrexia* 105° plus.

NOTE.—To convert Fahrenheit readings into centigrade subtract 32, multiply the remainder by 5 and divide the product by 9. To convert centigrade readings into Fahrenheit, multiply by 9, divide by 5 and add 32.

Diurnal range.

There is a normal daily variation reflected in nearly all fevers, the range being about 1° to 1.5°F. The lowest reading occurs in the early hours following midnight, the highest between four and six oclock in the afternoon.

„A dubious normal."

Faulty Figures.—Granting that 99°F. to 99.5° or even 99.8° may represent a normal variation in infants, the author believes that the body temperature range in adults, as ordinarily given by the physiologist, is too high and is based upon collective observations which included persons having unrecognized chronic infections or an incipient disease, such as pulmonary tuberculosis.

Obscure infections.

*Years of observation in private and public practice have convinced him that in adults under ordinary conditions, a persistent maximum mouth temperature exceeding or even reaching 99°F. is strongly suggestive of existent incipient tuberculosis, chronic infections of the tonsils, accessory nasal sinuses, prostate, gall-bladder, the appendix, the ovaries and tubes in women, peridental abscesses, or the insidious onset of some acute ailment.**

Tropical Diseases and Endocarditis.—In the case of persons returning from, or resident in, the tropics, the possibility of amebic dysentery and malaria should receive special investigation if any fever be noted. *Endocarditis of the chronic recurrent type gives rise to many errors.*

Heat and Exercise.—It should be remembered that violent exercise and excessive heat slightly and temporarily raise body temperature and that in

Age and youth.

infants and very old persons it may range half a degree or more higher than in those of intermediate age.

The temperature of children is extremely labile and may reach a high degree without serious significance, often subsiding as suddenly as it came.

A nonentity.

Febricula (Ephemeral Fever).—Fevers of short duration and unknown causation affect adults, but with the advances made in diagnosis, have become relatively rare, and "*febricula*" means little more than an obscure, trivial and transitory fever, and is unworthy of further description.

Inverse temperature.

For variable periods in certain cases of tuberculosis, acute or chronic, the morning temperature represents the daily maximum.

PATHOLOGIC VARIATIONS IN TEMPERATURE.—Malingerers.—*Hyperpyrexia* of extraordinary degree has been reported, but has probably

Clever artifices.

been due to deception on the part of the patient, it being a common trick of the malingerer or hysterical patient to heat the thermometer bulb by friction, by taking hot drinks just before the tube is applied or by cleverly utilizing a hot water bag.

The highest genuine temperature observed by the author was 110°F.

* A recent investigation of nurses' temperature undertaken by the author in a local hospital has further strengthened this opinion.

under the tongue, and occurred in a woman of hysterical temperament suffering from the extreme pressure pain of a spinal caries.*

Fever in Hysteria.—Elevation of temperature may occur in hysteria alone, a fact never to be forgotten. The highest personally observed (104°F.) was obtained during an hysterical paroxysm following great emotional shock, and the attack and the fever subsided almost instantly under sharp ovarian pressure. In this particular instance the physical exertion represented by the violent convulsive movements might account for the high temperature in part, at least. Puzzling cases.

Some authorities claim that temperature may persist for weeks in hysteria and even simulate typhoid, but until we know more of the real nature of hysteria and find fewer examples of its confusion with organic ailments, it might be safer to assume that genuine, long-continued fever excludes it. Rule of action.

Interaction of Infection and Resistance.—*The significance of high readings is variable and depends upon the nature and virulence of the toxin, the age and nervous condition of the patient, and also upon the degree of his vital resistance.*

Certain hyperpyrexias seem to indicate little more than an intense infection associated with vigorous constitutional resisting power, such cases being seen in connection with lobar pneumonia, in which a temperature of 105°F. is neither uncommon nor necessarily of fatal import. *On the other hand, one finds instances of virulent and fatal infection with but slight febrile reaction.*

Extremely high readings, if long sustained, indicate a most serious prognosis, and a sudden rise ("agonal fever") sometimes precedes death.

Subnormal temperatures may be observed in those suffering from collapse, extreme exhaustion, chronic exhausting or wasting diseases, insanity (especially melancholia), chronic incompensated heart disease, adrenal insufficiency and myxedema. Subnormal temperatures.

Oftentimes the surface temperature is subnormal and the rectal reading high, notably in cholera and certain cases of tuberculosis.

Febrile Types.—Fortunately for the clinician, the fever in many of the acute diseases pursues a more or less characteristic course or bears a peculiar relation to the appearance of other symptoms of the disease.

Fevers may be placed under four general heads: (1) *Intermittent.* (2) *Remittent.* (3) *Continuous.* (4) *Indeterminate or irregular.*

An intermittent fever, however far it may rise, drops to or below normal during some part of the twenty-four hours. A *remittent fever* shows marked remissions during the twenty-four hours; while in *continuous fever* the remissions are slight. These are but the types and one must understand that a continuous fever may at some stage show remissions, or a remittent fever encroach upon the intermittent; indeed, the temperature charts covering long periods may in such diseases as tuberculosis show every type of fever and yet be predominantly intermittent. The intermittent fever of this disease is often given the name *hectic.*

* The record case reported by Teale was one of *spinal injury* and the temperature recorded was 122°F.

Rough standards.

Roughly speaking, one may say that when the maximum daily variation in a moderately severe fever is less than 2° it is *continuous*. When more than 2° and yet with an average minimum distinctly above the normal, it is *remittent*, and any fever showing a tendency to periodically fall below normal is *intermittent*.

Fastigium and defervescence.

The maximum stage of fever is known as its **fastigium,** the term being applied especially to the fevers of the continued or remittent type and to the stage of the disease as well as to the fever itself. Its opposite is called the period of defervescence or decline.

Diagnostic Import of Fever.—Putting aside the rare cases of hysterical temperature and excluding deception and faulty technic, one may say *that fever is primarily of diagnostic value in proving the existence of some organic ailment or definite toxemia, as opposed to a functional disorder, a matter of no little importance in dealing with neuropaths and malingerers.*

Differential value.

Afebrile Cases.—The absence of fever, however, does not rule out any one of these conditions, for there is no disease ordinarily febrile that may not, though in rare instances, exist without fever or even with subnormal temperature. This fact is best exemplified in the case of typhoid, occasional pneumonias, especially those of old age, and certain cases of general peritonitis.

A deceptive type.

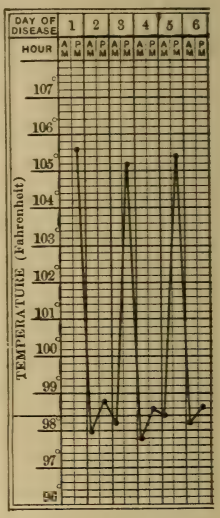


FIG. 18.—Clinical chart of tertian malaria. Typical and extreme example of the *"intermittent"* type of fever. (From *Wilcox's "Fever Nursing."*)

Chief Causes of Fevers.—By far the greater number of fevers are due to the action of pathogenic germs and their toxins, whether these be developed within, or, more rarely, without the body, as in the case of ptomains. Some nevertheless are due apparently to other toxic substances, even such as are generated by the vital processes of the living body itself. Such are the curious auto-intoxications associated with deficient or faulty metabolism as illustrated by acute gout.*

Ptomains and metabolic poisons.

The terminal stages of diseases of nutrition, malignant disease, and certain of the severer types of anemias are associated with fever and doubtless in many such instances obscure infections play an important part.

Cerebral.

Fever of central origin may be encountered in cerebral embolism, thrombosis, apoplexy, brain tumor, and direct injury, or even the changes incident to heat-exhaustion and sunstroke.

Starvation fever.

Inanition is sometimes associated with fever, usually of mild grade, which ordinarily occurs after severe exhausting ailments associated with much wasting of tissue and loss of strength.†

* This statement may require modification in the near future if the increase in our knowledge of basic sub-infections continues. The author confesses his inability to believe that the clinical phenomena of acute gout are adequately explained by any existing knowledge of its metabolic disturbances.

† The so-called "hunger" temperature may be most misleading in convalescent typhoids who have been too long starved. It is seldom observed under modern methods of feeding.

Occurring under such conditions, it is more likely to be due to a smouldering infection but it cannot be denied that cases of exhaustion temperature do occur and subside promptly when proper nutrition is attained.

THE PHENOMENA OF FEVER.—*One must first determine whether the fever came on suddenly or gradually, whether it was associated with chill or, in the child, convulsions, for how long it has endured, whether it is to be classified as distinctly intermittent, remittent or continuous, and, furthermore, whether it is associated with any history of infection or with the appearance of an exanthem. If the latter be the case its exact time of appearance and its relation to any rise or fall in the temperature must be noted.*

Consider onset, type and cause.

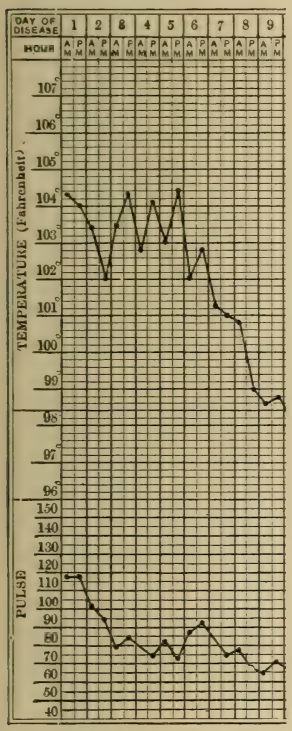


FIG. 19.—Clinical chart of a yellow fever patient, showing a pulse typically slow in comparison to the height of temperature. (*From Wilcox's "Fever Nursing."*)

Condition of the Skin.—Heat and Moisture.—In most fevers the skin is dry and palpably hot, but occasional cases depart from this rule and certain diseases such as acute rheumatism and cholera almost consistently violate it.

Digestive Organs.—Nausea, vomiting or diarrhea (initial, persistent or terminal), constipation, loss of appetite and a coated tongue are present in varying degrees in all severe acute febrile ailments.

The Nervous System.—Headache, and pain in the limbs are present in nearly all cases of fever, though often only at its onset.

Mental Condition.—*The mind may be clear or clouded and delirium may be present at the onset only or for long periods, continuously, or only at night.*

Delirium.—This may take the form of transitory illusions or hallucinations, may be violent and maniacal, mild, or of the low muttering, mumbling type of the typhoid state, the patient perhaps lying with open but unseeing eyes (coma vigil). Persistent delirium of the violent or low muttering type is of unfavorable prognostic significance and the exhausting effect of the former constitutes a serious peril and demands prompt therapeutic measures.

Its many varieties.

Children show an especial tendency to delirium even in fever of moderate intensity, whereas, it is rare in adults save in the most severe types of infection, in the intoxications or in central disturbances.

Readily excited in children.

Acute Febrile Delirium (*Bell's Mania*).—*This consists essentially of a violent, continuous, exhausting and maniacal delirium, associated with high fever and a typhoid state.*

It is a rare condition, peculiar to women and of unknown causation, is initiated by shock or other emotional strain, and may be associated with the

Probably cryptogenetic sepsis.

convalescent state. No specific pathologic findings are present after death and the disease may readily be confounded with the acute maniacal delirium occasionally present at the onset of pneumonia, typhoid and uremia.

It may be rapidly fatal, running its course in from one to three weeks. The differential diagnosis is one of exclusion and the author's experience would indicate that the condition is usually one of acute cryptogenetic sepsis.*

Pulse-respiration-temperature ratio.

The Pulse and Respiration in Fevers.—Both pulse and respiration bear a definite relation to the temperature curve. *The increase in pulse rate is ordinarily from* 8–10 *beats per minute for each degree of fever,* but a departure from this rule is commonly noted in typhoid fever, yellow fever and lobar pneumonia, and favorably affects prognosis in these diseases.

Watch the heart.

Heart-muscle Impairment.—*The heart muscle suffers to a variable degree in all severe infections and intoxications and must be carefully watched for signs of weakness, degeneration or valvular inflammation, this being especially true, as regards the valves, heart muscle and pericardium alike, in attacks of acute or subacute rheumatism, scarlet fever, severe true influenza, and diphtheria.*

In any infection, the examinations of the heart should relate, not alone to murmurs, but to variations from the normal in the heart sounds, extension of the cardiac borders and important variations in the cardiac rhythm.

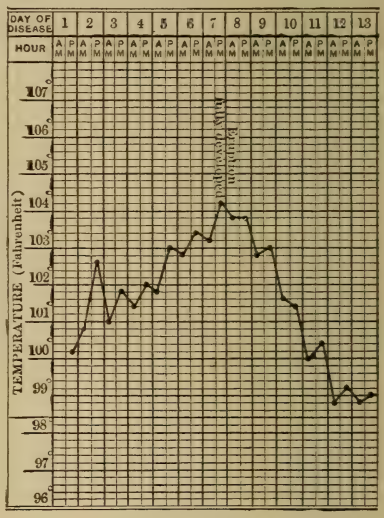


FIG. 20.—Clinical chart of measles, showing "*fastigium*" developed with eruption, primary rise, secondary fall, and defervescence following appearance of exanthem. (*From Wilcox's "Fever Nursing."*)

Myocardial "weakness."

The author believes that much of the weakness associated with acute infections is due to myocardial toxemia and that in no other way can one explain the rapidly induced and unduly persistent exhaustion evident upon exertion in various acute infections even though actual power on the part of the skeletal muscle groups is relatively well retained.

Pulse, strength and weakness.

"Sthenic" vs. "Asthenic" Fevers.—The pulse of high fever of the so-called *sthenic* type is not only rapid but full and bounding. In *asthenic* fevers (those attended by profound exhaustion) it becomes soft, weak, and in the typhoid state, dicrotic.

* In three instances the author has encountered precisely this disease picture *with recovery*, as a late and apparently isolated sequel of child-bearing. Blood cultures were negative but each attack showed a sharp leucocytosis at the onset.

It is at present described by some authorities as an "*exhaustion psychosis*" but the element of infection cannot well be disregarded. It may present many variants and need scarcely be considered as a clinical entity.

Temporary extrasystolic irregularity or even partial and temporary heart-block may occur and in acute and chronic affections of the kidney the pulse may be of high tension. Such a pulse should always direct attention to the kidneys and calls for examination of the urine if this duty has been neglected. In inflammation of the peritoneum it is small, hard and wiry.

Hypertension.

Wiry "peritoneal" pulse.

Helpful Types.—The accompanying charts show the peculiarities of fever in certain diseases, *but the student must not demand in his practical work that infections shall conform invariably to the text-book description.*

It is most important that the rule be known, but the common exceptions also must be recognized.

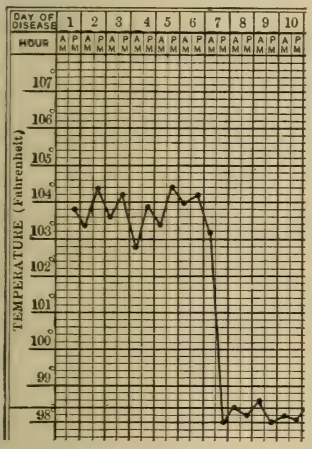


FIG. 21.—Clinical chart of acute lobar pneumonia. This typical case has terminated by *crisis* on the seventh day of the disease. (*From Wilcox's "Fever Nursing."*)

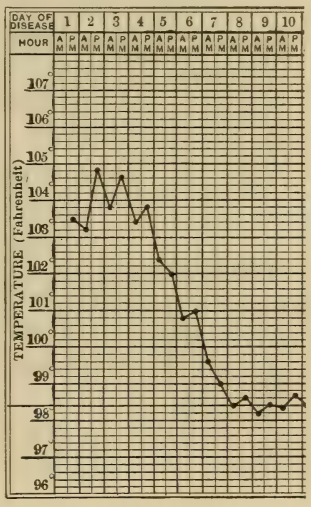


FIG. 22.—Clinical chart of scarlet fever. (*From Wilcox's "Fever Nursing."*)

Measles.—*In measles the temperature recedes twenty-four hours or thereabouts after the onset, rising with the coming of the rash on the fourth or fifth day.*

Rash on 4th or 5th day.

German Measles (*Rötheln*).—In rötheln the eruption appears on the first day or more rarely the second, and is usually the first thing noted. The fever rising much the same, but usually less than in measles.

Rash on 1st or 2nd day.

Scarlet Fever.—In scarlet fever the rapidly spreading rash appears on the first or at the beginning of the second day, following an abrupt onset with high fever.

Rash on 1st or 2nd day.

Varicella (*Chickenpox*).—In varicella the rash appears within twenty-four to thirty-six hours, associated with trifling fever.

Rash after 24–36 hours.

Variola (*Smallpox*).—In variola the violent onset, usually with chill and high fever, is followed by a marked remission, usually at the end of the third day, during which the rash appears in its papillary stage. During its subsequent changes the temperature rises, reaches its maximum with the purulent transformation of the vesicles and then gradually subsides. The diagnostic

True eruption on 4th day.

value of these differences in the relation of body temperature to the eruption is readily appreciated.

The Mode of Onset and Termination.—*In adults, the more severe acute*

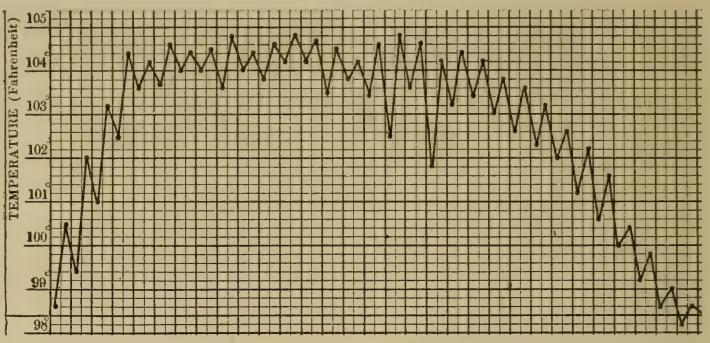


FIG. 23.—Typhoid fever chart, showing temperature typical, and unmodified by treatment. A rare combination at the present time. Serves to illustrate the so-called "step-ladder" temperature of the first week, "*continuous*" and "*remittent*" fever curves, and termination by "*lysis*." (*From Wilcox's "Fever Nursing."*)

Adults vs. Children.

infections are usually preceded by a decided "chilliness" or actual chill, with or without gastric disturbance, and, in children, by either chill or convulsions and frequently, by nausea and vomiting.*

Overwhelming infections.

In the malignant forms of typhus fever, smallpox, bubonic plague, yellow fever, malaria, and even of scarlatina, certain cases may die before the disease has reached its frank development.†

Insidious onset.

In several febrile diseases, the onset is insidious and without marked initial phenomena, and in senile cases this may be true of ailments ordinarily frank.

Abrupt Cessation vs. Gradual Recession.—The termination of fever is in most cases a gradual recession (*lysis*), but certain ailments of which classical lobar pneumonia is the type terminate suddenly (*crisis*).

Lysis vs. Crisis.

The critical phenomena vary somewhat, but usually the temperature falls to or below normal within

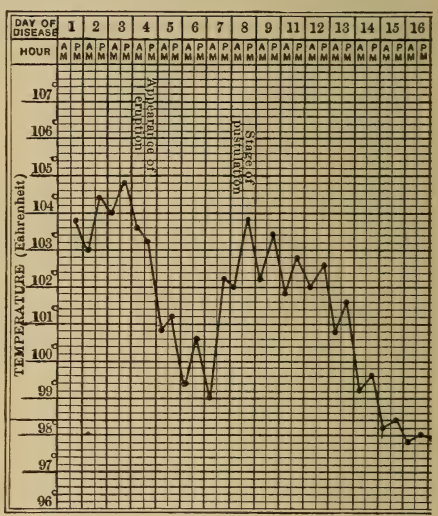


FIG. 24.—Clinical chart of smallpox, showing high initial temperature, rapid temporary defervescence following appearance of eruption, and the abrupt secondary rise just preceding pustulation. (*From Wilcox's "Fever Nursing."*)

* A severe rigor is certainly less generally observed even in pneumonia than the student is usually led to believe.

† In an epidemic of malignant scarlet fever observed by the writer a considerable proportion of the cases proved fatal within a period of less than twenty-four hours.

twenty-four hours, or often yet more abruptly. Profuse sweating, copious urination or perhaps profuse diarrhea may occur, suffering being replaced by relative comfort, and the contrast often most dramatic.

Pseudo-crisis.—A similar fall in temperature in diseases not associated with true crisis points usually to a serious complication, in typhoid, for example, to a hemorrhage or perforation. In pneumonia, a false crisis of marked degree is often followed by renewal of fever, the true defervescence occurring ordinarily on the day following. If true crisis does not follow one must suspect an extension and renewal of the process or the onset of some complication such as myocarditis, endocarditis, pericarditis, or empyema. *Profound myocardial toxemia may be associated with most misleading temperature recessions.*

Five crises.

In one case of migratory pneumonia observed by the author, lobe after lobe of the lungs went through a typical pneumonic cycle, the critical fall being promptly followed by chill, renewed fever and the usual signs of consolidation in the adjacant lobe.

COMA AND ITS CONGENERS.—Coma covers any state of prolonged unconsciousness from which a patient cannot be aroused by external stimuli.*

Coma vigil.

The dividing line between different states of unconsciousness and profound sleep is ill-defined and one merges into the other. The term *coma vigil* is applied to the peculiar complete or semi-unconsciousness of certain cases of typhoid fever associated with an open-eyed, low muttering delirium.

Stupor and lethargy both represent morbid sleep, the former profound as in alcoholism, the latter a mere drowsiness as in the premonitory stage of freezing.

Sources of error.

COMA.—Assuming that a patient is seen for the first time on the street, in the ambulance, hospital, or the home, and is comatose or stuporous, the difficulties in diagnosis are so great as often to baffle the diagnostician, but many avoidable errors occur through lack of proper method, ordinary knowledge and carefulness.

Information as to the conditions under which the attack of coma occurred should be first sought through some friend, relation or bystander.

Falls.

The appearance of the patient at the time of the attack, his movements and the direction and force of any fall he may have sustained, are also to be considered and oftentimes his *habits* or even his *previous health* may be ascertained.

Heatstroke.

The state of the atmosphere may at once suggest heat-exhaustion or sunstroke.

Stomach contents.

Inquiry must be made as to what the patient has taken into the stomach, having in mind the possibility of ptomain or drug poisoning, or sudden deaths or severe prostration following the overloading of the stomach in victims of the diseases of the heart and arteries. In public services, and especially ambulance cases, the history is often entirely lacking or untrustworthy, and the physician is thrown upon his own resources.

Ambulance] cases.

*In most instances profound coma is associated with a relaxed jaw, stertorous breathing and dry tongue, *e.g.*, morphin poisoning or apoplexy.

Poisons.

One must consider epilepsy, malingering, uremia, apoplexy, cerebral embolus, brain tumor, meningitis, diabetic coma, poisoning by opium, chloral and other narcotics: if delirium is present, belladonna; if convulsions, strychnia or uremia, and investigate the following points:

(*a*) **The Eyes.**—*Are the pupils equal or unequal, contracted or dilated? Do they react to light, is the conjunctival reflex present, and is there resistance to the separation of the lids?*

Unequally dilated pupils (the larger usually on the side of the lesion) suggest cerebral hemorrhage or general paralysis of the insane.

Contracted pupils suggest opium poisoning, or pontine hemorrhage. *In uremia they are usually dilated** and in all these conditions they fail to respond to light and the conjunctival reflex is absent. The same is true of cocain and the latter stages of chloral poisoning.

If convulsions are present with fixed, dilated pupils and the conjunctival reflex is absent, epilepsy or poisoning by strychnia, belladonna, or some other convulsant is at once suggested.

In alcoholism the pupils may be dilated, but usually respond to light.

Hysteria and malingering are, as a rule, readily detected by the prompt response to light, and the resistance almost invariably offered to the separation of the lids by the physician.

Flushing and cyanosis.

(*b*) **Color.**—*In alcoholism* the face is usually flushed. In *cerebral lesions* it may be flushed but is also usually cyanotic. *In uremia* there are ordinarily pallor and cyanosis and the latter condition may be excessively marked in the coma of *acetanilid poisoning.*

Usually complete.

Useful tests.

(*c*) **The Degree of Unconsciousness.**—*In the more serious forms of coma, insensibility is, as a rule, complete. The conjunctival sensitiveness and resistance of the eyelids to separation should be tested in all cases and deep, sharp and unexpected pressure over the ovarian region may resolve doubt in hysteric females, being often accompanied by a sudden complete or partial return to consciousness.*

The malingerer and even the "alcoholic" can seldom resist moving if the skin of the inner surface of the upper arm is suddenly and sharply pinched.

Conjugate deviation.

(*d*) **The Character of Attendant Convulsive Movements.**—*In cerebral hemorrhage* conjugate deviation of the eyes and head is most common. In the comatose terminal stage of *strychnia poisoning* both head and trunk muscles are convulsed, opisthotonos or emphrosthotonos are likely to be present, *but between the attacks the patient may be conscious and rational.*

In tetanus the convulsive seizures are likely to be limited to the neck and jaw, and complete relaxation between seizures does not usually occur.

Consciousness long maintained.

Neither in strychnia poisoning nor tetanus does complete, persistent unconsciousness supervene prior to the terminal stage, and often not at all.

In epilepsy the sequence of the seizures, if present or, as described by eye witnesses, *i.e.*, clonic followed by tonic convulsions, involuntary micturition, and perhaps tongue-biting are suggestive.

*A widely dilated pupil and moderate photophobia may precede the outbreak.

The ordinary malingerer is foolish to attempt convulsions on account of the many difficulties attending accurate simulation which can be overcome only in exceptional instances by certain of the elect of the brotherhood. Simulation difficult.

The Ophthalmoscope.—*Albuminuric or diabetic retinal changes may be present or the choked disk of brain tumor may be revealed, and the ophthalmoscope often proves invaluable.* Bright's and brain tumor.

Hysteric Coma.—*Although the convulsive movements are often strikingly epileptiform or tetanic, they are usually irregular and are associated with the typical hysteric facies.*

Furthermore, though the lips may be bitten, the tongue is not, whereas in true epilepsy biting of the tongue is a frequent accident and incontinence of urine almost a constant accompaniment of major seizures. *In hysterical tetanus the eyes are usually closed during a seizure and emotional utterances, sobbing, and crying are prominent.* The peculiar premonitory exhilaration and hyperacuteness of the special senses observable in strychnia poisoning are absent in hysteria and the onset of spasm is atypical. Many signs lacking.

In females sudden sharp pressure over the ovarian region may resolve doubt.

Frothy Lips.—Froth about the lips, if blood-stained, points to epilepsy, but is common to many of the convulsive states attended by unconsciousness. *Malingerers usually use soap in the mouth for this purpose.*

(*e*) **Paralysis.**—The limbs should always be handled to ascertain if paralysis be present in any member, the peculiar lack of all muscle tonus being usually readily determined and paralysis of the eye and face easily noted. Muscle tonus

Atrophy.—Paralysis of the extremities associated with marked atrophy suggests an old lesion which may or may not be related to the patient's present state. Antecedent lesions.

(*f*) **The Pulse.**—*In cerebral compression the pulse is slow, in uremia and apoplexy its high tension may be suggestive, in malingerers and hysterical persons it is the pulse of exertion, rapid, full and bounding and in other conditions it is, as a rule, of little value.* Arterial hypertension.

(*g*) **The Heart and Blood Vessels.**—A searching examination of both the heart and the accessible arteries is usually demanded and arterial hypertension, and sclerosis, together with an increased cardiac area, may prove of great importance. If valvular lesions are evident and especially if mitral stenosis be detected, the question of cerebral embolus at once arises and one might encounter the dying victim of a ruptured, slowly draining aneurysm which is emptying the patient's life blood into some closed cavity such as the pericardial sac, though death from aneurysm is oftener sudden. Acute dilatation. Embolus. Aneurysm.

(*h*) **The Temperature.**—In all cases the rectal temperature should be taken, even in those comas apparently alcoholic in their nature.

One of the commonest mistakes on the part of ambulance and police surgeons results from the failure to remember that pneumonia, like cerebral lesions both central and traumatic, is extremely common in the drunkard. Fatal errors.

As before stated, the presence of fever does not absolutely exclude hysteria and it is present in cerebral hemorrhage, the surface temperature being usually a degree or more higher on the paralyzed than on the non-paralyzed side.

Unusual but important.

(*i*) **The Lungs.**—Extensive pulmonary infarction, the acute onset of a massive pleural effusion, the sudden production of pneumothorax and the internal rupture of a large pulmonary abscess constitute some of the unusual causes of sudden unconsciousness observed by the author, and one must always bear in mind the possibility of internal hemorrhages and, as before stated, of *fulminant* acute disease.

(*j*) **The Breath.**—*An alcoholic odor*, though suggestive, does not prove drunkenness. *The so-called uremic breath* is reasonably characteristic, but closely approximated by that encountered in other conditions, and is a disagreeably aromatic odor that must be learned by actual experience. *The sweet breath of diabetic acidosis* is far more characteristic and often extremely penetrating.* *Certain poisons*, like laudanum, carbolic acid, hydrocyanic acid, ether and chloroform, yield a characteristic odor.

(*k*) **The examination of the blood** often proves of decided diagnostic value and should be applied in doubtful cases whenever possible.

Open hemorrhage may have occurred and at once suggests injury, self-inflicted or otherwise, hemoptysis, hematemesis, aneurysmal rupture or possibly the hemorrhagic accidents of hemophilia, leukemia or purpura.

(*l*) **Examination of the Urine.**—At the earliest moment a specimen of urine should be removed by catheterization and carefully examined with reference to contained solids, serum albumin, pus, blood, casts, or sugar, drug reactions and the like.

Few pathognomonic signs.

Diagnosis often Impossible.—*In conclusion it must be emphatically said that but few of the differential points given are absolutely distinctive, and there is no condition that more severely taxes the skill and common sense of the practitioner.*

Simple cases.

If the breath of a patient smells of laudanum, the pupils are contracted, the breathing stertorous, the skin cyanotic and clammy, and particularly if a bottle that has contained laudanum is found empty, the proof is sufficiently conclusive. Stertorous breathing, a flushed countenance, slow, hard pulse and the evidences of paralysis are sufficiently characteristic of a cerebral lesion.

Blind cases.

On the other hand, one often meets with conditions in which he is misled by a reversal of ordinary symptoms, confused or contradictory findings and a failure on the part of the underlying ailment to strictly observe the man-made laws of differential diagnosis.

Fruity breath.

Diabetic Coma.—(*Coma of acidosis*). Usually this form is readily recognized by the fruity, vinous, odor of the breath and the character of the breathing which is usually that of *strong*, deep, deliberate or unhurried inspiration and a somewhat short, sighing, expiratory phase (so-called "*Kussmaul* dyspneic type").†

*The author not long since detected it in passing upon the open street the little babe of a friend, which was supposed to be in perfect health, but was suffering from terminal diabetes, as determined by the family physician upon notification.

† Inasmuch as this type of breathing has been noted by all clinicians for centuries, the propriety of such a descriptive term is questionable.

The onset of such attacks always is preceded by an excess of diacetic acid and acetone in the blood and urine and, often, by irritability, fretfulness, headache and dyspeptic symptoms, concerning the possible significance of which family members should be warned. Danger signals.

Constipation, often extreme, is often present and may be a factor in exciting an attack, and starvation, colon flushing and brisk catharsis will often abort one presenting prodromal symptoms.

According to the author's personal observation, these attacks are quite frequently precipitated by cardiac overstrain, the heart being atonic and even readily dilatable in many advanced cases. Showers of short, broad casts may precede the attack. The pupils are usually widely dilated during a seizure. Cardiac overstrain.

The term "*air hunger*" as applied to the typical breathing described is peculiarly apt and interprets exactly the clinical picture. Air hunger.

The author feels that two distinct types of breathing encountered in practice merit the application of this term, viz: (1) The deep, urgent, strong, dominantly inspiratory type; (2) the superficial, hurried, gasping type with inspiratory dominance seen in many conditions associated with states of unconsciousness and extreme weakness. Two types.

To him, at least, the latter clearly expresses "air hunger," usually justified by the oxygen shortage present, from whatever cause, terminal or otherwise.

PAIN

A Purely Subjective Symptom.—*Pain as a symptom is purely subjective and may be trivial or agonizing, true or false, localized, referred or diffuse, momentary, temporary or persistent.*

Descriptive Terms.—Its differences are indicated by such descriptive terms as "dull," "aching," "sharp," "acute," "stabbing," "boring," "gnawing," "lancinating," "shooting," "colicky," "radiating," "diffuse," "tearing," "rending," "suffocating," "griping," "bursting," "smarting," "scalding," "burning," "grasping," "clutching," and the like, all of which doubtless reflect accurately the sensations of the victim. It may be deeply seated or superficial and may or may not be associated with tenderness due either to actual inflammation or to hyperesthetic areas.

Deceptive "Zones."—*In this connection the student is warned against a too implicit faith in the so-called "painful zones" which, though valuable and in general fairly suggestive, cannot be wholly trusted and too often lead one far afield.*

A painful area, dedicated by the usual diagram wholly to appendicitis, for example, serves as well for a local growth, post-operative adhesions, arrested ureteral calculus, incipient hernia, and even for the referred pain of certain pneumonias or an obstruction of the ileocecal valve. Furthermore, the *early* pain of appendicitis is likely to be diffuse and far removed from "McBurney's point." Sources of confusion.

Referred Pain.—*The site of pain is often far distant from its true source, and indeed certain referred pains may be dangerously misleading.*

Transposed pain.

The author has observed a case* in which an aneurysm of the *left iliac artery* caused pain over the appendix; others in which appendiceal pain was referred to the left side; yet others in which the pain of gall-stone colic first appeared in the region of the heart, and *vice versa.*

As has already been suggested, nothing is commoner than for the pain of pleurisy and pneumonia to be referred to the surgical regions of the abdomen along the terminal endings of an intercostal nerve, or for renal calculi, lodged in the lower portion of the right ureter, to closely simulate appendicitis. The referred abdominal pain of the insufficient heart is extraordinarily common and misleading.

The Character and Seat of Pain in Relation to Diagnosis.—*The pain of an acute inflammatory disease is usually associated with fever, and often with localized tenderness, the degree of either depending upon the nature of the affected structure, the individual susceptibility and resistance and the extent and situation of the inflammatory process.*

Acute inflammation.

In surgical diseases of the extremities, for example, the pain is ordinarily associated with distinctly localized tenderness and is of great value as a symptom. On the other hand, a central or a senile pneumonia may exist with little or no pain, or in other cases, by involvement of the pleura in a dry pleurisy, produce excessive distress.

Pain in Solid vs. Hollow Viscera.—*In any disease of a solid viscus inflammation of its parenchyma is a relatively or wholly painless process until extension to the internal or external limiting layers occurs; and even the hollow viscera are relatively insensitive to rough handling and trauma,* **though intense pain may attend their abrupt overdistention or violent contraction.**

Parenchyma insensitive.

Activity and pain.

Pain usually is distinctly related either to bodily movement in general or to functional activity of the part affected.

Effect of exudate.

Friction of the dry and inflamed serous surfaces causes respiratory pain in pleurisy, while in acute rheumatism the slightest movement of the affected joint is distressing. In all such instances nature seeks to relieve the condition by pouring out a fibrinous or serous exudate which fact explains the partial or complete relief of pain in fully developed cases of exudative pleurisy and pericarditis.

Neuralgia and neuritis.

Radiating pain is most characteristic of neuralgias and neuritis, and is recognized by its tendency to follow the known distribution of nerve trunks and by its association with marked tenderness over certain nerve paths or points of emergence.

Dull pain may take the form of general aching as in acute infectious diseases, or may attend congestion, capsular stretching or chronic low-grade inflammations, particularly such as affect the mucous or serous membranes.

Recent vs. Old Ulcers.—Irritation, acute inflammation, or recent ulceration of certain mucous membranes may be attended by severe pain, such, for example, as that of renal colic and of certain cases of gastric or duodenal

* Through the courtesy of my colleague, Dr. Archibald MacLaren.

ulcer. On the other hand, extensive but ancient ulcerations may be strikingly painless.

Gnawing or boring pain is frequently encountered in caries of the spine, aneurysm of the aorta, carcinoma of the stomach and inflammation of bone. It is also met with in some cases of old gastric ulcer, in gout, gall-stones and renal calculus.

Misleading Absence of Pain.—*In many of the conditions ordinarily associated with more or less severe pain this symptom may be wholly lacking in certain cases, even in the absence of stupor or coma. This statement applies, for example, to pericarditis, pleurisy, cholecystitis, cholelithiasis, gastric and duodenal ulcer, high grade coronary arteriosclerosis, pneumonia, certain cases of appendicitis of the gravest type, and even cancer of the stomach or bowels.*

Actual perforation of the appendix, stomach or duodenum may occur with entire absence of pain.

When Delay may be Fatal.—*Abrupt, atrocious abdominal pain with localized or general tenderness or decided defensive rigidity, symptoms of shock or collapse and an initial sharp drop in blood pressure, will represent almost invariably a critical surgical emergency.*

PAROXYSMAL PAIN.—Gall-stones, renal colic, colon colic, splanchnic arterio-spasm, spasm of the pylorus, appendicitis, locomotor ataxia, neuralgia and angina pectoris furnish the best examples of *paroxysmal pain* and nearly all radiating pains are of this type.

Colic.—The term colic applies chiefly to paroxysmal abdominal pain caused by overstretching or spasm of a hollow muscular organ, and more often, both combined. It includes that of biliary or renal calculus, appendicitis, lead poisoning, floating kidney (Dietl's crises*), mucous colitis, spastic constipation, strangulated hernia and abdominal aneurysm.

Hollow viscus pains.

Many of these attacks were formerly covered by the convenient term "gastralgia," but modern progress has proven that an actual *primary* gastralgia (neuralgia of the stomach) is so rare as to be almost negligible.

An almost obsolete term.

Cases of simple transitory colic *due to acute indigestion are common in both children and adults, but the utmost caution must be observed in the interpretation of an apparently simple abdominal pain, particularly if unduly persistent and associated with fever and tenderness on deep pressure.*

Transitory colic.

Intestinal Parasites.—*In all obscure cases of abdominal colic or indeed of chronic intestinal disturbance the stools should be carefully examined for parasites or their ova.*

Appendicitis.—*Many cases begin with general abdominal pain of a colicky type and twelve or twenty-four hours may elapse before it becomes distinctly localized at a point of maximum tenderness in or near the area lying midway between the anterior superior iliac spine and the umbilicus (McBurney's point) and associated with a more or less sharply defined defensive rigidity of the right rectus muscle.*

Onset.

McBurney's point.

The tenderness may be apparent before the pain becomes localized and

* Much less common than formerly supposed and usually attributable rather to pyloro-spasm, gall-stones, duodenal ulcer, spastic constipation, renal calculus or pyelitis.

Ureteral colic.

both are simulated by certain cases of renal colic in which a small calculus has become engaged in the lower portion of the ureter and, in children especially, by certain right lobar pneumonias. Nausea and vomiting at the onset, with fever, assist the diagnosis.

In many instances encountered by the author, the referred pain of a developing pleurisy or pneumonia has been mistaken primarily for appendicitis.

Chronic cases.

Chronic appendicitis often remains unrecognized or is only revealed by exploratory operation, but usually it may be determined by the *history, by temporary exacerbations, or rarely by actual palpation of the affected member or manipulative rœntgenography.*

Colon inflation.

The disease is frequently associated with symptoms of chronic indigestion of an indeterminate type. Appendix pain induced by inflation of the colon is said to resolve doubt in many cases and sharply localized distress occasioned by pushing inward upon the cecum, the fingers having first been deeply forced to its right side, is a sign of considerable value.

Pain, character and distribution.

Renal Colic.—*Sudden, intense and agonizing paroxysmal pain may result from stone or gravel, either in the kidney pelvis or when passing downward through the ureter. It has a maximal point over the kidney posteriorly and tends to shoot downward over the course of the ureter into the groin along the inner portion of the thigh and into the testicle, which is oftentimes retracted during the paroxysms. There may be grunting and straining expulsive movements.*

Usually afebrile.

Unlike appendicitis, with which it is not infrequently confounded, renal or ureteral colic is usually afebrile, and dysuria and frequent micturition are associated with albumin, blood, and oftentimes with gravel or actual calculi in the urine.

Nausea and vomiting are common, and in several instances of such colic, as well as in acute lobar pneumonia, the author has known of narrow escapes from or actual operation for appendicitis, apparently because of temporary lodgment of calculi in the lower end of the ureter on the one hand or misleading referred pain on the other.

Pressure point.

In renal colic, tenderness is usually found over the kidney posteriorly, and often anteriorly, being best elicited by deep pressure over the twelfth rib or, according to the author's experience, just beneath it at the acme of deep inspiration.

Ureteral and vascular kinks.

The Dietl's crisis of floating kidney may be associated with pain of the same type, with more or less tenderness over the region occupied by the displaced or displaceable organ or even a decided intermittent hydronephrosis, but it more closely simulates gall-stone colic than renal colic, so far as pain distribution is concerned.

Somewhat *passé*.

The incidence and severity of supposed Dietl's crises bear but slight relation to the degree of kidney mobility and in the light of recent knowledge we must regard this condition as a possible but extremely rare manifestation.

Gall-stone Colic.—*In typical cases this is indicated by the sudden onset of intense paroxysmal pain in the epigastrium or right hypochondrium, radiating both downward and upward, but chiefly upward, being often felt in the right shoulder-joint and under the right scapula. It is associated with localized*

Pain and tenderness.

tenderness on deep, forcible, sustained, upward and backward thumb pressure over the gallbladder throughout a deep inspiration followed by deep expiration.

It is accompanied by nausea or vomiting, which latter does not give as much relief to pain as in the case of ulcer of the stomach or duodenum, and may or may not be associated with jaundice.

Colon Colic.—*A paroxysmal attack oftentimes nearly or quite as severe as that of many renal colics may occur in cases of mucous colitis or spastic constipation.**

A source of error.

It is usually left-sided, the maximum point being over the region of the kidney, posteriorly, but ordinarily it lacks the typical thigh and groin radiation of renal colic and is either wholly or in a considerable measure relieved by emptying the bowel with hot enemata.

Lead Colic.—*A paroxysmal, diffuse, persistent pain, associated usually with obstinate constipation and relieved rather than increased by pressure,* should lead one to search for a "lead line" on the gums or that substance in the urine and inquire as to sources of possible lead poisoning.

Seek gingival "lead line."

Tabetic or Splanchnic Crises and Abdominal Aneurysm.—*The curious paroxysmal pain associated with the crises of locomotor ataxia, the obscure pain of* **spastic splanchnic** *abdominal crises, the referred pain of myocardial origin or abdominal aneurysm must always be borne in mind.*

Diagnostic pitfalls.

Colic Due to Gastric Ulcer.—*Violent paroxysmal pain may be encountered in connection with certain cases of active gastric ulcer and the pain is usually epigastric, deep-seated and often seems to pass through to the back.* It is usually associated with nausea and vomiting which ameliorates or relieves the pain and in its recurrences bears a definite time relation to the taking of food.

Pressure Tenderness in Ulcer.—During acute attacks the pain of gastric ulcer may be relieved by pressure; more often, however, as in the chronic form, any pressure is distressing and the maximum tenderness is distinctly localized. Dyspepsia, anemia and often a history of antecedent hematemesis may assist the diagnosis. *In by far the greater number of ulcers, such violent and localizing symptoms are lacking.*

ACUTE INTESTINAL OBSTRUCTION.—*In general, acute obstruction is characterized by severe or extreme, paroxysmal, colicky pain, becoming continuous, the vomiting of stomach contents, bile, and ultimately of fecal material. The obstinate constipation is often preceded by evacuations from that portion of the bowel lying below the point of obstruction, and in acute intussusception there is a characteristic or suggestive bloody dysentery. Tenesmus without bowel discharges is present in some cases of volvulus.*

Misleading diarrhea.

Symptoms Vary with Site.—If the obstruction involves the colon there is less vomiting, but marked abdominal distention and a relatively slight indicanuria. In obstruction of the lower ileum or ileocecal valve, the distended coils may form a "*ladder pattern*" on the abdominal wall and, if in the sigmoid or rectum, the descending, transverse and ascending colon may be projected by distention as a visible horseshoe curve.

Important distinctions.

"Ladder pattern" and "horseshoe curve".

* Of late, this condition is attributed by some authorities to insufficiency of the ileocecal valve but the sequence may be the reverse.

Ileocecal tumor.

An actual tumor is rare save in fecal accumulation or intussusception. In the latter condition three-fourths of the cases represent an obstruction at the ileocecal junction. It is most often encountered in infants, rarely in young adults.

Adults chiefly.

Volvulus.—Obstruction due to axial twist seldom occurs in youth, most often involves the sigmoid flexure or the region of the cecum and is associated with marked meteorism, rigidity and tenderness. Antecedent constipation

Sigmoidal cases.

and flatulence usually exist and in sigmoidal cases the obstruction may be so low as to make the condition evident when an attempt is made to introduce a large enema.

Many possible causes.

Strangulation.—In the adult this is the most frequent source of acute obstruction and is usually caused by old adhesions, various openings and pockets, slits in the mesentery and omentum, rupture of the diaphragm, hernias, and adhesions of the tip of a persisting Meckel's diverticulum.

Often a deadly combination.

Intestinal Paralysis.—In connection with abdominal operations and as a result of sepsis and peritonitis, there may be an entire absence of intestinal motility which constitutes what is essentially an acute obstruction associated with general distention. The stomach is often acutely dilated and overdistended with liquid, and there is an entire absence of normal sounds upon auscultation of the abdomen.*

Common and often misleading.

Fecal Impaction.—A fecal mass gradually accumulating in a sluggish or neglected bowel may be found either at the cecum or sigmoid flexure. It less frequently occupies the splenic or even the hepatic flexure, and may occlude the lumen wholly or contain a central channel through which a certain amount of material may pass. *A localized peritonitis with pain and tenderness may or may not be present in extreme cases, and in the absence of marked distention or a very thick wall, the mass is readily palpable.* The condition

Channeled impactions.

occurs as a result of chronic neglect of the bowels, chronic partial obstruction,

Cause and associations.

and in the aged, apathetic, hysterical or insane, may lead to serious errors in diagnosis if not considered in the differentiation of abdominal tumors (see "Abdominal Tumors").

Ancient ulcerations.

Of tumors, strictures and occlusion by foreign bodies little need be said. Cicatricial contraction results usually from old ulcerations, whether syphilitic, tuberculous, dysenteric or simple; less often from adhesion. Occlusion by tumor may be due to development of local growths, direct pressure, or associated adhesions.

General Comment.—Early and correct diagnosis is of cardinal importance in connection with intestinal obstruction, hence the leading features should be kept clearly in mind.

Meteorism, vomiting and indicanuria.

Important Diagnostic Points.—*The higher the obstruction the less is the meteorism, the earlier the vomiting, the more rapid the transition to fecal vomiting, the higher the grade of indicanuria, the earlier and more severe the collapse and urinary suppression.*

* Prompt emptying of the stomach by the stomach tube, maintenance of the *prone* position, and elevation of the foot of the bed, is oftentimes of the utmost importance in such cases, huge amounts of fluid being present in many instances.

Tumor and bloody stools.

Associated tumor points to an actual growth, intussusception or a fecal impaction.

Intussusception is most frequent in infancy and childhood, and the tumor is usually in the ileocecal region or in the sigmoid flexure. Marked tenesmus and bloody stools are most frequent in intussusception. In intussusception and in volvulus the lack of fluid capacity of the lower bowel as tested by enemata may be an important aid. Volvulus is most common in the fourth decade.

Ribbon or pencil-like stools.

The passage of ribbon-like or greatly narrowed cylindrical stools suggests partial obstruction, but may occur in cases of fecal impaction possessing central calibration or be due to spastic constipation, hemorrhoids or rectal spasm.

Misleading stools.

Furthermore, although acute constipation with retention of both gas and fecal matter is the cardinal sign of complete obstruction, the unoccluded lower portion of the bowel may still be able to eject its original content.

Fecal vomiting may require twenty-four to forty-eight hours to develop fully its characteristic brownish color and specific odor.

Foreign bodies of any kind may cause obstruction and are seldom suspected unless a clear history is obtained. Most frequently the victims are children or mentally defective patients.

Unusual causes.

Obstruction by Drugs, Worms, Etc.—The persistent administration over long periods of large doses of bismuth or magnesia may cause obstructive masses, and among other rare conditions are bunches of intestinal worms, masses of hair, or massive gallstones, which have made their way by ulceration from the gall-bladder to the bowel.*

Anal relaxation.

Obstruction may be so low down as to be felt by rectal palpation; and in descending colon and sigmoid obstructions the sphincter may be strikingly relaxed.

Fever is usually present, but is ordinarily slight, thirst is excessive and in a few days, the obstruction not being relieved, collapse appears and terminates promptly in death.

Sigmoidoscopy and palpation.

Diagnosis of Obstruction of the Lower Bowel by Inflation and Liquid Injections.—An anesthetic is necessary and should be pushed to full relaxation. Rectal palpation and direct inspection with the proctoscope and sigmoidoscope should be followed by the introduction of a long tube (Kelly's tube). Oftentimes the whole hand may be inserted though this procedure is not without danger. For the injection of water which is in every way superior to air, the patient should be placed in the knee-chest position, the warm water injected slowly under gentle pressure and its passage determined by both percussion and auscultation.

Injection of air or liquid.

Ileocecal valve a barrier.

Normal Colon Capacity.—A normal colon should hold about 8 liters (quarts) and the ileocecal valve *usually* blocks any flow into the lesser bowel. In children the injection may be varied according to age, though even an infant can take from 1 to 1½ liters.

COMMENT.—*The following points should be remembered in connection with* **abdominal pains:**

(*a*) **Jaundice** when present and associated with colic and localized tender-

* The only way that an *excessively large* gall-stone can escape.

ness suggests gall-stone colic, but its absence is of little consequence in disproving that diagnosis.

Afebrile vs. febrile colics.

(*b*) **Fever** is entirely absent, as a rule, in colon colic, spastic constipation, lead colic, gastric ulcer, and abdominal aneurysm. It is almost invariably present in acute appendicitis, frequent and sometimes of a septic type in gall-stone colic, and absent usually, but not invariably, in renal colic.

Significant findings.

(*c*) **The Urine in Abdominal Colics.**—*The examination of the urine should never be omitted* and may reveal the blood, pus cells and albumin of calculus or pyelitis, the high-grade indicanuria of ileus, the pale color and increased quantity suggestive of hysteria, actual gravel or the stone fragments of renal calculus.

(*d*) **Blood.**—The examination of the blood may reveal a leucocytosis pointing to an acute infective process; an anemia of etiologic or diagnostic importance; blood parasites, or the basophilic granulation of red cells suggestive of lead poisoning.

Sudden onset.

(*e*) **Onset.**—*In nearly all forms of abdominal colic the onset is sudden, with few or no prodromata, and nausea and vomiting almost invariably occur in the obstructive or inflammatory conditions.*

(*f*) **Misleadingly Referred Cardiac Pain.**—One of the most interesting manifestations of cardiac insufficiency is pain or discomfort in the upper abdomen, which may be so severe as to simulate gastric or duodenal ulcer or even gall-stone colic, though usually of the milder type which leads the patient to blame his stomach for a peculiar sense of constriction or oppression which may or may not also be felt under the sternum or over the precordium. *One of the commonest symptoms of cardiac insufficiency or overstrain in middle-aged patients is epigastric discomfort.*

A fact of importance.

Often determinative.

(*g*) **Past Illnesses.**—These are often of great importance in the differential diagnosis of obscure abdominal pain. Preceding attacks of renal colic, appendicitis, spastic constipation, gall-stones, gastric or duodenal ulcer and the like, may have been clear and well-defined, and often lead one to an earlier and more accurate diagnosis of the individual attack than would have been possible in the absence of such a history.

Examine for hernias.

(*h*) **Hernias.**—*Owing to the common failure to systematically examine hernial regions in cases of obscure abdominal pain, nothing is more commonly misinterpreted or overlooked than an incomplete hernia or one of unusual location.*

Weakened hernial rings.

Abdominal pain occurring during the day and absent at night when the patient is in bed, is especially suggestive, and actual protrusion may be absent though the application of a good truss to a relaxed hernial opening may relieve all symptoms.

Important and Obscure.—*Obscure dyspeptic symptoms, painful or painless, with or without nausea or apparently causeless vomiting, may arise from a tiny umbilical hernia, so small as to escape the eye or finger. Pain, localized or widely diffused, follows firm pressure over the umbilicus in such cases.*

Necessary data.

HEADACHE.—The following points should be considered, viz.: (*a*) *Location.* (*b*) *Character.* (*c*) *Severity.* (*d*) *Radiation.* (*e*) *Time of occurrence.* (*f*) *Duration.* (*g*) *Local tenderness.* (*h*) *Hereditary predisposition.*

(*i*) *The effect of internal medication* (in syphilis, anemia, malaria, etc.). (*j*) *The effect of external medication* (shrinkage of turbinates, paralysis of accommodation, etc.).

Anemic headache *affects either the forehead, orbits, vertex or occiput,* being more often neuralgic in type. It is usually moderate and associated with a sense of pressure, *but in extreme chlorosis is occasionally so severe as to resemble the headache of meningitis.*

Ordinarily mild.

Bilious or Dyspeptic Headache.—This toxemic variety includes as etiologic factors or associated conditions, gout, jaundice, the dyspepsias, and constipation. *It is irregular in duration and occurrence, frontal and congestive in type and associated with nausea.* There is a subjective sense of pulsation; it is increased by motion and by stooping, and more or less promptly relieved by emesis and catharsis. *Frontal sinus headache is often interpreted wrongly as bilious headache.*

Toxemic.

Frontal and congestive.

Brain Tumor.—This causes persistent headache, usually increased at night, associated with nausea and vomiting, and sooner or later with optic neuritis.

Brain Abscess.—This may cause severe and persistent headache resembling that of brain tumor, but is usually accompanied by a septic temperature curve, *and optic neuritis is usually absent.*

Eye-strain headaches may be either occipital, orbital or frontal. They are usually associated with the use of the eyes for close work, or, in certain cases, for distance; are relieved by sleep and properly fitted lenses and are frequently associated with conjunctivitis, tenderness of the eyeball or eye muscles and, sometimes, blurred vision.

SINUS HEADACHES.—These headaches have only recently been assigned the important place they deserve.

A common type.

Inflammation of the accessory nasal sinuses with blocked drainage is one of the most frequent causes of headache of a type often attributed to other causes, and it would be humiliating to know how many headaches of this origin have been treated vainly as sick headache, uterine headache, facial or cervical neuralgia, bilious headache and especially that of eye strain.

Source of error.

Sinus headaches vary somewhat in their manifestations but are alike in their tendency to start with mild or dull pain and increase more or less rapidly to an almost unbearable intensity. They are, moreover, for the most part, strikingly and suddenly ameliorated by the reëstablishment of sinus drainage, by means of vasoconstricting douches or special local procedure, and are significantly associated with and responsive to nasopharyngeal congestion.

Significant factors.

Frontal Sinus Headache.—The pain is chiefly frontal, usually throbbing and more intense in the early morning and often increased by the movements of the eyeball. *Tenderness may be marked over the frontal wall of the sinus or the inner aspect of the orbital roof.* Like the bilious headache, it may be intensified by stooping, sneezing, or coughing, and is often associated with intervals of vertigo, nausea, and even vomiting.

Antrum of Highmore Headache.—This is characterized by a sense of pressure and tension over the outer wall of the sinus and very often by severe frontal pain and toothache.

Ethmoidal Headache.—In this form the pain, though often diffuse, usually affects chiefly the region of the root of the nose.

Sphenoidal Sinus Headache.—This may be widely diffused but is usually manifested by maximal pain at the occiput.

Silent cases.

It should be remembered that each or all of the sinus lesions may cause general headache and that chronic sinusitis may be quite silent except at intervals when, through a coryza, an acute or subacute process supervenes and blocking occurs.

Inquire as to use of drugs.

Drug Headaches.—The overuse of drugs may lead to headaches which have no distinguishing features. A dull throbbing headache associated with ringing in the ear often follows the administration of a salicylate or quinin in full doses.

Arterial hypertension the rule.

Bright's Disease.—The headaches of nephritis are of varying localization and for the most part associated with arterial hypertension. Neuralgia is a frequent complication of Bright's disease, but the true uremic headache, which in chronic interstitial nephritis may appear years before any acute outbreak, is usually maximally occipital, and in all forms of nephritis *may* be the immediate precursor of uremic coma or convulsions.

*It is often an early morning headache of moderate or extreme intensity and relieved by black coffee, but sometimes closely simulates migraine. High arterial pressure from other causes produces headache of a similar type.**

Arteriosclerosis.—*These headaches are seldom severe, though often persistent; are associated with transient vertigo, impaired memory and intellection; and are often forerunners of cerebral hemorrhage.*

The cause is suggested by rigid temporals or radials and by the fact that the headaches are often relieved by the administration of such a drug as nitroglycerine.

Unrecognized factors.

In the author's experience the so-called arteriosclerotic headaches have been seldom unassociated with chronic renal changes and arterial hypertension. Furthermore, they are often accompanied by distinct cardiac weakness and varying degrees of dilatation and may respond promptly to treatment directed solely to the relief of this latter condition.

Therapeutic test.

Malaria.—Both persistent headache and obstinate neuralgia may be encountered in connection with malaria. Unless it is distinctly periodic, the only characteristic feature is its subsidence under appropriate drug treatment (quinin).

Migraine (*"Sick headache," "megrim," "hemicrania"*).—Heredity plays a large part in migraine which is regarded by some authorities as an epileptic

* Unilateral, predominantly temporal headaches are quite common in certain cases advanced chronic nephritis with hypertension and in such instances apoplexy constitutes a relatively common complication. It would seem from the author's observations that in such instances the corresponding side of the brain is the more likely to be the seat of such a lesion.

equivalent or actual variant.* It is characterized by its unilateral localization, nausea, vomiting, and subjective ocular phenomena.

Females are attacked most severely and typically and a considerable proportion of the cases (30 per cent.) commence in childhood (fifth to tenth year. Eye strain, adenoids, carious teeth, pelvic disease, food sensitization, gastric disorders, overexcitement, overstudy, shock, unhygienic surroundings, and dietetic errors are common *exciting factors*, but all are often lacking.

Renal cases.

The author has seen a large number of typical cases of the most extreme and persistent type, occurring in middle-aged women especially, associated with a renal insufficiency, without other signs of the chronic nephritis with which headaches of this type are occasionally associated.†

Periodicity.

Periodicity may be marked even to the hour of the day, or it may bear a definite and invariable time relation to menstruation. Unquestionably, as suggested, this condition is often confounded with the headaches of renal insufficiency and arterial hypertension.

Auræ.

Symptoms.—The attack appears usually in the early morning, often preceded by localized numbness and tingling, hippus, vertigo, visual paresthesias (*flashes of light*,‡ *fortification figures*), cramps, spasms, or even *aphasia* associated with mental excitement or decided depression.

Hemicrania.

The headache is unilateral, usually right-sided and sharp, stabbing or boring in character. Nausea and vomiting are common symptoms, movement, light and sound increase it; it may become bilateral and extensive and pallor or flushing may be marked.

Duration.

Usually the duration is one day or less, sometimes two or three days, a night's sleep and oftentimes a long day nap bringing relief. In children the *possibility* of a larval meningitis or brain tumor must be remembered.

It may be present in childhood and absent in adult life and even when persistent often becomes less frequent and less severe in middle age.

Clavus.

Hysteria.—*There is no headache characteristic of hysteria and the classical "clavus" (pain at the vertex as if a nail were being driven into the skull) has been encountered but rarely by the author and in such instances it was associated with extreme anemia or arterial hypertension.*

* Both statements rest upon decidedly insufficient grounds one would think after examining the testimony. Of 3000 individuals questioned, only 2 per cent. were found to have been wholly free from headaches identical with or closely allied to migraine. "With such a wide prevalence heredity is a negligible factor. It is like the doctrine of original sin" (Jelliffe). As to the close relation of migraine to epilepsy one can say only, that when *radical differences* exist between two ailments and absolutely nothing is known as to the pathology of either, we have no right to link them together because of minor and fragmentary resemblances. To attach, either by direction or indirection, the taint of epilepsy to this common condition is wholly wrong, until some substantial proof is obtainable. Exactly the same protest may be made against the universal assumption that somnambulism is an epileptic equivalent. Anaphylaxis is assuming great importance as an etiologic factor.

† Several of these interesting patients, originally recorded about fifteen years ago, have since died of a frank interstitial nephritis with hypertension.

‡ "Scintillating scotoma."

A common form.

Asthenic and Psychasthenic* Headache.—Usually mild and indeterminate, frequently occipital, as in mild uremic headache, the important characteristic of this form is its morning maximum and the tendency to disappear after eating and under physical exercise and mental preoccupation.

Points of tenderness and distribution.

NEURALGIA.—*The pain of severe neuralgia is acute, radiating, distinctly localized and associated with superficial points of tenderness. Its characteristic feature lies in its definite relation to known nerve trunks, their points of exit and their distribution.*

Seek source of toxemia.

It may affect any portion of the body, though most common in the head, and is probably always an expression of chronic infection, toxemia, pressure or injury, though frequently *initiated* by extreme fatigue, particularly when this is associated with subnutrition and exposure to cold and damp. Severe attacks are remarkable for their persistence, intractability, and tendency to irregular or periodic recurrence.

Sinuses, teeth and kidneys.

Sinus infections and peridental septic foci doubtless account for a large proportion of so-called neuralgic cases affecting the head, and neck and an astonishingly large number of such headaches occurring in the author's middle-aged patients are associated with high arterial tension.

Seek focal infection.

The possibility of an actual nephritis or a renal inadequacy, as indicated by an insufficient excretion of urinary solids should always be borne in mind, and invariably a definite source of chronic infection or toxemia must be considered and sought.

The tender points in neuralgia represent points of nerve trunk emergence, their entrance into muscles, or their terminal filaments. Maximum tenderness is usually easily determined and widely diffused sensitiveness of the most extreme type is sometimes encountered.

Often agonizing and intractable.

Trifacial Neuralgia (*Tic douloureux*).—*Either the ophthalmic, the upper maxillary or the lower branches of the trifacial nerve may be affected; the last being the most common; the first, the most severe form. In all varieties the pain is intense and the points of tenderness marked.*

Ophthalmic.—If the ophthalmic division be chiefly affected, the pain is especially severe about the region of the eye, which is often injected and tender. Lachrymation may be present, and there is marked tenderness in the supraorbital region, over the bridge of the nose, and occasionally at the occiput and upper cervical spines. *Transient blindness may occur in severe cases.* This form and the next one are frequently associated with accessory sinus and dental infections.

Superior Maxillary.—*If the upper maxillary* is chiefly affected, the maximum tenderness is at the infraorbital canal and disease of the antrum of Highmore is often a cause.

Inferior Maxillary.—*With involvement of the lower branches* the pain radiates to the ear, and along the lower teeth, the maximum painful points corresponding to the auriculo-temporal nerve.

Herpes frequent.

All branches are usually more or less affected, and a severe herpetic eruption

* The term is used in its literal sense as meaning "brain fag" or the mental instability usually called "neurasthenic" (see "Neurasthenia").

may occur along the track of the superficial nerves in all three forms. The condition of the maxillary and frontal sinuses should always be determined.

Cervico-brachial Neuralgia.—This variety, frequently associated with rheumatic affections, involves especially the sensory nerves of the brachial plexus. It is not infrequent, occurs chiefly in women, may be due to remote primary causes, such as locomotor ataxia, uterine disease, sinus or tonsillar infections or carious teeth, and is characterized by the distribution of the pain and points of maximum tenderness, which may be over the ulnar nerve, at the elbow, near the wrists, in the axilla, at the inferior angle of the scapula, over the deltoid muscle, or even on either side of the lower cervical spine. Herpes, anesthesia and vasomotor disturbances may occur.

Maximal tenderness.

Cervico-occipital Neuralgia.—This affects the sensory branches of the upper cervical nerves, is often caused by cervical caries and usually associated with a point of maximum tenderness midway between the mastoid process and the atlas.

Intercostal Neuralgia.—This occurs most frequently in those debilitated by overwork, malnutrition, toxemia or co-existing diseases, and chiefly in young adults of the female sex. It is characterized by sudden pain in the chest, of the neuralgic type and but slightly affected by respiration, yet following the intercostal distribution. The maximum points of tenderness correspond to the exits of both dorsal and anterior branches *and it chiefly affects the left side.* It is ordinarily of brief duration, but may last several weeks.

A Source of Erroneous Diagnosis.—Intercostal neuralgia is of special importance in connection with angina pectoris, pleurisy and the acute pain *of intra-abdominal diseases.*

Furthermore, the hitherto accepted statement that intercostal neuralgia chiefly affects the left side is significant. Mild pains of cardiac origin and even the severer atypical manifestations of angina pectoris are frequently so miscalled.

A common error.

No less important is the relationship of thoracic pain and tenderness to the dilated, insufficient drop heart so often encountered in the asthenic individual.

Herpes zoster cannot be distinguished from intercostal neuralgia until the eruption appears. It is usually unilateral, rarely bilateral and is associated with an *herpetic eruption* appearing in patches over the affected intercostal nerve.

Its pain is atrocious, and inasmuch as it may precede the eruption by many hours, is oftentimes most puzzling and misleading if zoster be forgotten.

Sciatica (*Sciatic neuritis, sciatic neuralgia*).—In the presence of the ordinary etiologic factors, toxemia or actual infections, sciatica is usually initiated by exposure to cold, wet and excessive fatigue, direct injury, pressure from pelvic tumor, a faulty chair seat, chronic constipation, preëxisting disease of the vertebræ, disease or relaxation of the sacro-iliac joint or lesions of the spinal cord.

Leading Symptoms.—Acute seizures are characterized by a sudden onset, the intense pain following the course of the sciatic nerve, and often extending well up into the lumbar region. Intervals of comparative immunity alter-

Pain characteristics.

nate with paroxysms of a most excruciating type, the pain sometimes shooting from above downward and giving the sensation of an actual shock as it reaches the heel.

Subjective numbness, tingling, and disturbed temperature sense may be present. Many cases with imperfect clinical manifestations occur. Very similar symptoms may be encountered in cases of impaired circulation due to productive or obliterative endarteritis of the lower extremities. The pulsation of the dorsalis pedis and posterior tibial arteries should, therefore, invariably be tested and bilaterally compared.

Pressure Points.—Pressure applied to the middle of the thigh posteriorly, the sciatic notch, the posterior aspect of the knee and calf, the external malleolus or the dorsum of the foot reveals the points of tenderness.

Tenderness.

Sacro-iliac Pain.—This is usually a dull pain felt chiefly over the sacro-iliac joints, and, aside from actual disease, is encountered usually in joint strain or displacements. It is relatively common in pronounced "chronic congenital asthenia" in association with the tendency to poor nutrition, delicate physical structure, gastroptosis, nephroptosis, and the generalized muscular and ligamentous relaxation which characterizes that common ailment.

Common cause of backache.

It is one of the commonest causes of chronic backache in women and may often be promptly relieved by a proper hip band, belt, or special corset which steadies and fixes the sacro-iliac joints.

Easily tested.

The condition, if of any marked degree, is readily detected by the pain in the joint and spasm of the musculature which results from any attempt to stoop low with straight knees.

Cervical Ribs.—This congenital anomaly may be bilateral (80 per cent) or unilateral, palpable or impalpable, troublesome or symptomless, and the accessory rib itself may be either long, or, short and "stubby," but all, or practically all arise from the 7th cervical segment.

If bilateral they may be wholly unlike in the degree of development.

Long symptomless.

If symptom-producing, this effect is manifest only in adult life, coincident with increasing rigidity of the bony structures and the dropping of the shoulders which usually initiates and confirms the pressure symptoms. Even when bilateral and palpable as to one, the other may be revealed only by roentgenography.

Oftentimes both are easily detected by the fingers *in the supraclavicular fossa.*

The condition is unquestionably commoner than previously assumed, but, relatively, is rare.

Source of symptoms.

Symptoms reflecting pressure upon the brachial plexus (chiefly represented by neuralgia or neuritis along the area supplied by the nerve of Wrisberg or the ulnar), the subclavian artery and its vein. The phrenic is sometimes affected and in a few instances disturbances of the sympathetic have been reported.

The condition should ever be in one's mind when meeting obscure symptoms such as might result from this condition.

Effect of posture.

In some instances pain is present only when certain postures are assumed

and is relieved by standing erect with shoulders thrown back, raising the arms above the head or sleeping on the back without a pillow.

Miscellaneous Neuralgic Pains.—Pains of a neuralgic type may of course occur in any region of the body, and be confined to a single trunk or even a single terminal. Thus it may be limited to the breast (*mastodynia*), finger (*digital neuralgia*), the plantar nerves (*plantar neuralgia*), or be associated

FIG. 25.—Double cervical rib. A frequent source of diagnostic errors, with relation to neuritic or neuralgic pain and obscure localized circulatory disturbances. (*Dr. Frank S. Bissell.*)

with the stretching of the plantar ligaments in incipient flat-foot, in which case the pain is in some instances limited to the third and fourth metatarso-phalangeal joints (*Morton's foot*).

Persistent moderate pain or discomfort, associated with hyperesthesia about the *lower zone* of the left breast, is a common and important, though inconstant, sign of a cardiac insufficiency, often temporary, with or without associated valvular lesions, and in most instances easily remediable.

In many cases the area of maximum tenderness follows the lower left border of a dilated insufficient laboring heart as it contracts toward the median line under rest and cardiac stimulation.*

Lumbo-abdominal neuralgia is characterized by pain in the back, buttocks and loins, radiating to the genital and hypogastric regions, and by areas of *superficial tenderness*. It is commonly associated with chronic toxemia, general debility, chronic constipation, disease of the pelvic organs and exposure to wet and cold under conditions of fatigue.

In femoral neuralgia the pain is limited to the front of the knee and outer front of the thigh. In diseases of the hip-joint the reflex knee pain (inner aspect) is extremely common.

NEURITIS.—The distribution of neuritic pains and their general characteristics are of necessity much the same as in neuralgia, though much more severe and persistent.

Pain Severe and Persistent with Exacerbations.—The disease may be acute or chronic, circumscribed, and limited to a single trunk, or widespread and *multiple*.

Distinctly inflammatory in type it tends to produce primarily and essentially a degeneration of the affected peripheral nerves. Aside from the more persistent and constant character of the pain, and its intensification by movement or passive congestion of the affected area, the exquisite tenderness on pressure over the nerve itself is characteristic.

Tenderness extreme.

Furthermore, in severe cases there is a special tendency to marked cutaneous hyperesthesia followed by anesthesia and such marked changes as redness or pallor, edema or joint swelling and herpetic eruptions.

In the extremities the skin often becomes characteristically glossy through trophic changes, and the disturbances of nutrition are indicated by muscle atrophy, the reaction of degeneration, loss of dependent reflexes, changes in the nails, loss of hair, desquamation of the epidermis, etc. True swelling of the nerve trunk and an erythema indicating its course may be present and if the motor fibers are affected, changes appear, varying from easily induced fatigue and slight loss of power to actual paralysis.

Trophic changes.

Many, if not most, of the cases characterized by shoulder-girdle pain, a type extremely common and usually termed neuritis, are attributable to bursitis rather than neuritis.

MUSCULAR RHEUMATISM.—This common and painful ailment most often affects the lumbar muscles (*lumbago*), muscles of the chest (*pleurodynia*) and the cervical muscles (*rheumatic torticollis*).

In all its forms, the chief characteristic is pain on movement, which produces more or less characteristic attitudes, almost complete loss of function and voluntary rigidity.

Tenderness on pressure.

Fundamental Diagnostic Points.—*One of the most important points of distinction as between this painful affection of the muscles, and neuralgia,*

* A beautiful demonstration of the onset of such pain and localized tenderness was recently given by an acute gross dilatation of the heart, occurring in the author's office, from the effect of fright.

neuritis, pleurisy, and such other affections as it may simulate, lies in the fact that diffuse tenderness of the involved muscles themselves, when grasped, is a prominent feature; that pain is distinctly related to muscular contraction, and that it is absent or greatly ameliorated when the muscle is at rest.

Muscle contraction pain.

The disease is due undoubtedly to a definite infection in most instances and is closely allied to that form of acute or subacute rheumatism which primarily and chiefly affects the tendons.

Seek a focal infection.

GENERAL COMMENT.—*In the majority of painful ailments the seat of pain represents more or less exactly the location of the affected part, and any attempt to list the enormous variety is futile and useless.*

The more important referred pains are described under the diseases of the different organs.

Shoulder Pain.—It should be remembered, however, that shoulder pain may be due to local disease of the joint, sprains or fractures, neuralgia, neuritis, bursitis, tenosynovitis, myalgia, trichinosis, disease of the diaphragm or of the colon flexures, *angina pectoris, aneurysm, the minor anginas of cardiac insufficiencies of various types*, and, on the right side especially, to inflammatory diseases of the liver, gallbladder, duodenal abscess, and, it is said, to movable kidney.*

Pain at the inside of the knee *suggests hip-joint disease; down the front of the thigh, ovarian and testicular disease, or sometimes a developing or established femoral or inguinal hernia.*

Knee, groin, or thigh pain.

Pain radiating to the fold of the groin *or to the testicle is common in renal colic.*

Headaches *have already been discussed and the author is convinced that they will not conform to any arbitrary classification according to location, though such has been attempted under the older, etiologically inaccurate, grouping.*

Futile classification.

Sacral and mid-lumbar pain is frequently due to the drag of a large, fat, pendulous abdomen or is of uterine origin, and movable kidney is often associated with a painful area over the corresponding sacro-iliac joint, probably due to coincident ligamentous relaxation so common in the congenital asthenia of which visceroptosis is part and parcel. Neuralgic coccygeal pain indicates coccygodynia, but equally sharp pain may accompany rectal fissure.

Lumbar pain.

Lower spine.

Aside from these factors backache, usually low down, may indicate asthenia, pelvic disease, excessive fatigue, spinal caries, renal disease (loin weariness is especially common), excessive venery, sacro-iliac disease, strain or displacement and various other conditions.

"Flat-foot" must be borne in mind as a cause of pain, not only in the feet and legs, but, occasionally, of the back as well.

Common and important.

Pain under the scapula, when not pleuritic, most commonly indicates hepatic lesions if on the right side; cardiac, gastric, colonic or splenic disturbances, if on the left; but may be due to any one of many other causes.

* The last is a most dubious statement with relation to one of the commonest of conditions. No such connection has been observed by the author.

Lower-abdominal-quadrant pain suggests colitis, sigmoid irritation, appendicitis, hernia, ovarian or uterine disease or varicocele.

Pubic pain is chiefly attributable to the pelvic organs and cystitis.

Epigastric pain covers gastric and duodenal ulcer, certain cardiac insufficiencies and actual anginas, splanchnic arteriosclerosis, early appendicitis, functional gastric disease (pyloro-spasm, neuroses, displacement), pancreatic and vertebral diseases, the referred pain of pleurisy, pneumonia, etc.

Often cardiac.

The "girdle sensation" follows the waist line, represents constriction rather than pain, and points to injuries or tumors of the spinal cord and its meninges, chronic myelitis or *locomotor ataxia.*

In the back interscapular pain is often troublesome and usually indicates mere indigestion or flatulent distention, though aneurysm, cardiac insufficiency, gastric ulcer and caries must not be forgotten.

Median dorsal pain at the shoulder level may also occur in aneurysm, pericarditis, and diaphragmatic irritation. Ulcer of the stomach may produce *median pain lower down*, with tenderness to the left of the spine over the tenth and eleventh interspaces.

Pain in the heel may be due to sciatica, gout, ovarian and testicular lesions, intra-abdominal growths or prostatic disease.

General aching is a common symptom of nervous and muscular debility or fatigue, usually precedes the onset of acute infectious diseases, and is common in acute and chronic rheumatism, trichiniasis, scurvy, locomotor ataxia, gastrointestinal or hepatic diseases and anemia.

A loaded, overstretched colon, spastic constipation or mucous colitis may produce most extreme paroxysmal distress.

TENDERNESS

Usually Indicates Structure Involved.—*In inflammations of accessible structures tenderness is associated with pain and usually conforms in position to the organ affected.*

The Head.—A tender *scalp* suggests syphilis, hysteria, rheumatism, neuralgia and migraine; tenderness of the *malar bone*, neuralgia or antrum disease; of the mastoid process, a neuralgia, periostitis, inflammation of the local gland or mastoiditis. Tenderness at the *back of the neck* suggests caries, sinusitis or asthenia; *linear or interrupted spinal tenderness*, asthenia, rheumatism, caries, periostitis, actual spinal disease or hysteria. *Lumbar tenderness* is common in lumbago and inflammatory disease of the related intra-abdominal structures; *dorsal tenderness* in advanced thoracic aneurysm or posterior, mediastinal tumors of any kind. *A tender sternum or ribs* suggests ostitis or periostitis but is often encountered in the major and minor anginas of cardiovascular disease. *Abdominal tenderness* is related to all acute and many chronic diseases of the contained viscera, and if wholly superficial suggests neuralgia, referred pleuritic pain or hysteria. *Hypogastric tenderness* is usually directly related to acute or chronic inflammation of the under-

lying structures. *The tenderness of sciatica* like its pain occurs chiefly at the sciatic notch, the middle of the thigh and knee, the ankle and heel. *Joint tenderness* is often puzzling and suggests rheumatism, arthritis deformans, synovitis, gout, gonorrhea, tuberculosis, sepsis, hysteria, or simple sprain according to the conditions developed by the case history.

PERVERSIONS OF SENSATION.—Paresthesias.—Those disturbances of sensation so denominated may be wholly unrelated to organic disease of the brain and cord. Among the more important are:

1. Subjective Sensations of Heat and Cold.—These sensations suggest asthenia, malnutrition and anemia, as well as hysteria, lateral sclerosis, syringomyelia and locomotor ataxia. In profound toxemias, such as alcoholism or in actual disease of the cord, they are usually combined with other paresthesias. The distribution may be general or local, the sensation indefinite or exactly like actual contact with a hot or cold object. Hot flushes both subjective and visible are among the commonest events of the menopause.

2. Formication and Itching.—Itching may be pronounced in hysteria, neurasthenia, chronic alcoholism or gout, lead poisoning and various diseases of the cord. It is almost invariable in jaundice and one of the signs indicating the taking of morphin (nose rubbing). Both itching and formication may antedate or follow an apoplexy and the latter is a common symptom as a result of actual irritation, diseases of the brain and cord, diabetes, cocainism and pelvic disturbances.

Morphinism and cocainism.

3. Numbness and Tingling.—Often associated with decided burning sensation, these symptoms are especially common in connection with the exacerbations of high arterial pressure of chronic interstitial nephritis, in productive or obliterative arteriosclerosis of the extremities, neuritis, or pressure irritation of nerves and the use of such a drug as aconite, in addition to the causes of formication and itching before mentioned.

Oppression.—The sensation known as precordial oppression or compression is common in mediastinal tumors, cardiac and aortic disease and arterial hypertension with myocardial disease as well as in certain pulmonary lesions. In hemoptysis it may either precede or accompany the visible hemorrhage. Epigastric oppression may also be present in gastric and hepatic disease, but is more common in the functional digestive disorders or in hematemesis. It is a very common symptom of *minor, but important*, cardiac insufficiency.

Precordial type.

Epigastric type.

Head constriction is almost invariably associated with hysteria or psychasthenia, and pelvic oppression or "bearing down," is usually due to actual pelvic disease or in the male to diseases of the bladder or prostate.

The "band sensation."

Faintness and "Sinking" Sensations.—In major *angina pectoris* such sensations, together with severe precordial oppression, extreme pain, and a sense of impending dissolution, form an agonizing and terrifying complex. Lesser degrees are common in gastrointestinal ailments (chiefly functional), concealed or open hemorrhage, in all of the cardiac diseases, in asthenia with dilated or readily dilatable drop heart, hysteria, and especially in asthenic hypochondriasis.

Serious vs. trivial causes.

Wide etiologic range.

Syncope.—This may be purely a nervous manifestation, sometimes related to terrifying sights and sounds or violent emotion. It may accompany actual disease of the heart or blood vessels, follow the aspiration of ascitic or pleural exudates, a large hemorrhage, or surgical operation, and may also be the result of pain, exhaustion, excessive heat, or the overadministration of certain drugs.

The attack.

The sudden and extreme pallor and the weakness of respiration and heart action which may cause the loss of the pulse beat at the wrist and an apparent arrest of breathing, are usually momentary and often preceded by vertigo and nausea. Temporary loss of consciousness occurs in all severe seizures and in some instances, demonstrable, transitory cardiac dilatation is coincident.

Fatal syncope.

Fatal syncope is a not uncommon event in many serious diseases and may constitute a most distressing accident in pneumonia, diphtheria, pericardial effusion, endocarditis, the removal of large exudates or transudates, and many other conditions aside from primary diseases of the heart muscle.

False syncope.

Hysterical women frequently simulate syncope but the hysterical facies, retained color, the pulse and usually, a lack of the absolute immobility and relaxation characterizing true syncope, make detection easy. In doubtful cases, sharp and sudden pressure over the ovaries may resolve all doubt as the malingerer often shows an excessive tenderness in the ovarian region.

Subjective Sensations of Heat and Morbid Flushing.—Fatigue, hysteria, pelvic disease and the menopause are the most frequent causes of troublesome flushing ("hot flashes") and in certain individuals causeless blushing is a troublesome phenomenon. The sensation of epigastric heat known as pyrosis is frequently associated with a sense of excessive fullness as distinct from the pressure sensation previously described and is most common in the functional disturbances of the stomach.

"Heart-burn."

Psychic vs. Physical fag.

Subjective Weakness.—In hysteria and asthenia or in mere temporary "brain fag," a patient may experience a sense of positive physical fatigue; or, in the former, pseudo-paralytic symptoms either transient or persistent may occur.

Test of brain fag.

In mere "brain fag" the factitious sensation of physical "exhaustion" may entirely disappear under active and strenuous exertion or be forgotten in the interest excited by a good play or book. It is frequently due to lack of exercise, the overuse of tea, coffee and tobacco, or to insufficient sleep.

May be cardiac.

Fatigue, persistent or readily induced, is also one of the commonest symptoms of cardiac insufficiency, as is demonstrated in the myocardial toxemias of acute infections and must be given its full value in diagnosis and treatment.

Important distinction.

"Globus hystericus" is a sensation of obstruction, constriction, pressure or tickling referred to the throat. It is common in hysteria, and *may* be associated with troublesome spasm, but represents too often a misinterpretation of the genuine choking, sense of oppression, compression, or clutching so commonly present in minor and major cardiac insufficiencies, with resulting errors of omission more or less disastrous to the patient.

INSOMNIA.—*This troublesome symptom may take the form of inability to sleep, disturbed sleep, starting during sleep, early waking or prolonged wakeful periods.*

Its causes, too varied to permit full discussion, vary from mere worry or temporary nervous excitement to actual disease of the cardiovascular system or of the brain itself. Causes manifold.

Inquiry should include the condition of the stomach and bowels, a careful examination of the urine, heart and blood vessels and the estimation of arterial tension, the mental work done, quantity of tea, coffee, tobacco and alcohol consumed, exercise taken, unusual sources of worry and mental strain, excessive study at night, eyestrain, and the age of the patient. Scope of inquiry.

Frequently a light lunch at bedtime, slight modification in the diet, hours of meals, relation of exercise to the meals, the cutting off of excesses of any kind or the mere admission of fresh air at night to the sleeping-room will put an end to attacks of insomnia. If asthenia be the cause, nothing short of a prolonged rest or recreation cure is of any use. Occasionally climatic change alone will produce sleep and permanently correct the disturbance, while, not infrequently, relief follows *a complete cessation of all conscious efforts or devices to induce slumber.* Trivial or intractable.

In both acute and chronic disease the amount of sleep obtained by the patient is important and should be as carefully noted as is the food intake. Insufficient sleep during the twenty-four hours is always the chief point to be determined, rather than the question of how the nights pass.

One frequently finds that naps during the day are accountable for disturbed and broken nights and that the total slumber for twenty-four hours is actually in excess of the needs of the patient.

It is perhaps unfortunate that a minimum of eight or ten hours of sleep has become a fixed standard regardless of occupation and individual requirement.

The small amount of sleep which suffices for the needs of healthy individuals who are habituated is oftentimes most striking.

Drowsiness during the day and early waking or disturbed sleep at night constitute a common symptom of cardiac insufficiency, and are often promptly relieved by appropriate measures directed to the strengthening of the heart. A much neglected cause.

Old people undergo a slowly progressive circulatory inadequacy and are, as a rule, early wakers and light sleepers, the conditions of childhood being reversed.

VERTIGO.—(*Giddiness, "Swimming of the Head"*).—

Vertigo is , in most instances, a trivial and transitory symptom depending upon impacted wax in the auditory canal, disturbances of digestion, eyestrain, or asthenia, but nevertheless should always receive careful attention. Usually trivial.

In the young the causes are usually trivial and removable, though in persistent cases *brain tumor and minor epilepsy* must always be considered. In the middle-aged and old it is much more significant. The lines of investigation indicated are as follows: The young vs. the old.

Varieties of Vertigo.—Vertigo is usually sudden in onset and intermittent or periodic in type. Usually a sudden change from the recumbent to the erect posture initiates it, and it may be limited to the early morning hours. It may take the form of a subjective sensation of whirling or falling which persists with the eyes closed, or may occur only when looking at moving objects, as in riding, driving, swaying, or swinging.

Effect of posture.

It usually disappears or is greatly relieved when the patient is lying down, but in some instances, and, notably in its arteriosclerotic form, is often increased by lying down.

Points to be Remembered.—*Persistent vertigo not distinctly associated with the eye, accessory sinuses, ear, digestive disturbances, or asthenia, demands a thorough investigation of the heart and blood vessels, the urine, the reflexes, syphilis, possible epilepsy and even brain tumor and should include an examination of the fundus oculi.*

(*a*) **The Existence of Asthenia.**—This condition covers by far the greater number of persistent or persistently recurrent cases in persons under the age of forty and is often associated with coincident digestive trouble, cardiac overstrain, eyestrain, anemia, hysteria, malnutrition, or even the habitual use of narcotics or stimulants.

(*b*) **Ménière's Disease.**—This condition is supposed to be due to disease of the labyrinth or semicircular canals and is characterized by the *association of vertigo with tinnitus aurium* and a tendency to fall to the left or right (see "Ménière's Disease").

(*c*) **Arteriosclerosis and Cardiac Disease.**—This vertigo covers the greater number of cases observed in *elderly people*. It is important as related to exacerbations of high arterial pressure and the threat of apoplexy, to aneurysm, aortic regurgitation, arteriosclerosis, arterial spasm, and other cardiac lesions or cerebral disturbances due to other causes.

(*d*) **Sunstroke or heatstroke** sometimes leaves behind a marked vertiginous tendency.

(*e*) **Eye Strain.**—Errors of refraction and ocular insufficiencies should be carefully investigated.

An aura or "*petit mal.*"

(*f*) **Epilepsy.**—Vertigo may be either an aura of major epilepsy, or represent an attack of petit mal. The condition of the stomach and bowels is of great importance and constipation is frequently responsible for recurrent vertigo and pseudo-epilepsy in spasmophilic cases.

(*g*) **Lesions of the Brain and Cord.**—Brain tumor, abscess, meningitis, cerebellar and pontine lesions, cerebral syphilis, cerebral thrombosis, general paresis, locomotor ataxia and disseminated sclerosis may be associated with varying degrees of vertigo.

(*h*) **General Diseases.**—Vertigo is frequent in auto-intoxication, gout, Bright's disease, febrile states, extreme weakness, as in severe anemias, the cachexias and convalescence from acute diseases.

(*i*) **Reflex.**—Auditory vertigo due to conditions other than Ménière's disease, as in cases of ear-drum pressure; *naso-pharyngeal vertigo due to pressure within the nose or in the sinuses; laryngeal vertigo* associated

usually with a coughing fit in laryngeal affections—are the leading reflex types.

(*j*) **Gastric Disturbances.**—These are held accountable for one of the commonest forms of vertigo.

(*k*) **Excesses.**—The habits in relation to the use of tea, coffee, tobacco, alcoholics, and the question of sexual excess, should be carefully investigated.

DYSPNEA

Clinical Definition.—*The term "dyspnea" should be limited to those cases of labored, regular, arrhythmic, or greatly accelerated, respiration in which there is insufficient oxidation of the blood, whether this be due to heart weakness, actual obstruction to the free ingress or egress of air, a diminished pulmonary area, or to other causes of impairment of its chemical exchanges. The term "accelerated breathing" or a specific descriptive term should cover other varieties.*

Subjective vs. Objective Dyspnea.—Dyspnea may be purely subjective and represent a sensation of oppression or respiratory inadequacy without marked increase in frequency or disturbance of rhythm, but severe dyspnea is usually both subjective and objective and associated with varying degrees of cyanosis.

Both are important

Subjective Dyspnea.—The mere *sensation* of dyspnea is practically limited to cardiac insufficiency, uremia, and psychasthenia, and even though orthopnea be present there is oftentimes no associated cyanosis.

Obstructive Type.—Any condition preventing the free entrance of air into the lungs will cause dyspnea, hence it accompanies severe quinsy, and marked narrowing or stenosis of the glottis, trachea or bronchi, whether the cause be direct obstruction, tumor or foreign bodies; inflammation, as in the case of laryngeal diphtheria and pneumonia; or spasm, as in croup or asthma.

Stridor.—(*Usually due to glottic obstruction*).—*Stridulous breathing is invariably of laryngeal origin and usually due to spasm, paralysis, edema, foreign bodies, membrane, tumor or severe inflammation of the glottis.*

A vitally important sign.

In certain cases it represents pressure on the nerve trunks and resulting spasm or paralysis of the laryngeal musculature, as in aneurysm, enlarged bronchial glands, massive pericardial effusions, etc. It is typified by the "*crowing*" of a croupy child, and invariably represents true dyspnea of the obstructive or stenotic type.

"Crowing."

Its diagnostic features are stridor (whistling, hissing) and a pronounced inspiratory descent of the larynx; this movement being so great oftentimes, and especially in short-necked children, as to embarrass the surgeon in tracheotomy.

The breathing is slow in obstructive dyspnea so long as respiratory power remains good.

Circulatory Dyspnea.—Other cases are referable to circulatory disturbances, especially those of incompensated mitral and tricuspid lesions, in which instances the condition is largely one of pulmonary stasis, the aëration

Pulmonary stasis.

areas being normal, but the blood current obstructed and its chemical exchanges diminished.

Emphysema and pneumonia.

In other instances both factors are concerned, as, for example, in emphysema and acute lung diseases where there is both pulmonary stasis and insufficient air exchange.

Lung fibrosis and tuberculosis.

To a less degree the same conditions prevail in pulmonary fibrosis, the pressure of tumors or of pleural or pericardial effusion and in advanced pulmonary tuberculosis. In the last instance the amount of dyspnea present when the patient is at rest is often strikingly disproportionate to the area of lung involved though usually evident upon exertion. In severe anemias the impaired hemoglobin content alone or a combination of circulatory and hemic insufficiency is accountable, and, in uremia and diabetes, toxemia is the prominent feature.

Anemia, uremia, and diabetes.

Snoring respiration.

Stertorous Respiration.—In cases of profound coma, extensive post-nasal adenoids or chronically enlarged tonsils, as well as in ordinary sleep in certain individuals, there is a snoring respiration which is not, however, a true dyspnea.

Diabetes.

Air Hunger.—This peculiar form of dyspnea is best illustrated in diabetic coma and best described by the name given. (See Diabetic Coma.)

Of great importance.

Dyspnea on Exertion.—This symptom, vitally important whether subjective or objective, accompanies sèvere chronic bronchitis, emphysema, early tuberculosis, pleural effusion of the latent type, anemias, uremia, exhausting diseases or debility, obesity and *imperfectly compensated cardiac lesions.*

Significance.

Persistent Dyspnea.—This indicates severer grades of the conditions mentioned in the preceding paragraph, stenosis of the air passages or certain profound toxemias. It is particularly marked in obstructive lesions, terminal cardiac incompensation and advanced emphysema.

Inability to Hold the Breath.—Inability to hold the breath may be a part of any severe dyspnea, and is not infrequently the only evidence of that condition in certain minor cases of cardiac incompensation.

Inspiratory vs. expiratory dyspnea.

Paroxysmal Dyspnea.—*The commonest pulmonary forms are croup, laryngismus stridulus, and true asthma; the two first mentioned being of the type of inspiratory dyspnea, the other predominatingly expiratory.*

Spasm of the glottis is a component of the spasmophilic diathesis but in various forms and degree may be associated with hysteria as well as with organic nervous disease.

True Bronchial Asthma.—This may be perfectly simulated by the dyspneic paroxysms sometimes observed in uremia, by similar seizures due to mediastinal pressure, and by that paroxysmal-dyspnea alternative of angina pectoris for which alone the term "*cardiac asthma*" should be reserved.

Misleading paroxysmal dyspneas.

The ordinary form of dyspnea associated with cardiac disease is non-paroxysmal, and extreme only under exertion, in cases of massive pulmonary embolus, or as the result of the pressure of secondary transudates.

In terminal decompensation, certain cases of aortitis or an associated pulmonary edema, very severe types of dyspnea may occur, often paroxysmal.

Pedunculated tumors below the glottic chink may cause violent and even fatal paroxysmal dyspnea. *Cases of spasmodic paroxysmal dyspnea due to aneurysm of the aortic arch produce most misleading pseudoasthmatic seizures which repeatedly have deceived the elect.*

Aortic aneurysm.

Associated Changes in the Chest Outline.—Certain permanent changes, such as the *forced inspiration type* of emphysema, the *unilateral enlargement* of chronic pleural effusion or tumor and the *unilateral retraction* of fibroid phthisis and pleural adhesion, need no further description here. In asthma, the lower diameters of the thorax are increased during the paroxysm because of the expiratory type of the spasmodic dyspnea. In broncho-pneumonia and other forms of obstructive dyspnea a marked inspiratory narrowing and recession of the interspaces occurs because of the inspiratory negative pressure and the direct drag of the diaphragm. This need not be confused with the slighter contraction sometimes observed in chronic emphysema, visceroptosis and even in normal individuals.

Broncho-pneumonia.

Orthopnea.—*This term refers especially to a dyspnea induced or intensified by recumbency and demanding the sitting attitude for its amelioration or relief. It is most commonly noted in cardiac incompensation, spasmodic asthma, advanced emphysema, certain of the pleural or pericardial effusions, thoracic aneurysms, mediastinal growths and abscesses, or gas or fluids causing pressure within the abdominal cavity sufficient to embarrass diaphragmatic movement and obstruct the action of the heart or lungs.*

The obligatory sitting posture.

In some instances, as stated previously, the patient must not only sit up, but at times stand erect. In others he is comfortable only when leaning forward.*

Extreme instances.

In some instances a subjective sense of suffocation may compel *the abrupt assumption* of a sitting or even erect posture. As stated previously, death may follow the natural attempt of the attendant to forcibly restrain the patient and outweigh the risk involved in the sudden exertion.

Scylla and Charybdis?

The physician must decide whether the action is obligatory and represents a genuine uncontrollable necessity or is merely an expression of extreme nervousness or intractability.

VARIATIONS IN RESPIRATORY RHYTHM.—Mere irregularity is common in children whether awake or asleep. It also appears as an *ominous sign* in massive cerebral apoplexies, brain tumor, meningitis, shock and collapse, and not infrequently as a symptom of chorea.

Trivial or ominous.

Jerky Respiration.—This may be inspiratory as in hydrophobia or hysteria, or expiratory and often grunting in intercostal neuralgia, acute pleurisy, rib fractures, stab wounds, renal and gall-stone colic, or in the presence of any paroxysmal pain, especially of the abdomen.

Inspiratory or expiratory.

Wavy Inspiration.—This is characterized by an undulatory movement of the chest and may occur in severe typhoid, pneumonia, and other conditions associated with great prostration.

Respiratory undulation.

Biot's Respiration.—This term applies to respiration interrupted regularly or irregularly by apneic intervals of varying length. Seen most frequently

* It has been stated that this latter position is pathognomonic of acute aortitis, but in the author's personal experience it is a common symptom in mediastinal tumor.

in meningitis; it may be encountered in pneumonia in a moderate form, and is ordinarily a precursor of death.

Portentous if marked.

Cheyne-Stokes Breathing.—*This term covers irregular breathing interrupted by apneic intervals but characterized by the fact that, following a pause, the respiration recommences as shallow slow breathing, steadily increasing in depth and frequency until a maximum is reached, when it subsides in the reverse order.*

A precursor of death.

It may accompany the coma of organic disease of the brain, apoplexy, tumor, uremia, opium poisoning, severe or terminal myocardial decompensation and meningitis. It may also be observed in diabetes and various

Wide range of incidence.

acute infections, such as cerebro-spinal fever, septicemia, typhoid, pneumonia and the exanthemata. In feeble children, and in the aged, it may occur during sleep and, in rare instances, in the adult suffering from the chronic

May exist for years.

diseases mentioned above it may be present both waking and sleeping for long periods. In an ambulatory case reported by Osler it was so severe and troublesome as to interfere with eating.

Exhaustion of center.

Traube's theory of lowered irritability of the respiratory center, if combined with the fatigue hypothesis of Rosenbach, would adequately cover the phenomenon. This would assume a primary, progressive, preliminary exhaustion of the respiratory center arising from the chronically insufficient blood supply, a resultant temporarily diminished excitability of the center,

Overaction.

and a recurrent and more or less rhythmic overreaction which is due to the cumulative increase of the asphyxial stimulus during the period of apnea, and continues through the first imperfect series of renewed respirations. The

Recurring exhaustion.

deeper breathing of the fastigium temporarily adjusts the balance but so exhausts the center that no response occurs until the period of apnea has again so vitiated the blood as to produce again an excessive stimulus.

Increased Frequency.—Mere fever increases frequency as do most dyspneas not stenotic in type, and it often accompanies pulmonary, cardiac, and renal lesions, and neuroses.

Slow breathing is commonly seen in coma, collapse, and all conditions associated with stertor.

Hiccough.—*This sudden spasmodic contraction of the diaphragm, often temporary and negligible, may become one of the most serious complications of acute or chronic disease.*

Prolonged seizures.

The attacks, ordinarily short, may be prolonged for days or weeks and rapidly exhaust the patient.

Causes.—*Hiccough may be due to (a) organic or functional disease of the nervous system; (b) of the abdominal organs; (c) constitutional diseases, (d) miscellaneous causes, such as dyspepsia, flatulence, severe acute infections, the typhoid state, alcoholism, pneumonia, pulmonary gangrene, chronic heart disease, pregnancy, etc.*

It would be useless to give in detail the various conditions with which it

An agonizing complication.

is associated as *it may occur in any exhausting disease, acute or chronic.* Occurring with *inflammation of the diaphragm*, whether from the pulmonary or peritoneal side, it constitutes an agonizing complication.

SHOCK AND COLLAPSE.—Shock.—Patients suffering from the severer grades of shock show pallor, a lowered temperature, a cold wet body surface, a weak thready pulse, slow, shallow and often sighing respiration, great physical weakness and prostration. The Hippocratic countenance or at least an expression of great anxiety is present. All of these grave symptoms are combined in many cases with extraordinary mental calm and clearness and oftentimes with a great amelioration of, or entire freedom from, any preëxisting pain.

Striking facies.

The ominous subsidence of any antecedent or initial pain and mental distress is often misleading.

Among other causes of shock are acute hemorrhagic or suppurative pancreatitis, strangulated hernia, severe accidents, particularly those attended by crushing violence, and, in its lesser degrees, certain of the severe acute infections, renal and hepatic colic, angina pectoris, and other conditions associated with *extreme pain.**

Collapse.—This is commonly associated in medicine with those intra-abdominal diseases which have a surgical side, for example, perforation of the appendix, of a gastric or duodenal ulcer or hepatic abscess, but the term applies to any case in which extreme depression of the vital forces and circulatory failure arise.

Surgical associations.

Concealed Hemorrhage.—An extensive open hemorrhage almost invariably alarms and excites its victim but serious internal and concealed hemorrhage presents symptoms of shock and collapse together with a certain curious restlessness and oftentimes yawning, nausea and air hunger.

An important syndrome.

Sources of Concealed Hemorrhage.—Among the most common are hemothorax, aneurysmal rupture, duodenal ulcer, typhoid, gastric ulcer, ectopic gestation or pelvic hematocele from any cause, traumatism, the hemorrhagic diathesis, and very rarely in pulmonary tuberculosis a large hemorrhage into an old cavity which may for a time be concealed.

In any obscure case of shock or collapse the stools should be carefully examined for blood and in married women the possibility of ectopic gestation cannot be ignored. The clear mind so often encountered in the earlier stages may later yield to a low delirium and, occasionally, convulsions.

* The student should read the recent papers of Dr. Crile as expressing the most modern theory of shock and its prevention. He shows the effect not alone of trauma but of psychic and toxic factors upon the cerebral and spinal nerve cells.

DISEASES DEPENDENT UPON OR ASSOCIATED WITH CHANGES IN THE BLOOD OR BLOOD-MAKING ORGANS AND DUCTLESS GLANDS

EXAMINATION OF THE BLOOD.—*This department of medicine, valuable alike to the internist and to the surgeon, affords a means of exact diagnosis in syphilis, malaria, the leukemias, filariasis, relapsing fever, typhoid fever, Malta fever and trypanosomiasis: fundamental evidence in cases of pernicious anemia, chlorosis, secondary anemia, Hodgkin's disease and diabetes mellitus, and valuable corroborative evidence in the acute pneumonias, appendicitis, septic infections, trichiniasis, lead poisoning, gout, malingering, certain cases of malignant disease and many other ailments.*

Scope and value.

In addition one must consider the valuable information represented by negative blood findings.

The blood is the circulating medium for the exchange of cell food, chemical substances acting as stimuli, and excretory and secretory products of the vital activities.

The products of the glands of internal secretion, actual nutritive substances and simple or complex chemical substances initiating heart action are examples.

It removes alike the strictly excretory substances and the toxins of disease. Blood bacteriology, serology and parasitology and the refinements of blood chemistry and physics do not fall properly under discussion.

This section has to do almost exclusively with blood morphology as modified by disease.

Necessity of thorough training.

Thorough laboratory training and accurate, painstaking work are essential to success and it is unfortunately true that a large proportion of the findings obtained under other conditions are quite worthless and often most damaging.

Scope of Section.—It is obviously impossible to deal seriously with serology or the refinements of blood pathology in a book of this kind and the reader is referred to the many excellent special works dealing with these interesting and complex subjects.

Only such diseases and diagnostic procedures as are of direct clinical importance and ready *availability* are dealt with in what the author hopes may prove a practical and adequate way.

Clinical Essentials.—(*a*) *The determination of hemoglobin.* (*b*) *The red cell count.* (*c*) *The white cell count.* (*d*) *The examination of fresh blood.* (*e*) *The examination of the smear preparation* (1) *stained,* (2) *unstained.* (*f*) *The study of the various "clump" or agglutination reactions in certain acute infections* (serum diagnosis). (*g*) *The study of exudates.* (*h*) *Complement-fixation tests.*

Clinical Tests of Less Importance.—(*a*) *Counting blood plates.* (*b*) *Estimation of alkalinity.* (*c*) *Cryoscopy.* (*d*) *Specific gravity determination.* (*e*) *Coagulation period.* (*f*) *Determination of blood volume and ratio of corpuscles to plasma.* (*g*) *Viscosity tests.*

Terms in Common Use.—**Anemia,** a deficiency in corpuscles, coloring matter, or total blood volume. **Oligocythemia,** a deficiency of red cells. **Oligochromemia,** a deficiency in hemoglobin. **Oligemia,** deficient total blood volume.

The total volume of the blood is about one-twentieth ($\frac{1}{20}$th) of the body weight* and in severe anemias may be reduced 50 per cent.

This may occur temporarily in severe hemorrhage or, more persistently as the result of repeated severe hemorrhages and also in the hemolytic and cachetic or inanition anemias of the more serious grades.

Hydremia.—This state represents a disproportionate amount of water in the blood either from loss of solids or actual water increase, aside from accidental temporary variations due to sweating, excessive ingestion of liquids and the like.

The color of normal arterial blood is due to the oxyhemoglobin contained in its erythrocytes.

Normally, the hemoglobin content of such blood lies between 13 and 14 grams per 100 c.c. with which is combined loosely about 21.6 per cent. by volume of oxygen, and 13 per cent., by volume, of CO_2.

Double that amount of CO_2 is contained in the plasma of such arterial blood and the total CO_2 content of *venous* blood is by volume about 48 per cent.

Practically all forms of non-obstructive dyspnea clinically encountered are due to deficient oxygen content however induced.

Leucocytosis.—The term is commonly used to denote an abnormal increase in the number of leucocytes, the polymorphonuclear type predominating. The average per cubic millimeter is normally about 7500. Normal count.

Lymphocytosis.—An unusual increase in the number of *mononuclear non-granular* leucocytes.

Leucopenia.—The opposite of leucocytosis and equivalent to *hypoleucocytosis*, an abnormally or unusually low leucocyte count.

Plethora.—A term covering what was formerly supposed to be a pathological condition, but now used to indicate an increase in the total quantity of blood. The term "plethoric" is applied to those individuals with a ruddy countenance due to dilated or unusually prominent capillary network. The condition is now of slight clinical importance, though it is certain that there is a direct relation of the blood volume to the musculature and to the size of the heart.

Polycythemia, an abnormal increase in the number of red cells may, rarely, be a symptom of importance, particularly when associated with enlarged spleen and cyanosis (*erythremia*) or acetanilid addiction.

Color Index.—*The color index represents the result obtained by dividing the hemoglobin percentage by the percentage of red cells.*

The normal figure for hemoglobin is 100; the corresponding erythrocyte standard being 5,000,000 cells in men and 4,500,000 in women.

* By Haldane's carbon monoxide method. By Keith's method it is found to vary between one eleventh and one thirteenth of the body weight.

Example 1.—In a given case the hemoglobin is 30 per cent., the red cells number 3,000,000, or 60 per cent. 30 divided by 60 equals $\frac{5}{10}$; a low color index, indicating chlorosis.

Example 2.—Hemoglobin 30 per cent., red cells, 1,000,000 or 20 per cent. 30 divided by 20 equals 1.5; a high color index such as is found in pernicious anemia of the hemolytic type.

Example 3.—Hemoglobin 50 per cent., red cells 2,500,000 or 50 per cent. 50 divided by 50 equals 1; a color index suggesting secondary anemia.

To Obtain Blood for Examination.—It is essential that the actual technic should be thoroughly understood, and intelligently and expeditiously carried out.

Sterilization.

Precautions.—One should not trust to the flow of blood for sterilization either of the skin or the instrument employed.

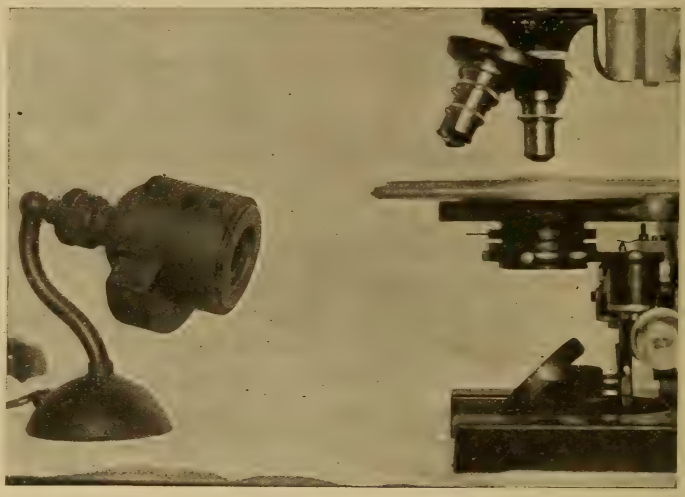


FIG. 26.—Matthews' microscope lamp with iris-diaphragm. (*Todd.*)

The ear lobe or the finger-tip should be carefully cleansed with alcohol, bay rum, or some similar substance always available, and *the needle or lancet used should be sterilized in the flame.*

Bleeders.

The patient may be asked whether he "bleeds easily" or the reverse with a view of determining the size, depth of the puncture, and avoidance of serious complications in cases of hemophilia. Two cases of this kind in the author's clinic bled steadily for several hours from a very minute puncture.*

Best instrument.

For making the puncture an instrument with a sharp cutting edge should be used. The Hagadorn needle or the *spring lancet with a trocar point* is most suitable, the ordinary needles being poor substitutes. Some workers prefer to use a suitable piece of broken cover-glass which is firmly grasped between the finger and thumb at the point required to permit necessary penetration and at the same time limit the depth of the puncture. I

*As a matter of fact the risk of any serious or *fatal* hemorrhage is *practically* negligible.

quickly and smartly done the stroke is almost painless and the resulting blood-flow free.

The puncture itself should be sufficiently free and deep to obviate *squeezing or excessive friction*, and, furthermore, the instrument should permit a quick, painless stroke,* and the first two or three drops should be discarded. *The blood should flow spontaneously and, to maintain it, light brisk friction serves admirably, but pressure should never be employed.* To check it, firm, steady pressure is sufficient, or in the case of the finger, vertical elevation of the arm. Method.

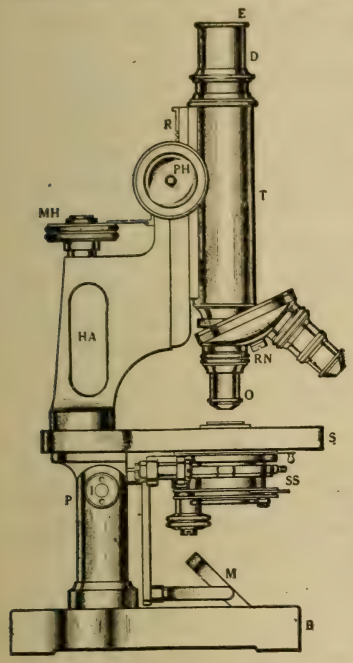


FIG. 27.—Handle-arm microscope: E, Eye-piece; D, draw-tube; T, body-tube; RN, revolving nose-piece; O, objective; PH, pinion head; MH, micrometer head; HA, handle-arm; SS, substage; S, stage; M, mirror; B, base; R, rack; P, pillar; I, Inclination joint. (*Todd.*)

An edematous area or one in which local cyanosis is marked should be avoided in obtaining specimens for examination. The former yields a diluted blood, the latter a misleadingly concentrated specimen.

Care and Preparation of Slides and Cover-glasses.—All material should be of the best sort, ⅞ inch square cover-glasses being superior to the round, and both these and the slides should be *thin*. When received from the dealer, they should be thoroughly washed in soap and water and then placed in the following solution: Hydrochloric acid 1 part, absolute alcohol 29 parts, water 70 parts, or they may be placed in solution containing equal parts of alcohol and ether. When required, they should be wiped thoroughly, polished with tissue paper or with soft linen, and, if convenient, passed through a flame. A small drop of blood will spread satisfactorily and evenly between a perfectly clean slide and its cover-glass or between two cover-slips, especially if these are slightly warm. Form and size. Preparation.

Making the Smear.—To make a good smear preparation the cover-glass must be absolutely clean and a *small drop* only, should be lightly touched by one, the other placed quickly upon it, an instant allowed for the spread of the blood, and the two separated by a sliding (not a lifting) movement, as shown. The cover-glass should never touch the skin. This is the best method for general use, for neither the "scrape" method nor the cigarette-paper procedure is well adapted to all specimens of blood, least of all to smears taken in pernicious anemia. Even spreads indispensable. Correct technic.

FIG. 28.—Blood smear. Ordinary method.

Another method consists in using the edge of one cover-glass and *drawing* it lightly across the drop or gently *pushing* the edge of the slide over the

* Blood may be taken from young children in this way without arousing them from sleep.

surface, yet another, of a similar procedure with microscope slides and in the use of the cigarette-paper edge as shown.

Yet another, involves the use of an ordinary sewing needle, which is carried lightly but quickly across the slide, on the flat.

Good smears.

A good smear should show the cells evenly distributed throughout large areas and should not look to the eye smeary or thick. If carefully preserved the dry unstained specimen will keep for months.

FIG. 29.—Making blood smear with two cover-slips. The direction of the stroke may be reversed if one prefers the "gentle push" to the "light draw," but the former method is more difficult.

Forceps are not needed at any stage of the procedure, but the stained preparations are more brilliant if the slip is immediately, though slowly and carefully, dried above a flame.

Nothing but persistent and painstaking practice will enable one to make good smears but the result is well worth the time and labor expended.

Fixation of Dried Preparation.—The use of the *self-fixing* Wright stain makes a prolonged description of the older methods unnecessary.

Rapid fixation and staining.

Smears may be "fixed" for rapid work with the old triple stain by any one of four simple methods: (1) *Passing the cover-slip through a small flame 3 or 4 times.* (2) *Boiling one minute in absolute alcohol.* (3) *Immersion for one minute in a 1 per cent. alcoholic solution of formalin.* (4) *Exposing smear for a few seconds to formalin vapor.* The older "heating" methods are delicate, require for good results a special oven or heating stage, and are not necessary for ordinary clinical work.

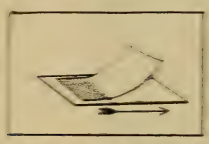


FIG. 30.—Cigarette-paper method of preparing blood films.

Examination of the Fresh Blood.—Fresh blood is readily examined by allowing a small drop to spread evenly under a perfectly clean, moderately warm, cover-slip and slide. This procedure is valuable in the observation of: (*a*) *Leukemia.* (*b*) *Malarial organisms* (and their ameboid movements if the stage is kept warm).* (*c*) *Filaria.* (*d*) *Relapsing fever.* (*e*) *Trypanosomiasis.* (*f*) *Rouleaux formation.* (*g*) *Increase of fibrin.* (*h*) *Increase of blood plaques.* (*i*) *Low color index.* (*j*) *Approximate number of red and white cells.* (*k*) *Cell deformity.* (*l*) *Wide departures from normal coagulation time.*

Value.

Limitations.

Nucleated red cells are not thus revealed and the rough count of leucocytes made possible is not dependable. If the cover-slip edges are sealed with a little vaseline one may preserve the fresh specimen for hours.

Hemoconia.—The presence of the mysterious and clinically negligible "blood dust" or hemoconia is indicated by a rapid vibration of these tiny colorless highly refractile, unstainable bodies, which may be fat particles, nuclear or cell fragments or extruded granules.

Fibrin.—Increase or decrease of fibrin and rouleaux formation may be determined roughly after the specimen has stood for ten or fifteen minutes.

* Brownian movement and the contractions preceding crenation and cell death can hardly be confounded with true ameboid movement.

No smear is made, but the cover-glass lightly touches the blood drop and is then placed upon the slide. The stage should be dimly illuminated to show the fibrin threads which frequently radiate from groups of blood plates.

Fibrin is increased in pneumonia, acute rheumatism and various septic infections, and diminished in leukemia and pernicious anemia, typhoid fever, malaria, measles, variola, Graves' disease and various conditions associated with extreme inanition. Hyperinosis.

In general the fibrin increase (hyperinosis) occurs in diseases in which leucocytosis is present though in leukemia itself a diminution (hypinosis) occurs. Hypinosis.

Examination of the Stained Specimen.—Modern staining methods depend upon the selective affinity shown by the different constituents of the cellular elements of the blood for certain anilin dyes, which are divided into three groups: (*a*) *basic*, (*b*) *acid*, (*c*) *neutral*. Basis of staining methods.

By using combinations one may in a single step attain differentiation. *The basic dyes*, such as methyl violet, methylene blue or hematoxylin (chromatin stains) act chiefly upon nuclei. *Acid dyes*, such as eosin, orange G. or acid fuchsin are protoplasm stains, while *neutral dyes* (resulting from the mixture of acid and basic colors in solution) color beautifully the "neutrophile" granules of the leucocytes.

Staining Solutions.—Ehrlich's triacid or triple stain now has been displaced by Wright's modification of Jenner's stain, an alkaline eosinate of methylene blue, which, *by at once fixing and staining the specimen*, eliminates the laborious and delicate heating process of the older method. Rapid and simple.

Wright's Modification of Louis Jenner's Stain.—The student or practitioner had best purchase the "soloid" tablets* or procure a solution ready made through some drug supply house, as the formula is somewhat complex.† It should be kept tightly corked to prevent precipitation.‡

* *The "soloid" tablets of Burroughs & Wellcome offer a ready method of making up fresh stain.*

One soloid is tritrated with 10 c.c. of pure methyl alcohol, the dissolved stain decanted and the undissolved stain again extracted with another 10 c.c. of alcohol. This process is repeated a third time so that 30 c.c. in all is used. The stain should then be passed through a filter.

† *Formula.*—(*a*) Make a 0.5 per cent. aqueous solution of sodium bicarbonate, place in Erlenmeyer plaque, add 1 per cent. of Grübler's medicinal methylene blue; place in steam sterilizer for an hour. (*b*) After cooling, add while stirring with glass rod, a 1–1000 aqueous solution of Grübler's water-soluble yellowish eosin until the color of the original mixture changes to purple and presents a lustrous yellowish scum upon its surface. (About one-fifth as much eosin solution as methylene blue solution will be found necessary.) (*c*) Collect this scum by filtration, dry it, and with it saturate methyl alcohol (100 c.c. of the latter will dissolve about $\frac{1}{10}$ of a gram of the dry precipitate). (*d*) Filter and add 25 per cent. of methyl alcohol. The stain is now ready for use and, *if kept tightly corked, should neither precipitate nor show impairment though kept for a long period.*

‡ The Giemsa stain is another Romanowsky modification of great value, but does not yield the best results with neutrophilic granules.

T. M. Wilson's stain, yet another Romanowsky modification is used in many laboratories, but has no decided superiority to Wright's modification of the Jenner stain.

Technic of Staining.—(1) *The dried but unfixed smear is completely covered with the stain for one minute.* (2) *Distilled water is added to the stain on the cover-slip drop by drop until a greenish metallic scum appears and the margins show a reddish tint.* (3) *After three minutes the stain is washed off with water leaving a purplish specimen, which is washed until the film is yellowish or pink* (4) *gently dried between filter paper, and* (5) *mounted in balsam.*

Action on cells

Results.—The appearance of the various cellular elements is as follows: the *erythrocytes* are orange or pink throughout. *Erythroblasts* show deep blue nuclei, blood-plaques are purple, mast-cell granules deep purple, *polymorphonuclear leucocytes* show lilac or dark blue nuclei, *neutrophile granules* lilac, *eosinophile granules* pink, fine *basophile granules* a deep blue stain, *bacteria* and such organisms as malarial parasites blue. *Myelocytes* show a purplish or dark lilac nucleus and reddish or dark lilac granules.

Ehrlich's Triacid Stain.—*The ready-mixed powder* for making this stain may be bought of any drug supply house and the student or practitioner should not attempt to make the original.

Formula: Ehrlich-Biondi powder, gr. xv. Alcohol (*absolute*), 1 c.c. Distilled water, 6 c.c.

Fairly good rapid work may be done with a good triple stain in the absence of Wright's stain by heating the smear carefully as in the staining of tubercle bacilli and staining for thirty seconds or less, washing, drying and mounting (Cabot).

Other heat fixation methods are now so little used that a description is not deemed necessary. Scott fixes by a few seconds' exposure to formalin fumes before applying Jenner's stain and shows beautiful results.

HEMOGLOBIN.—Hemoglobin or, more properly, oxyhemoglobin constitutes about nine-tenths of the bulk of the red corpuscles. It is a proteid substance containing 4 per cent. of hemochromogen, an iron-holding body, and 96 per cent. of concentrated, almost insoluble albumin *which readily forms unstable compounds with oxygen.* The great value of its determination by clinical tests may be readily appreciated.

Tests for Hemoglobin.—*An expert observer can make a rough estimate of the percentage of hemoglobin from the appearance of the ordinary stained or even the unstained smear preparation as may be readily appreciated by referring to Fig.* 36, *but for accurate work several forms of apparatus have been devised, all depending upon a comparison of a given specimen of blood, either whole or in a known degree of dilution, with a fixed color scale.*

Inaccurate.

Tallqvist's Hemoglobinometer.—*This, the simplest and least reliable,* consists of lithographed color bands. Each has a central perforation and represents the color of blood in dilutions running from 10 per cent. to normal.* A drop of the patient's blood is taken up by the absorbent paper and the resulting stain is placed under the central perforation of the color bands and comparison is made *as soon as the stain has lost its wet gloss, not after complete drying.*

* The color scale is bound with 50 sheets of special paper, each divisible into three parts, furnishing material for 150 tests.

This is a simple, rapid, but *inaccurate* method, permitting an error of at least 10 per cent., and the color scale fades, if not kept from the light. It is distinctly inferior to Dare's instrument which takes hardly a minute more of time.

Dare's Hemoglobinometer.—A circular disc of tinted glass, representing variations in blood-coloring matter of a known degree, is brought into direct contrast by transmitted candle light with a film of the fresh whole blood drawn by capillary attraction between two glass plates, one transparent, the other translucent and white. A detachable observation tube and a circular shield protect the eyes from extraneous light, and the percentage of hemoglobin may be read directly from the scale. This excellent instrument may be used in daylight if pointed at some dark object, and its readings are not materially affected by an excess of leucocytes.*

Simple and reliable.

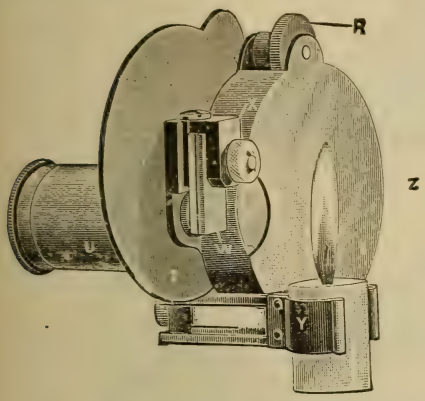


FIG. 31.—Dare's hemoglobinometer. U. Observation tube. T. Shield. W. Removable plate with capillary opening for holding blood. X. Thumb-screw holding same. Y. Candle holder and candle. S. Case holding color disc R. Milled wheel or revolving color disc.

Von Fleischl's Hemoglobinometer.—This well-known instrument or its more accurate modification has been largely superseded by the simpler and cheaper instrument of Dare. If it is used one should procure Miescher's modification. The following precautions are necessary to good results: (*a*) *The capillary tubes for taking the drop must be absolutely clean and should be tested out for equality of calibration when purchased.* (*b*) *All blood must be removed from the surface before mixing, leaving the caliber exactly filled.* (*c*) *The blood must be taken quickly, washed out into the chamber, and thoroughly mixed in the shortest possible time.* (*d*) *The observer should face the end of the movable colored wedge with the thumb-screw on his right.* (*e*) *Decision as to color should be made quickly to avoid uncertainty and confusion.* (*f*) *The thumb-screw should be sharply turned in order to obtain as vivid a contrast as possible until the final match of color is achieved.* (*g*) *Should the blood solution appear turbid and lack proper color, as in the case of leukemia, add a few drops of a dilute aqueous solution of potassium hydrate.* (*h*) *Where the hemoglobin is below 30 per cent., double or treble the usual amount of blood should be used, the percentage obtained being divided proportionately.* (*i*) *The examination should be made in a dark room, or by means of a light-proof box by candle light, and in any case some form of tube should be used for observation, a simple roll of black paper being ordinarily sufficient.*

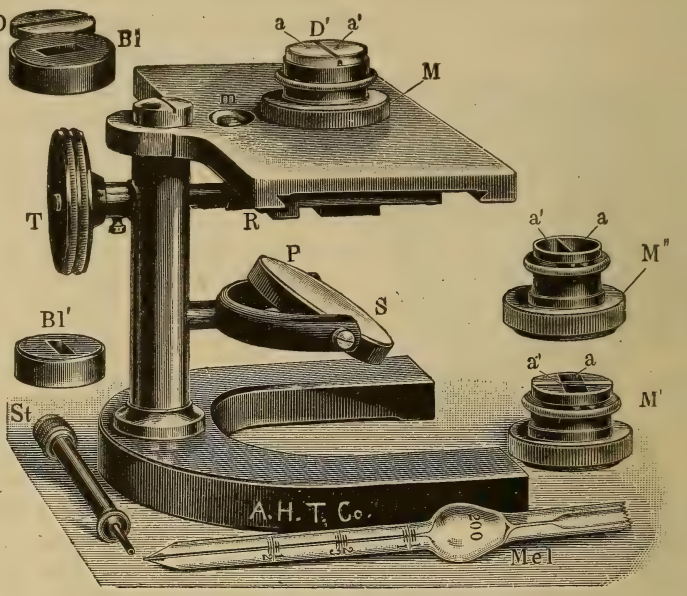
Hemoglobin Estimation by Specific Gravity.—By the use of tables to be

* This instrument is now made with a battery handle and push-button circuit breaker which adds greatly to its usefulness.

Unnecessary.

found in all the larger works dealing with this subject the hemoglobin present in a given specimen may be very accurately estimated by Hammerschlag's modification of Roy's method. The procedure is too cumbersome and fussy to be recommended to the practitioner and will not be described.

Oliver's Hemoglobinometer.—As shown by Fig. 33, this instrument depends upon the scale of colors based upon diluted blood. Its use is suffi-



Courtesy of A. H. Thomas & Co.

FIG. 32.—Von Fleischl's Hemoglobinometer (Miescher's Modification).—Description.—Milled wheel at left (T) moves a tinted glass wedge (R) under the fixed metal stage surmounted by double chamber reservoir which receives light from the calcium sulphate reflecting disc (P S) below. Half of the same chamber is filled with the diluted blood contained in the measuring capillary pipette (Mel). The other contains only plain water but receives its light from the colored wedge. By moving the wedge back and forth the colors are matched and the percentage reading is shown on a scale visible through the opening (M) just in front of the supporting-upright. The blood is obtained in exactly the same manner as for a blood-count, the diluent being calcium carbonate solution (0.1 per cent.) and the tube permitting the observer to use dilutions of 1:200, 1:300 or 1:400 according to the height to which the blood column is allowed to rise (marks $\frac{1}{1}$, $\frac{2}{3}$, $\frac{3}{4}$ respectively) before diluting and thoroughly mixing. Increased accuracy is obtained by the use of a grooved cover glass (D') which fits over the slightly raised partition dividing the two chambers, which must each be so filled as to present a convex meniscus. The necessary narrowing of the field is secured by cap-diaphragm (Bl') The average of at least ten determinations is required for accurate work.

ciently indicated by the diagram and it has no advantage over the two preceding methods.

Gower's Hemoglobinometer.—This little instrument has the merit of extreme simplicity, yet in practice one may lose more time by its use than with the other more elaborate instruments, inasmuch as a slight error in dilution means a repetition of the whole process.

An excellent instrument.

Sahli's Hemoglobinometer.—This admirable instrument resembles Gower's hemometer, but is well adapted to quick, accurate work.

The empty graduated tube is filled to the mark 10 *with a decinormal solution of HCl which is saturated with chloroform.*

The blood is then added by means of a measuring pipette and the mixture made thorough and complete. Finally, after mixing for exactly one minute, distilled water is added, guttatim, until the color corresponds to that of the control tube which contains an acid hematin solution.

The color comparison is easy and definite, but 90 should be considered an average normal and the instrument should be kept in its case or in a dark place and inverted once or twice before using.*

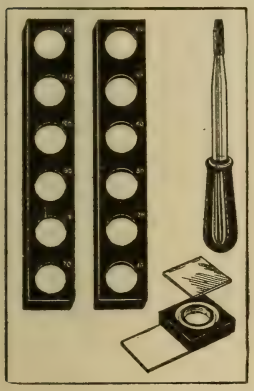


FIG. 33.—Oliver's hemoglobinometer. The discs shown as white in the illustration are colored to represent the various blood dilutions and direct comparison is made with a solution of the actual blood obtained by a measured mixing capillary pipette furnished with the instrument. Intermediate readings are obtained by placing squares of tinted glass over the fluid under examination.

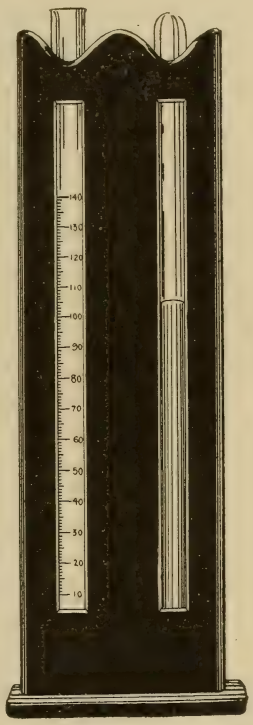


FIG. 34.—Sahli's hemoglobinometer.

The author believes that the hemometers of Dare and of Sahli best combine simplicity with adequate clinical accuracy.

Universal Micro-colorimeter.—Dr. Theodore Kuttner of New York has devised a pocket-size colorimeter which apparently is well adapted to several procedures chief of which are (*a*) the hemoglobin test, (*b*) the estimation of glucose in the blood by a modification of Benedict's method, and (c) the phenolsulphonephthalein test.†

* The instrument registers a *maximal* normal represented by the blood of healthy young Swiss males. Ninety per cent. would represent fairly an average normal.

† *Journal A. M. A.*, July 17, 1915, pages 245 and 246. *Journal A M. A.*, April 29, 1916, 1370–1373.

An abstract of Dr. Kuttner's description is appended covering its application to these three procedures.

Description of the Instrument.—The same calibrated and color tube employed in the Sahli-Gower hemoglobinometer has been adopted for this instrument.

The Sahli allows both the calibrated and color standard tubes to be viewed at full length; the new instrument, however, shows only a small part of the tubes when making color comparisons. This facilitates the color reading and is less fatiguing to the eye. An additional feature is a prism (*A*), to be described, which visually causes the contents of the tubes to approach each other more closely, thereby accomplishing greater accuracy in the comparison of colors.

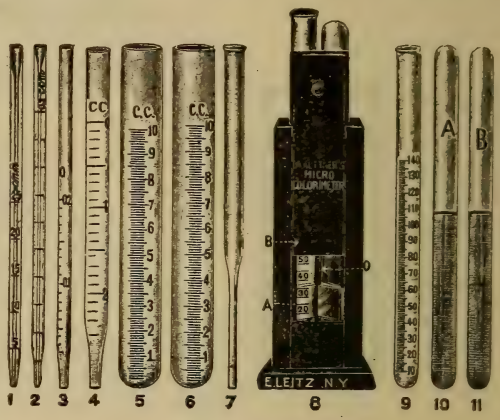


Fig. 35.

The instrument consisting of a closed upright box (dimensions 2.5 cm. by 8.5 cm.) is smaller than the Sahli. At the top there are two openings: one for the color tube, the other for the calibrated tube. Near the lower front part is a window provided with the above-mentioned prism (A—Helmholtz double plates), which serves the purpose of having the colors of both tubes appear close together, forming one continuous color band. The prism is easily removed for cleansing and can be quickly readjusted. A sliding door (*B*), which can be raised and lowered, protects the prism from dust and injury. The color standard and calibrated tube are separated from each other by a partition, which prevents light from being reflected from one tube to the other.

Directions for the Estimation of Hemoglobin.—(*Standard color tube "Hematein" = 15 gm. Hgb = 20 c.c. O_2.*)—Into the calibrated tube No. 9 enough of a $\frac{1}{10}$ normal solution of HCl is poured to reach the mark 10. With the capillary pipette No. 1 blood is drawn to the 20 cmm. mark, the sides of the pipette wiped with absorbent cotton and the blood expelled while dipping the pipette to the bottom of the calibrated tube. Water is sucked up 2 or 3 times and these washings added to the tube. This is set aside for 1 to 2 minutes and then compared with the standard color tube in the instrument. Water is added drop by drop, the solution well mixed after each addition, until the color matches that of the standard. When this is accomplished, the percentage can be read off directly by noting the figure which the meniscus has reached on the scale. As percentages occurring much below 50 are liable to error, it is best to use more blood when such anemic cases are encountered. Thus, in order to obtain greater accuracy, 2, 3 or 5 times the amount of blood should be taken, with capillary pipette No. 2 also twice the

amount of $\frac{1}{10}$ normal HCl and the same process should be followed, but the result must be divided by 2, 3 or 5.

Directions for the Estimation of Sugar in the Blood.—(*Standard Color Tubes A and B*).—According to Dr. Epstein's modification of Benedict's method, the procedure is as follows:

0.2 c.c. of blood is drawn with pipette No. 3 and discharged into a graduated test-tube containing 1 drop of 2 per cent. fluoride of sodium solution or calcium oxalate, the pipette rinsed 2 or 3 times with water, the rinsings added to the test-tube, then water to the 1 c.c. mark. Saturated solution of picric acid is added up to the 2.5 c.c. mark, the mixture well shaken and now either filtered or centrifuged. Of the clear, supernatant liquid 1 c.c. is measured with a pipette and boiled down to 2 or 3 drops in a boiling test-tube, 0.5 c.c. of a 10 per cent. sodium carbonate solution added and again boiled to crystallization, which occurs when the contents are concentrated to about 2 to 3 drops and becomes brownish-red in color, according to the amount of sugar present, when the process is completed. A few drops of water are added, the tube warmed and contents transferred to the calibrated tube of the micro-colorimeter. The tube is rinsed with sufficient water, a few drops at a time, the total volume reaching the mark 50 and compared in color with the standards. If it is darker than *A*, but lighter than *B*, then the former is used as standard, and compared in the instrument by adding water drop by drop to the fluid and mixing. When the colors have exactly matched, the height to which the fluid has risen is read off on the scale, this figure divided by 1000 representing the percentage of sugar in the blood. If the darker tube *B* is used, the result must be multipled by 2. Using this tube with the meniscus having reached mark 85 on the scale, then $85 \times 2 = 170$.

$$170 \div 1000 = 0.17 \text{ per cent.}$$

As it often happens that diabetic blood may contain 0.3 per cent. or more of sugar, it would be necessary to use less blood. It is better to take two specimens, one as already stated and another with pipette No. 2, which measures only 0.1 c.c. of blood, then proceeding in the same manner as already described, but multiplying all obtained figures again by 2. Computation would then be as follows: If the color matched the darker tube *B* at 85, but using only 0.1 c.c. of blood then

$$85 \times 2 \times 2 = 340$$
$$340 \div 1000 = 0.34 \text{ per cent.}$$

If the color matched the darker tube *B* at 140, the computation is thus:

$$140 \times 2 \times 2 = 560$$
$$560 \div 1000 = 0.56 \text{ per cent.}$$

Normal blood contains from 0.06 per cent to 0.12 per cent. sugar. It is best to take the blood before breakfast.

Directions for the Phenol-sulphon-phthalein Test.—(*Color Standard Tubes No. 1, No. 2 and No. 3*).—The patient's bladder is emptied (if necessary

with catheter), about 250 c.c. of water given to drink and ½ hour later 1 c.c. of phenol-sulphon-phthalein solution containing 6 mgm. of substance is injected intramuscularly in the region of the buttock. Each ampule contains somewhat more than 1 c.c. of the sterile solution.

The urine is collected in small beakers containing a few drops of Sodium Hydroxide solution every 2 or 3 minutes. When the first pink is seen as the urine comes in contact with the alkali, the time is noted. The patient is then required to urinate again in 1 hour and in 2 hours in two separate containers. Each quantity is then measured, 10 c.c. of 10 per cent. of sodium hydroxide solution added and each made up to 200 c.c. with water.

Of this red colored fluid 2 c.c. is withdrawn, using the 2 c.c. pipette and made up to 10 c.c. with water in graduated test-tube (No. 5 or 6). Enough of this solution is placed in the calibrated tube of the Micro-colorimeter by means of the transferring pipette to the 50 mark on the scale. It is now compared to the color standards 1 and 2. If it is darker than 1, it is diluted drop by drop with water until the color matches, the percentage being read off the same as in the Sahli-Hemoglobinometer. If tube 2 is used, the reading must be divided by 2.

Should the color be too light, after it had been diluted to 10 c.c., then only a dilution to 5 c.c. may be used and compared the same way, but the final figure obtained is to be divided by 4. If the urine contains very small amounts of the excreted dye, then the urine should be used without further dilution directly in the calibrated tube up to the 50 mark, but it must be compared to special color tube No. 3, which has a yellowish tint, and the reading divided by 10.

Between 40 and 60 per cent. (average 50 per cent.) of the dye is eliminated during the first hour and from 60–85 per cent. totally after 2 hours and only traces eliminated during the following hours.*

Care and Use of the Pipettes.—Four pipettes are furnished with the instrument: No. 1., up to 25 cmm.; No. 2. $\frac{1}{10}$ c.c. divided into ten divisions; No. 3. $\frac{2}{10}$ c.c. divided into ten divisions; No. 4. 2 c.c. divided into ten divisions.

Pipettes must be clean and dry before use.

When the required amount has been drawn into the pipette the tip of the tongue should be placed against the mouthpiece attached to the rubber tubing of the pipette. Before expelling its contents, the pipettes are held in a horizontal position and wiped with absorbent cotton. After use, they should be immediately cleaned, first with water, then alcohol and then ether.

ERYTHROCYTES AND LEUCOCYTES

Chief normal forms.

Classification of Leucocytes.—*The simplest modern classification deals with six normal varieties*: (1) **The polymorphonuclear neutrophiles** *which constitute from* 60 *to* 75 *per cent.* (*average* 70 *per cent.*). (2) **The small lymphocytes,** 20 *to* 25 *per cent.* (*average* 20 *per cent.*). (3) **The large mononuclear**

* The test is described more fully in its proper section.

PLATE I.

Chief varieties of cells encountered in health and disease (Wright's stain). 1. Normal red cell. 2. Common form of polymorphonuclear leucocyte. 3. Lymphocyte. 4. Eosinophilic myelocyte. 5. Eosinophilic leucocyte. 6–6. Neutrophilic leucocytes: upper left, transitional form, on right neutrophilic myelocyte. 7–7. Large mononuclears. 8. Normoblast. 8. Normoblast showing division of nucleus. 9. Normoblast nucleus. 10–11. Basophilic leucocytes. 12. Megaloblast.

(*averages* 5 *per cent.*). (4) **The transitional forms,** 2 *to* 4 *per cent.* (5) **Eosinophiles** 0.5 *to* 5 *per cent.* (*average* 4 *per cent.*). (6) **Basophiles,** "*mast*" cells 0.1 *to* 0.5 *per cent.*

Sources of White Cells.—It was believed formerly that all granular leucocytes originated in the bone marrow and that the lymphocytes chiefly came from the lymphadenoid tissues, but the latest researches indicate that the red bone marrow is the *chief* source of all varieties.

Lobulate nucleus.

1. The Polymorphonuclear Neutrophile.—This cell measures 7.5 to 12μ, averages about 11μ, and is characterized by its irregular lobulated nucleus, staining dark blue and connected by chromatin strands, and the presence of neutrophilic granules in the protoplasm which take a lilac or pink color with Wright's stain.*

Single round nucleus.

2. The Small Lymphocyte.—This measures 5 to 10μ and contains a single round deep-staining nucleus almost filling the cell and often obscuring the border of protoplasm, the latter being almost wholly unaffected by the triple stain, but colored sky-blue if the Romanowsky method be employed.

Relative excess of protoplasm.

3. The Large Lymphocyte.—This measures 10 to 15μ and carries a round or oval nucleus staining pale blue, and a relatively large amount of protoplasm. It differs from

4. The transitional leucocyte, only in lacking the indented crescentic or reniform nucleus of the latter.

Coarsely granular "polymorphs."

5. Eosinophiles (8 to 11μ).—These are readily recognized by their coarse, acid-staining granules and polymorphonuclear type. The granules are especially brilliant in certain parasitic infections and septic conditions (Gulland).

Basic "polymorphs."

6. Basophiles (Mast Cells) (7.5 to 12μ).—The distinguishing feature of the basophile cell is the presence of its fine, basic-staining granules. The nucleus is polymorphous and stains a pale green with the eosin and methylene-blue mixtures. The characteristic granules are best shown by Wright's stain or that of Leishman and not at all by the triple stain.

Immature form.

UNUSUAL OR ABNORMAL FORMS.—The myelocytes (*marrow cells*) (15–20μ) are large immature forms of the granular leucocytes and may contain neutrophilic, eosinophilic or basophilic granules, thus constituting three sub-varieties. They are the dominant cells of the normal bone marrow.

The nucleus is spherical or oval, tends to be eccentric, poor in chromatin, staining feebly or moderately with the blue, occasionally shows division and often a marginal light circle.

The presence of the granules distinguishes the myelocyte from the myeloblast and large mononuclear forms and the lack of lobulation or twisting of the nucleus from the polymorphonuclear cell. Occasionally it is slightly indented.

With Wright's stain the neutrophilic myelocytes show lilac or pink granules with a purplish admixture. Eosinophilic myelocytes show the ordinary coarse, brilliant, eosinophilic granules, occasionally showing some basophile admixture.

* Ehrlich's triacid stain is also very effective. In general, a mixture of an acid and basic dye is requisite.

Normally lacking in circulation.

Significance.—Found in the circulating blood the cell is invariably pathologic, occurring in many cases of profound anemia or excessive leucocytosis, occasionally in lymphatic leukemia and Hodgkin's disease, but only in small numbers, whereas, in myelogenous leukemia they are the most characteristic and predominant of the white cells.

The Meta-myelocyte.—This represents a transitional stage between the myelocyte and the transitional form of the polymorphonuclear leucocyte.

The cell is relatively small, the nucleus is more strongly basophile and gives evidence of beginning segmentation. It is present only in association with the myelocyte.

The Myeloblast.—This scantly-or non-granular form, dominant in embryonic myeloid tissue, probably the mother cell of the *granular* (marrow) myelocyte, differs from the large lymphocyte morphologically, chiefly in the following particulars (Naegeli).

(*a*) *Constant* absence of the clear perinuclear zone.

(*b*) Decided differences in nuclear form and structure.

(*c*) Greater number of nucleoli.

(*d*) In strongly heated specimens stained with methylene blue the lymphocyte nucleus is pale and shows 1 or 2 nucleoli very distinctly; the myeloblast nucleus is stained intensively and does not show its nucleoli (2 to 6).

The greater chromatin content of the myeloblast nucleus is also evident if the triacid stain is used. Small forms are occasionally encountered.

Naegeli strongly recommends the use of the Schridde-Altmann staining process for differentiation.

This is a tedious and prolonged procedure, the technic of which is to be found in Naegeli's book or some of the recent works devoted wholly to this branch.

These cells are rarely present save in the crises of myeloid leukemias and perhaps pernicious anemia and the infantile forms.

Pro-myelocytes.—These represent a transition form between the myeloblast and the myelocyte.

The nucleus is large and poor in chromatin, nucleoli are present (none visible in fully developed myelocyte) and the protoplasm is strongly basophilic and contains a few, small, scattered granules.

Türck's Irritation Forms.—These are large cells, probably identical with the "pathologic" myeloblast, containing absolutely no granules in their protoplasm, usually showing vacuoles, especially likely to appear during an acute infection and of little or no clinical importance or significance.

Giant Marrow-cells.—(*Megakaryocytes*).—These progenitors of the blood platelets are huge cells with large convoluted nuclei.

Their occurrence in the blood is rare, accidental and extremely brief, as they cannot pass the capillaries.

Lymphoblasts.—A replica of the small mononuclear cell in its morphology, but the equal of the large mononuclear in size, is found

occasionally in the blood of normal children and in the adult suffering from disease.*

It is important only because confusing, when the possibility of its presence is not known.

The Schridde-Altmann Granules are merely acidophil granules within the leucocytes brought out by the use of the special stains of Schridde or Freifeld and have no special clinical value or significance.

"*Azure Granules*" within the lymphoblast protoplasm are said to be inconstant in ordinary leukemia of the lymphatic type but constant in the acute form.

They are developed best by the Giemsa or Wilson stain and so shown occur only in the *small lymphocyte* as *red* granules.

Giant Lymphocytes.—(*Pathologic Lymphoblasts*).—These abnormally large cells carry a round nucleus, poor in chromatin, staining but slightly with methylene blue or the triacid stain, somewhat better with eosin-hematoxylin and showing very clearly its nucleoli.

This form includes the cells with lobulate nuclei described by Rieder, but in these there exist only deep fissures or indentations (Naegeli).

Such cells may constitute the dominant type in acute leukemia, and usually indicate an extremely rapid and malignant type of case (Sternberg-Naegeli).

Melanemia.—Pigmented leucocytes occur in cases of melanotic sarcoma and, in malaria, free in the blood, during the segmentation stage of the disease.

Iodophilia.—If a dry but unfixed smear be *gently* pressed down upon a generous drop of specially prepared iodin and potassic iodid solution† the normal blood cells take a uniform yellow tint, but in *septic conditions* both intra- and extra-cellular brown granules are seen or a diffuse protoplasmic staining occurs, especially affecting the neutrophilic leucocytes and, rarely, the basophiles and myelocytes.

The reaction is *reasonably constant* in septic conditions (*not in pure tuberculous abscesses*), but is present in many other diseases, such as purpura hemorrhagica, acute miliary tuberculosis, certain typhoids and malignant disease. It is of genuine value in the differentiation of septic from non-septic effusions and joint diseases.

Perinuclear Basophilia.—Neusser's perinuclear basophilic granules occurring in specimens submitted to the triacid stain were regarded by him as indicating the uric acid diathesis, but now are proven to be artefacts and of no diagnostic significance.

The Inclusion Bodies of Döhle.—These may be found in the blood smears of patients suffering from scarlet fever and in several other acute infections (syphilis, measles, erysipelas).

* Probably the parent cells of the normal "large mononuclear leucocyte," and lymphocyte.

† Iodin 1, potassium iodid 3, water 100, gum Arabic 50.

They are readily detected under simple staining with Loeffler's methylene blue, as coccus-like, irregular, rodlike or crescentic bodies lying usually within the polymorphonuclear leucocytes.

Plasma Cells.—These may reach the circulation in congenital hemolytic jaundice, multiple myeloma and leukemia, though normally encountered in small numbers only in the bone marrow, lymph glands, spleen and connective tissue.

Pathologically they are associated with the granulomata and are especially prominent in syphilitic lesions.

They are strongly basophilic and granular, often triangular in outline and the single nucleus of "wheel-spoke structure," is small and eccentrically placed.

A clear perinuclear zone is invariably present in these cells.

THE RED BLOOD CELL.—Each cubic millimeter of human blood should contain from 4,500,000 to 5,000,000 erythrocytes, the former number representing the average normal for the female, the latter that for the male. A fluctuation of half a million cells may be assumed as within normal limits. The average size of these cells is 7.5μ, but a wide normal variation is possible (6.5 to 8.5μ).

The normal.

Under pathological conditions one may meet with (a) extremely small, (b) giant, and (c) nucleated, erythrocytes.

Abnormal Erythrocytic Forms.—*Variations in Shape and Size.*—Deformity and great variation in size may occur in varying degrees in all profound anemias. Thus we may have *poikilocytes*, showing all sorts of bizarre forms, the *megalocytes* (9 to 20μ), and the *microcyte* (3 to 5μ).

The megalocyte is especially common in anemias of the pernicious or the profounder secondary type, containing, according to Da Costa, an excess of hemoglobin in the former and a deficiency in the latter.

The poikilocyte is characteristic of anemia in its more extreme forms, but is chiefly observed in pernicious anemia and leukemia. Cabot lays stress upon the abundance of the oval forms in the former disease.

The microcyte is more commonly associated with chlorosis or relatively severe grades of secondary anemia.

Unusual Erythrocytic Staining Reactions.—In severe anemias and especially in myelogenous leukemia and pernicious anemia one meets with cells which have lost their normal affinity for an acid stain and take indifferently both acid and basic elements of a mixed stain. The coloring is often irregular and in the nucleated forms may affect the nucleus as well as the protoplasm. The greater the deficiency of hemoglobin in the individual cell the stronger is the *polychromatophilic* tendency and its affinity for the basic element in the mixed dye. The *corpuscles of Poggi* take the basic stain in fresh unfixed specimens.

Polychromatophilia.

Erythroblasts.

Nucleated Red Cells.—The erythroblasts are classified by their size as **normoblasts, megaloblasts** and **microblasts.**

The normoblasts (7.5 to 10μ) have a deep basic-staining nucleus with sharp definition and coarse nuclear structure, occupying usually about half

the cell body, but often very small and not infrequently divided or undergoing karyokinesis. They are said to occur in the course of severe anemias in successive crops or blood crises lasting but a few hours, but *are seen chiefly in severe chlorosis, myelogenous leukemia, pernicious anemia or after severe hemorrhage*, and are in general an indication of blood regeneration. Small nucleated. Significance.

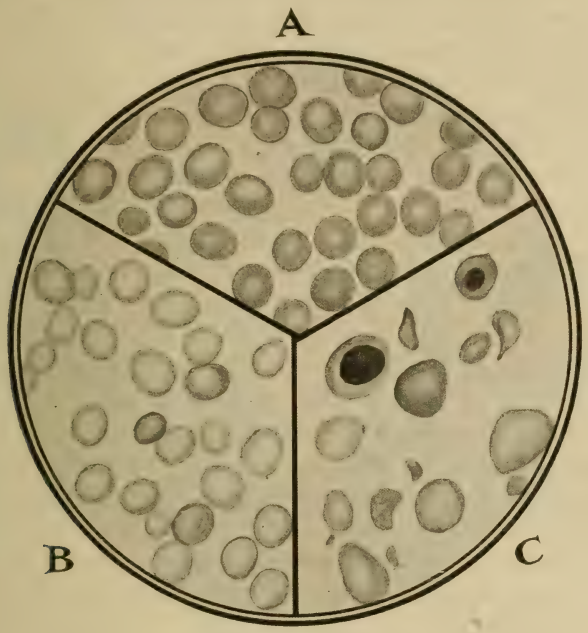


FIG. 36.—A. Normal blood. B. Chlorosis. C. Pernicious anemia. The plate shows the sharp contrast between cells normally rich in hemoglobin and the light cell of chlorosis and also the poikilocytosis and marked variation in size noted in pernicious anemia. (A normoblast and megaloblast also appear.) Stained smears.

The megaloblast (11 to 20μ) has a central or peripherally placed nucleus of fine structure which stains a faint blue or green and in triple stain the specimen is surrounded by a definite white ring. The nucleus is usually large, more rarely strikingly small and deeply staining like the normoblast or microblast. It represents defective hemogenesis and signifies degeneration, being found as a *prevailing type of erythroblast only in primary pernicious anemia, nitro-benzol poisoning*, and *bothriocephalus anemia*. In any severe anemia an occasional cell of this type may be found. Characteristics. Significance.

The microblast (5 to 6μ) presents essentially the same appearance, aside from size, as the normoblast and has the same clinical significance.

Unusual Forms.—Various mixed types are encountered (mesoblasts) which defy differentiation, showing contradictions in size and staining reaction; some, which Da Costa regards as immature normoblasts, show a pale nucleus with acid stain stippling and polychromatophilic protoplasm. Yet others, large in size, contain a small deeply basic staining nucleus and faintly stained protoplasm. These he would classify as megaloblasts. Mesoblasts.

Basophilic Erythrocytes may be encountered, showing fine, coarse, spicular or even ovoid granules of regular or irregular distribution. These cells occur in lead poisoning, in chlorosis with intestinal auto-intoxication and practically all of the profound or pernicious forms of anemia, but are absent in the anemia of renal and hepatic disease, the acute infections, syphilis and diabetes.

Differential value.

Significance.

Cabot has described and drawn extraordinary *intra- and extra-cellular ring bodies* in lead poisoning, pernicious anemia and lymphatic leukemia. They have also been found by Da Costa in the profound anemias of sepsis. Basic ring bodies were previously described by Strauss and Rohnstein. It is now known that these for the most part take the acid stain of Wright's solution, rarely the blue.

Ring bodies.

FIG. 37.—Basophilic stippling and Cabot's ring bodies. (*After Cabot, Ewing and DaCosta.*)

Of slight importance.

Maurer's Spots.—These bodies appear in the infected erythrocytes of victims of estivo-autumnal malaria, as irregular, dark violet-red, spots of variable size.

Special staining is required* and they lack clinical significance other than their association with the (readily demonstrable) malarial organism.

Schüffner's Stippling.—(*"Schüffner's spots"*).—These very minute dark red dots are seen chiefly in the infected erythrocytes of *tertian malaria* and are of no clinical importance.

"Jolly bodies."

Howell's Bodies.†—These are perfectly round, sharply defined bodies occurring at times in the nucleated red cells, usually single, but, in rare instances multiple, staining precisely as nuclear substance would, occurring chiefly, as might be anticipated, in blood crises, but occurring oftenest in the normally staining, non-polychromasic cell.

It will be noted that they are quite unlike the nuclear fragments frequent in the many forms of anemia associated with the appearance of the erythroblast.

THE BLOOD CELL COUNT.—The differentiation of the various types of anemia requires that the number of cells contained in 1 cu. mm. of any given blood be determined directly and differentially and this is readily done with the modern apparatus. The whole process as hereafter detailed should be *smartly* carried out and the different steps outlined be carefully observed.

Instruments and technic.

The Thoma-Zeiss hemocytometer consists of two graduated capillary pipettes, *i.e.*, *the erythrocytometer and the leucocytometer* for counting red and white cells respectively, and a stage carrying a ruled square millimeter which is itself divided into 400 squares of $\frac{1}{20}$ mm. The plateau carrying the ruled surface is surrounded by a moat, the outer walls of which are $\frac{1}{10}$ mm. higher than the ruled plateau.

Pipettes, counting chamber.

* Maurer's modification of the Romanowsky.

† Often called incorrectly "Jolly Bodies." Reported by Howell in 1891; in Germany by Jolly in 1908. The term "Howell-Jolly" bodies is equally incorrect.

This, the older type of blood-counter, is still generally used though the newer counting chambers are much superior.

Accurate dilution.

The Red Count.—The blood is drawn into the pipette by suction or capillary attraction from the drop obtained by puncture until it reaches the point marked 1. The point is then *rapidly* wiped dry and the diluent *quickly drawn in* until it fills the bulb and reaches the mark 101.*

Thorough mixing.

While drawing in the diluent the pipette is revolved between the finger and the thumb to set in motion the mixing bead contained in the bulb and when filled, again thoroughly shaken and revolved for half a minute. *This mixing should be repeated each time before expelling a drop for examination.*

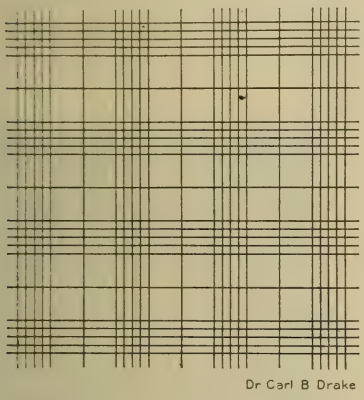


FIG. 38.—Ruling of the Gorgajew-Pappenheim counting chamber.

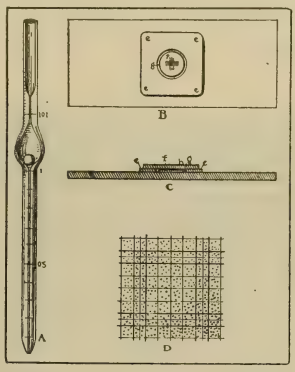


FIG. 39.—Thoma-Zeiss blood counter, showing pipette, counting chamber and a part of the ruled field.

Higher dilution.

One has then a mixture, each drop of which presents a blood dilution of 1 : 100.

Many prefer a 1 : 200 dilution readily obtained by using the mark 0.5 as the upper limit for the indrawn blood. The blood rises quickly if the pipette is perfectly clean and may exceed the proper level if not watched or checked by withdrawing the point and quickly wiping it. If the higher dilution is used any excess is readily blown out or better *drawn down by touching the tip with filter-paper or blotter.*

Disposal of excess.

The next step consists in expelling the diluent occupying the capillary portion of the tube, after which a small drop of the mixture in the bulb is placed upon the central disc (shown in Fig. 39 (B)), the cover-glass is placed in position and the cells given time to settle.

Transfer to counter.

The drop should just fill the central plateau without running over into the

Caution.

* The diluent usually used is Toisson's solution. Formula: Methyl violet 5B (0.025), sodium chloride (1.0), sodium sulphate (8.0), neutral glycerin (30.0), distilled water (160.0).

Hayem's solution contains mercuric chloride (0.25), sodium chloride (0.5), sodium sulphate (2.5), distilled water (100.0). Any such solution must possess two qualities—it must prevent coagulation and be isotonic.

moat around it when the cover-glass is applied, else the process must be repeated after thoroughly cleaning and drying both the cover-glass and the counting surface.

If the proper distribution of the drop upon the plateau has been attained, concentric color rings ("Newton's rings") should be visible when the surface of the applied cover-slip is held toward the light at, or slightly below, the level of the eyes.

Test of accuracy.

The absence of these rings vitiates the count and indicates usually dust or moisture on the glass. The correct size of the drop itself must be learned by experience. It should cover the plateau without overflowing.

Proper field.

The specimen, when placed under a microscope carrying a Leitz No. 6 objective and No. 4 ocular or their approximate equivalents, *should show a uniform distribution* of blood cells over the entire field, ruled or unruled.

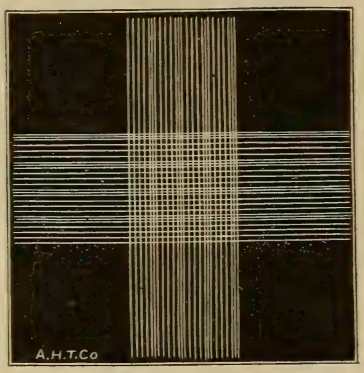


FIG. 40.—Thoma–Zeiss ruling.

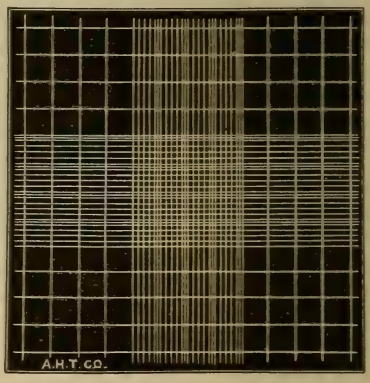


FIG. 41 —Neubauer's ruling.

Counting.—*One must count now the number of red blood cells lying within a definite known number of ruled squares, including for each square those in contact with or overlapping two of its four sides.*

Necessary checks.

For thoroughly accurate painstaking work, such as would be necessary in actual research, the entire 400 squares should be counted and several drops of the mixture so treated. For clinical purposes a less number may be taken if the blood is well distributed, but several should be examined. With pencil and paper at hand an accurate record of the cell count should be jotted down as the work proceeds.

Rationale.—The number representing the total cells counted must be divided by the number of lesser squares in which they were contained. The result of this division would represent the average number of cells in each ruled square. The sides of the small squares each measure $\frac{1}{20}$ mm. and the depth of the liquid $\frac{1}{10}$ mm. yields a third dimension. Multiplying $\frac{1}{20} \times \frac{1}{20} \times \frac{1}{10} = \frac{1}{4000}$ cu. mm. as the cubic content of each square used during the test.

The blood itself has been diluted 100 times. The cubic area of each smaller ruled square is $\frac{1}{4000}$ cu. mm. We desire to record the number of erythrocytes in each cubic millimeter of the blood under examination.

We must, therefore, multiply the average number of cells per square by 4000 *to get the number in* 1 *cu. mm. of the mixture, and this result by* 100, *the degree of dilution of the blood, to get the actual number of erythrocytes per cubic millimeter of the undiluted blood itself.*

Simple rule.

This resolves itself into the following simple formula in dilutions of 1–100: *Divide the total number of red cells counted by the number of squares covered, and multiply the quotient so obtained by* 400,000.

Example.—One hundred squares are found to contain 1200 red cells: 1200 ÷ 100 = 12 cells per square = 12 cells in $\frac{1}{4000}$ cu. mm. of blood diluted 100 times. 12 × 4000 × 100 = 4,800,000 cells per cubic millimeter.

Rapid method.

If but 40 *squares were covered,* 480 *cells would have been counted and, by an arithmetical coincidence, all* 40*-square counts with a dilution of* 1–100, *and all counts of* 80 *squares with a dilution of* 1–200, *may quickly be determined by placing four ciphers at the right of the figures representing the total cell count, i.e.,* 40 squares (1–100 dilution) contain 480 cells, 80 squares (1–200 dilution) contain 480 cells; annexing four ciphers in each instance, we have 4,800,000, the actual count per cubic millimeter.

If the entire 400 ruled squares composing the large square were counted in this case, the total cells enumerated would have been 4800, or ten times the number of cells in our 40 squares and would represent the number of red cells in $\frac{1}{10}$ cu. mm. of blood diluted 100 times, *i.e.*, $\frac{1}{1000}$ of the number contained in a cubic millimeter of the undiluted blood.

Hence in counting 400 *squares the exact number of red cells per cubic millimeter of blood may be determined by annexing three ciphers to the number representing the total of cells enumerated in the entire large square.*

Precautions.

If to be again used at once, air should be blown through the mixing tube by means of a hand bulb and rubber tube, not the mouth. The counting chamber should be cleaned with cold water only and should not be exposed to high degrees of heat or, for long, even to direct sunlight, for fear of softening the cement.

Instruments and technic.

The Leucocyte Count.—For this the large-calibered pipette is used with a diluent consisting of a 1 per cent. aqueous solution* of *glacial* acetic acid which leaves visible only the white cells and the nucleated reds.

This pipette is graduated in tenths to a point 1 (one) for the measurement of the blood used and shows also a symbol 11 (eleven) above the bulb containing the mixture of blood and glacial acetic acid solution. When filled, the blood is ten times diluted.

The most accurate counts are made by using 0.5 *rather than* 1 *as the basis of dilution, and even this requires a good-sized drop of blood. Furthermore, the pipette must be kept nearly horizontal at the time and after the diluent is added, else it will run out of the tube.*

* A 1 per cent. solution of glacial acetic acid is more certain in its action and a few drops of methyl violet or gentian violet stain may be added to advantage.

Türck's chamber.

The diluent is added to the point 11 and the count made with the erythrocyte counting chamber, or better with Türck's chamber in which the central ruled square (*one square millimeter* ($\frac{1}{10}$ cu. mm.) containing 400 small squares) is surrounded by eight partially ruled squares of the same size. The total obtained by counting the leucocytes in the nine square millimeters (representing 3600 small squares) is multiplied by 200 (dilution 20 by 10 to get cubic millimeters) and divided by 9, assuming a dilution of 1–20 as above described.

Basis of count.

The actual count is based, therefore, upon the total count of white cells in any number of square millimeters, multiplied by the dilution, multiplied again by the third dimension of the cubic millimeter (10) and divided by the number of large squares (square millimeters) covered in the count.

Use of erythrocyte counter.

If the ordinary counting chamber is used, the entire field of 400 ruled squares should be counted and the total number of leucocytes found multiplied by 100 if the dilution be 1–10 or by 200 if it be 1–20. The process is greatly facilitated by inserting in the tube of the ocular an eyepiece diaphragm which will cut off exactly 100 of the small squares. This may be bought, or can be made by the physician from metal or cardboard. The leucocytes in 400 small squares of the erythrocyte chamber may then be rapidly counted together with as many additional outside diaphragm fields as are necessary to correct work.

Drops required.

*Accurate white blood counts require patience and the counting of a large number of fields, for a slight error has a disastrous effect.** *Usually 2 drops of the 1–10 or 4 drops of the 1–20 dilution will enable one to count 140 to 150 cells.*†

Laborious but valuable.

Differential Counting.—This involves the examination of from 500 to 800 leucocytes in fixed and stained blood smears, and a determination of the exact number of each variety present. The field should be systematically and carefully gone over, and if ordinary care is observed the procedure offers no difficulty for one who is sufficiently skilled to recognize the various types of white cells and the abnormal erythrocytes which should be included in any such procedure. The process is tedious but oftentimes necessary and valuable, and necessitates, as a rule, the counting of at least two or three entire stained smears.

Important Detail.—*The blood counter pipette must in every case be cleaned* **immediately after using:** *first, with water; second, with alcohol; third, with ether; and the glass bead should be absolutely free and movable after the process is completed.*

Many Types of Blood-counters.—There are many modified forms of the "blood-counter" of the Thoma type and amongst them is one of American make and easily procurable.

This is the Thoma-Levy hemocytometer and is thus described by the manufacturers:

* The ingenious self-filling and self-measuring pipette of Durham has many advantages but requires separate mixing vessels.

† In the leukemias the leucocyte count is best made with the ordinary erythrocyte counter and pipette for the dilution should be 1–100 rather than 1–10 or 1–20.

"The Bürker type of counting chamber, of either the new or old construction, has a further advantage over the original Thoma construction—which consists of a circular ruled disc cemented on the slide in the center of a circular cell, also cemented on the slide—in that capillary attraction is used to fill the Bürker cell after the cover glass is in position. This method insures a much more uniform distribution of corpuscles over the entire field and the effect of atmospheric pressure upon the depth of the solution is materially lessened. These new Thoma-Levy counting chambers of the Bürker type are now supplied with various rulings, *i.e.*, Thoma, Zappert, Türck, Neubauer, Fuchs-Rosenthal, etc. The Neubauer ruling is now recommended as the most satisfactory for modern technic. The method of ruling used in the manufacture of these chambers provides a line with absolutely clean-cut edges and of distinctly increased visibility when the chamber is filled with solution for the count. This increase in visibility of the ruling greatly lessens the eye fatigue experienced in making repeated counts.

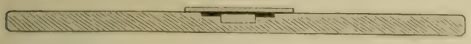


FIG. 42.—Vertical longitudinal section of slide, with cover glass in position, showing new method of construction.

FIG. 43.—Thoma–Levy counting chamber with original Thoma ruling.

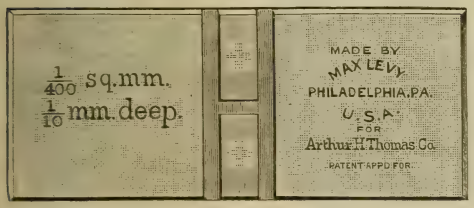


FIG. 44.—Thoma–Levy counting chamber, Bürker double type with two Neubauer rulings.

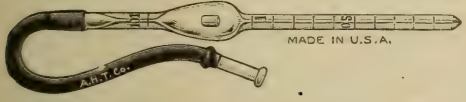


FIG. 45.—Mixing pipette for red corpuscles.

FIG. 46.—Mixing pipette for white corpuscles.

"In the old type of Bürker chamber two ruled areas are provided upon rectangular pieces of glass cemented on the main slide. These ruled rectangles were separated by a small moat to allow free passage of the diluted blood. On either side of these ruled rectangles two unruled rectangular pieces of glass were cemented which extended the entire width of the slide. These were exactly 1/10 mm. thicker than the ruled rectangles, so that when the cover glass rested on these the required depth of solution over the ruled area was attained.

"In the Levy construction a rectangular depression is cut into the slide itself extending across the entire width. In the middle of this depression is permanently fixed a rectangular strip of glass, also extending entirely across the slide, and on this are the rulings. When the cover glass is placed in position on the slide itself the solution over the ruled areas is of the re-

quired depth. The method of construction entirely removes the possibility of the loosening of the cell by the drying out of the balsam cement and reduces the possibility of the loosening of the ruled counting surface.

"In the Bürker-Neubauer counting chambers the rectangular glass in the center of the cell is divided by a central moat; each half being provided with a Neubauer ruling, so that both red and white counts may be made at the same time without the necessity of cleaning and refilling the counting chamber." (Fig. 44.)

Courtesy of E. Leitz, New York.

FIG. 47.—Hemocytometer (the Thoma-Metz).

FIG. 48.—Eye-piece ruling of Thoma-Metz.

The Neubauer modification of the original Thoma ruling is probably the most satisfactory of all. In it the central square millimeter is exactly the same as in the original Thoma ruling. Surrounding this there are 8 additional square millimeters, each subdivided into 16 smaller squares, and these are of extreme value by reason of the greater accuracy and convenience which they afford in counting the white blood corpuscles. (See Fig. 41.)

The actual technic for using the Bürker chamber is extremely simple and may be briefly summarized as follows:

1. The ruled areas and slide are carefully cleaned and made absolutely free from dust.

2. The cover glass is applied and Newton's rings* must be obtained as evidence of the approximation of the two surfaces.

3. The tip of the projecting ruled rectangle is quickly touched with the point of the filled red or white blood pipette, permitting the diluted blood to

* The Newton's interference rings are not readily obtainable in the Levy construction as in the older type because of the mat or ground surface of the slide upon which the cover glass rests. Because of this mat surface accurate approximation of the surface of the cover glass and slide is readily obtained.

The original Bürker instrument and Gorgajew-Pappenheim modification of it carry clamps which aid in obtaining and maintaining accurate contact. (*Author.*)

flow under the cover glass to just the extent sufficient to cover completely the ruled rectangle but not flow over into the moat on either side. No bubbles should form in this process.

4. After allowing three minutes for settling, the evenness of distribution is determined either by low-power examination with the microscope or by placing the counter upon the stage of the microscope, which is illuminated by a mirror with the diaphragm open as wide as possible. If now the counting surface is viewed obliquely with the unaided eye, any irregularity in the distribution of the cells is easily recognized by variations in the density of the blood film. If such irregularities are observed, the cover glass should be removed, the chamber carefully cleaned, and the procedure repeated.

The Thoma-Metz Hemocytometer.—The counting plate is mounted in the plane of the ocular diaphragm of eyepiece No. 11. The Leitz objective No. 6, is used in connection with it. The dilution of the blood is carried out with the aid of two Thoma pipettes of regular type, following the known method.

The large square in the center of the counting plate (Fig. 48), is divided into four smaller squares and serves for the counting of the **red blood corpuscles.** The large square is surrounded by a **circle** which is divided also into four parts of equal area, serving for the counting of **white blood corpuscles.**

Figure 48 represents a diagram of the counting plate itself. The regular chamber of 0.1 mm. depth is used for the counting by this method.

Directions for the Counting in a Solution of 1 Cu. Mm.—(*A*) *For Red Blood Corpuscles.*—The dimensions of the large square of the counting plates are so calculated that one side of same covers 0.1 mm. of the object. The area, therefore, is equal to $0.1 \times 0.1 = 0.01$ sq. mm., and the cubic contents of that area of the object as covered by the entire square is equal to $0.01 \times 0.1 = 0.001$ cu. mm. The number of red corpuscles in the solution—diluted 1:100—amounts therefore, to $100 \times 1000 = 100{,}000$ times the corpuscles found in the square counted.

Example.—52 red corpuscles found in the square. $52 \times 100{,}000 = 5{,}200{,}000$ red corpuscles in one cu. mm., solution.

In order to achieve a reliable average count it is advisable to move the slide and count in various zones of the solution.

(*B*) *For White Blood Corpuscles.*—The circle established for the counting of white corpuscles covers ten times as much area as does the square for the counting of red corpuscles. This circle, therefore, covers an area of $10 \times 0.01 = 0.1$ sq. mm. of the counting chamber of a depth of 0.1 mm. and the cubic contents amounts to $0.1 \times 0.1 = 0.01$ cu. mm. The actual number of white corpuscles as contained in the solution—diluted 1:10—will therefore amount to $10 \times 100 = 1000$ times the number as found in the circle.

Example.—8 white blood corpuscles found in the circle. $8 \times 1000 = 8000$ white corpuscles in 1 cu. mm., solution.

In order to achieve a reliable average count, it is advisable to move the slide and count in various zones of the solution.

The two factors: 100 × 1000 for red blood corpuscles are engraved in "**red.**" 10 × 100 for white blood corpuscles are engraved in "**white**" on the counting slide.

Advantages.—The counting plate is produced in a photographic way and can be focused sharply for every eye by means of the adjustable eye lens, and therefore, the ruling appears in sharp lines in the counting field.

For the counting of red as well as white blood corpuscles, separate fields are used. These fields are, in their area, so adjusted that a unit for multiplication is established. This factor allows **quick counting.**

The apparatus is adjusted for an optical equipment of **objective No. 6 (4 mm. focal length) and ocular No. 11 (5 X),** as these are the lenses mostly used for blood-counting work. Small variations, as far as lenses of other makes are concerned, can be overcome through the adjustment of the tube length. On the bottom of the slide, a square is drawn and the proper optical relation is established when this square covers the large square of the counting plate.

(The author has had no experience with the instrument and has been unable to secure one for test purposes.)

Seldom employed.

Oliver's Hemocytometer.—This ingenious instrument is shown in Fig. 49, and save in the severer types of anemia and in leukemia where the excess of leucocytes vitiates any optical method, is fairly accurate in skilled hands.

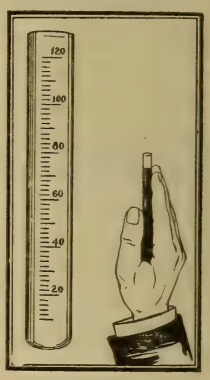


FIG. 49.—Method of holding Oliver's hemocytometer.

Method.—A small amount of Hayem's solution is placed in the glass tube and into it is stirred the blood taken up by the capillary pipette. In a dark room it is then diluted gradually with the Hayem's mixture until a bright horizontal line becomes visible as the observer looks through the mixture at a candle flame beyond. Each point on the scale represents 50,000 red cells.

Precautions.—(*a*) One looks at the edge of the tube, not its flat face. (*b*) The pipette should be thoroughly clean before using. (*c*) The tube should be inverted to secure proper admixture each time that the diluent is added. (*d*) An imperfect marginal line forecasts the appearance of the complete transverse line and warns one to go slowly with the dilution. (*e*) A small Christmas candle should be used in a dark room. (*f*) The tube should be close to the eye and the observer fully 10 feet from the candle.

The procedure is seldom employed at the present time. It has too limited a range and the personal equation is too great a factor.

Technic.

The Hematocrit (*Hedin-Daland*).—This consists of a graduated capillary tube, each degree of which represents 100,000 erythrocytes. The tube is filled with blood and placed (with a similar tube opposite) in the centrifuge which should be revolved at high speed for at least two minutes when the

red column will represent the erythrocytes present as indicated by the scale. The tube when filled by suction is dried and closed at its other end by the vaseline coated finger which is kept in place while the rubber suction tube is withdrawn. The results obtained are rapid but only approximate when used in the severer types of anemia. As pointed out by Capps, it affords a means of obtaining information relating to cell volume.

It is a simple matter to calculate the normal height of the red blood cell column in the hematocrit tube as compared with the height of the total blood column. This is usually 50 on the scale tube and this figure is reckoned as 100 per cent. normal.

Volume index.

The figure so obtained is divided by the figure representing, in percentage, the number of red cells per cubic millimeter counted in the blood of the same individual, as compared with the normal, 5,000,000 cells per cubic millimeter The quotient is the red cell "*volume index*" and parallels in a considerable degree the "*color index*" heretofore described, both as regards its variations and their clinical significance.

The normal red cell "*volume index*" is 1. In primary pernicious anemia it is high; in secondary anemias, reduced; in chlorosis, distinctly low.

These variations, if marked, are usually obvious to the trained observer in an examination of the simple well-made blood smear.

The method may find its wider usefulness in the detection and measurement of blood changes other than those of anemia.

LEUCOCYTOSIS AND LYMPHOCYTOSIS.—Assuming that the term leucocytosis covers an increase of the polymorphonuclear neutrophilic cells, both relative and absolute, it is evident that *one must know first what constitutes a normal leucocyte count; second, what conditions other than disease may increase the number.*

The Normal Average.—This varies from 6,500 to 10,000 to the cubic millimeter of peripheral blood, the average being 7500.

Physiologic Leucocytosis.—The most important is the so-called digestive leucocytosis which represents on the average an increase over normal of about one-third. The variation is greater after a heavy proteid meal than on a vegetable diet and reaches its maximum usually within from two to four hours after a meal. The increase itself may be purely polymorphonuclear or, more rarely, is common to all normal forms. It may be delayed in cases of subacid dyspepsia and is very marked in nursing infants and diabetics.

Digestion leucocytosis.

The so-called preagonal leucocytosis may be observed immediately before death, the counts occasionally reaching 20,000 or even 30,000 cells. Cabot states that in pernicious anemia this may take the form of so decided a lymphocyte increase as to simulate lymphatic leukemia; nevertheless, as a rule, it is a polymorphonuclear increase due probably to the stasis of a failing circulation and a profound toxemia such as that which may be employed in the experimental production of a leucocytosis. In such instances the primary increase is lymphocytic, the polymorphonuclear increase following later on. It is important to remember that both in the later months of pregnancy in primiparæ and during the first week following delivery a

Misleading.

Pregnancy.

New-born babes.

moderate leucocytosis is the rule. So also in new-born babes the count usually ranges from 18,000 to 20,000 during the first two days of life and after the second week and during the first year it remains at 10,000 to 15,000 per cubic millimeter. The type of physiologic leucocytosis is almost invariably polymorphonuclear though there may be a coincident lymphocyte increase in the new-born, in malignant disease, or in hemorrhage. See "lymphocytosis" on following page.

Differentiation of Leucocytosis from Leukemia.—The number of white cells present does not suffice to differentiate the one, a mere transitory symptom, from the other, a peculiarly intractable and fatal chronic disease.

Crucial point.

It is rather the type of white cell which dominates the clinical picture, or the blood findings as a whole combined with other signs and symptoms of known character and association that sets **leukemia** *apart so clearly and definitely as to leave little opportunity or excuse for error.* With rare exceptions leucocytosis means a polymorphonuclear increase alone or, more rarely, a mere general increase of leucocytes of the normal types.

Various associated conditions.

The Leucocytosis of Disease.—*So many diseases are associated with a more or less marked leucocytosis that it is only necessary to enumerate some of the more striking instances, and more useful to remember the exceptions, or, more particularly, the diseases in which a diminution* (**leucopenia**) *occurs.*

Sepsis.

Practically all inflammations, particularly of serous membrane, and any suppurative process attended by toxemia, would ordinarily produce leucocytosis, yet we may have these, lacking such a phenomenon (*a*) *because the inflammation*

Basis.

or toxemia may be too slight to excite a reactive change; (*b*) *because the toxin overwhelms the organism, thus preventing leucocytic reaction;* (*c*) *because the organism is primarily too feeble to respond.*

In illustration one may cite lobar pneumonia as representing a disease in which leucocytosis is ordinarily marked, and typhoid fever in which a leucopenia is ordinarily found.

These diseases stand at opposite poles, one being sthenic and the other asthenic; one of brief duration, the other prolonged and tedious; one showing early and profound toxemia, the other allowing most of its victims to escape by resolution before the system is overwhelmed by toxins.

Highly important.

The case of lobar pneumonia which shows a leucopenia is practically hopeless. The case of genuine typhoid fever carrying a leucocytosis is one with complications.

Acute appendicitis offers another illustration. In this disease one may have an inflammation and toxemia so slight as to produce no leucocytosis; an overwhelming toxemia with the same result; or, as it is the rule, a well-defined leucocytic reaction representing a good defensive mechanism opposed to sharp infection.

It is useless to attempt to name or even tabulate all the conditions giving rise to leucocytosis. In abscess it is practically constant, but in *malignant disease* a high count may be found only in rapidly growing tumors of the kidney, lungs or liver, and in the author's experience especially in metastatic hepatic invasion from gastric carcinoma. In *ptomain poisoning, uremia, cholemia,*

and gas poisoning the leucocytosis may be marked. So also in cases of *acute delirium, convulsions, after surgical operations* or indeed *after general anesthesia.* A polymorphonuclear percentage above 85 strongly suggests the presence of actual suppuration or gangrene. The blood findings of the various diseases are discussed under the individual headings.* Confusing factors.

Leucopenia.—*This covers any leucocyte count running below* 5000 *cells, excluding, of course, those cases of reduction associated with profound anemia.*

As might be expected, this condition may be present as a result of malnutrition or starvation, but its chief importance is its occurrence in certain diseases of the infectious and toxemic type.

Among these are typhoid, paratyphoid, malaria, Malta fever, influenza, measles and rötheln, tuberculosis (in the absence of septic symptoms), leprosy, trypanosomiasis, and less commonly in chlorosis, chronic infantile gastro-enteritis, and, following the use of agaricin, atropin, camphoric acid, ergot, picrotoxin, sulphonal and tannic acid. List of diseases.

Lymphocytosis.—*This may be either relative or absolute, the former without any necessary increase in the total white cell count, the latter covering both an increase in percentage and in the total lymphocytes. The latter is almost always a lymphatic leukemia.*

It should be remembered in this connection that in infancy the lymphocytes and transitional cells may constitute 55 or 60 per cent. of the total white cells, gradually dropping to the normal figure at the age of four or five with a corresponding increase in the polymorphonuclear forms. Therefore, the increased percentage has little significance at early ages. Normal in infancy.

Either the large, small or transitional mononuclear forms may predominate in any case, and the condition is invariably pathologic when occurring in older children or in adults. It is usually associated with profound malnutrition, and especially marked in the terminal stages of diseases of that type as well as in marked anemia and in several of the acute and chronic infections characterized by leucopenia, *notably malaria, typhoid, Malta fever, scarlet fever, diphtheria, measles, tuberculosis and trypanosomiasis. So also we find the condition marked in ailments involving the lymphatic glands or spleen or the invasion of lymph channels by malignant growths.* As regards drugs, it is said to follow the administration of *thyroid, quinin and pilocarpin.* Its occurrence in syphilis in which it is combined with eosinophilia is of some diagnostic importance. Abnormal. Syphilis.

Eosinophilia.—The determination of this condition is made by a differential count establishing their proportion, associated with a formal count of the total leucocytes, the latter multiplied by the former giving the total number of eosinophiles in a cubic millimeter.

It is of lessened importance as a clinical sign because of its presence in a great number of diseases, but its constancy in trichiniasis, hydatid disease and in the victims of the more important intestinal parasites is of considerable value. Value.

* It is affirmed by some authorities that the percentage increase of polymorphonuclears (relative preponderance) represents the degree of toxic absorption and the total numerical increase of leucocytes measures the resisting power of the organism.

The attempt made by some of the foreign hematologists to create a group of which eosinophilia should be diagnostic has not proven successful.

Diseases yielding reactions.

It is, as stated, of some clinical value in syphilis when combined with lymphocytosis and its constant presence in spasmodic asthma is of interest. In scarlet fever there is at first a low white count but after a few days a marked leucocytosis and decided eosinophilia are present. Both are said to be absent in measles.

A double process.

Counting the Blood Plates.—*Indirect Method.*—This requires a diluting fluid* through a drop of which placed upon the finger the puncture is made. After mixing, a differential count is made to determine the ratio of plaques to erythrocytes. It is then only necessary to count the erythrocytes by means of a counting chamber to know the number of plaques present in the given specimen.

Use erythrocyte counter.

Direct Method.—The direct method of Wright and Kinnicutt is preferable. One employs as diluting fluid solutions of *brilliant cresyl blue* (1:300, aqueous) and *potassium cyanide* (1:1400, aqueous).

The solutions are mixed just before use in the proportion of two parts of the former to three parts of the latter.

Caution.

The original solutions must be freshly prepared, or, refrigerated if kept as stock solutions.

Appearance of platelets.

The blood platelets appear clearly defined as oval, round, or somewhat elongated bodies stained lilac and sharply contrasted with the leucocytes, which show a deep blue nucleus and light blue protoplasm. The red cells are decolorized.

The size of the platelets is variable, being larger when few are present, smaller when occurring in increased numbers, but they average about 3 microns in diameter.

Normal count.

Normally, their numbers vary from 150,000 to 300,000 per c.mm., and *the count is made exactly as in the case of the erythrocytes.*

Determination of Hemolytic Resistance.—If blood is added to water it immediately "*lakes*," *i. e.*, gives up the hemoglobin of its erythrocytes to the liquid. If the osmotic pressure is equalized by the addition of some suitable substance such as sodium chloride (0.9 per cent.) the solution becomes isotonic and no "laking" occurs.

By using properly graduated solutions of sodium chloride or other substances the resistance of any given blood to hemolysis may be determined.

The same process is applied in determining the proper donor of blood for transfusion for both donor and patient must belong to the same group with respect to cell resistance whether to iso-hemolysins or iso-agglutinins.

Total Blood Volume.—It is wholly probable that the ingenious and relatively simple procedure of Keith, Rowntree and Geraghty, will supplant the methods of Haldane and Smith, Morawitz, Zuntz and Plesch, Abderhalden and Smith, and von Behring.

Rationale.

The principal feature of this latest procedure is the introduction into the blood of a rapidly diffusible, slowly absorbable dye, known as "vital red"

*Aqueous solution of sodium chloride 1 per cent. and potassium bichromate 5 per cent. (Determann).

(disodium-disulphonaphthol-azotetramethyl triphenyl methane), and colorimetric comparison with a properly standardized mixture of the dye and the unstained plasma of the patient. Its slow absorption and elimination and relative non-toxicity (3 c.c. per kilo of body weight being wholly without poisonous effects) insures the possibility of sufficient time retention to insure thorough mixing with the plasma, without appreciable loss of the dye, unless to some slight extent through fixation by the tissues.

The results obtained agree more closely with the older ideas than with the modern views as to the relation of total blood volume to body weight. Keith and his co-workers find that the average blood volume is 85 c.c. per kilo of body weight, equal to one-eleventh of the body weight, and varying between that figure and one-thirteenth.

Normal average.

As regards the partition of the blood components 57 per cent. was found to represent plasma, and 43 per cent. erythrocytes and leucocytes.

Normal ratio.

The plasma content as determined by the hematocrit varies from 42 to 56 c.c. per kilo of body weight.

One of the interesting results reported bears upon blood volume in arterial hypertension, or hypotension. In the chronic hypertension of renal cases, it was found normal or even decidedly reduced. In obesity the blood (and plasma) volume is reduced, in anemia often increased as to total plasma.

The actual technic while relatively simple is too extended to receive detailed description in a work of this type and too time consuming and exacting in its demands to make it of general availability to the practitioner in the comparatively infrequent instances in which its employment might be of especial clinical interest.*

Time consuming.

A *B*

FIG. 50.—Delayed coagulation of blood, a simple test. (*After Da-Costa.*)

Coagulation Time.—It is sometimes important to estimate the coagulation period in hemophilia, purpura and in "obstruction of the biliary tract with or without jaundice" (Cabot), as in these conditions clot formation may be greatly retarded.

Often important.

For accurate work the method and instrument of Russell and Brodie as modified by Boggs may be used; or for rough determinations, in the absence of better appliances, the simple method represented by Fig. 50. In the former coagulation should occur within from three to six minutes; by the simpler method, in from two and one-half to five minutes. The latter merely involves the placing of several drops of the suspected blood upon slightly warmed microscope slides which are tilted upward at varying intervals until they appear as in *B* of the plate, the elapsed time representing the coagulation period. Another rough but useful method consists in placing the drop of blood upon a *perfectly clean glass slide* and passing a needle through it every twenty or thirty seconds until the needle drags the clot.

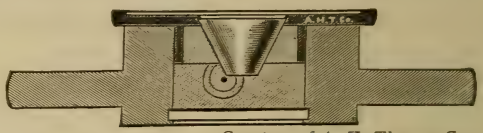
Normal periods.

The Boggs coagulometer consists of : (*a*) a round glass-bottomed "moist

*Reference to the extended original article is necessary. Keith, Rowntree and Geraghty, Archiv. Int. Med., Oct. 15, 1915, Vol. 16, No. 4, pp. 547–576.

chamber"; (*b*) a cover fitting into and closing the chamber and carrying in the center a cone of glass the truncate apex of which lies within the "moist chamber"; (*c*) a capillary tube opening into the side of the chamber through which a jet of air can be pumped with a hand bulb so as to impinge upon the drop of blood taken upon the tip of the glass cone for examination; (*d*) a small opening in the upper surface of the cover of the chamber, which serves as an air outlet.

Procedure.—The lobe of the ear is punctured and the time of appearance of the blood accurately noted. The tip of the glass cone receives a blood drop, the cell is at once covered, the chamber is placed upon the stage of a microscope, and at intervals of 30 seconds a soft current of air is played upon the blood drop. Three essential stages of *movement* are seen to ensue as affecting the blood *corpuscles:* (1) Independent. (2) Clumped. (3) Mere "radial" mass response to the air current, denoting complete coagulation.*



Courtesy of A. H. Thomas Co.

FIG. 51.—The Brodie-Russell coagulometer as modified by Boggs.

Wassermann Test.—The discovery of the *spirochæta pallida* was closely followed by this reaction for syphilis which combines the phenomenon of hemolysis with that of complement fixation (Bordet-Gengou).

The test departs from the strict and definite "fixation of complement" reaction in permitting the use of antigen derived from certain *normal* tissues (Noguchi) as a substitute for the specific antigen hitherto deemed necessary to such tests. (See "Wassermann Test".)

The reader is referred to the works devoted to serology for a full discussion of the test which is so delicate and so beset by confusing factors as to be of little value in the hands of anyone save the trained and up-to-date serologist.

The value of positive reactions is undoubted, when these represent the work of the specially trained expert.

Negative reports should not greatly influence the question of test treatment or be permitted to greatly influence an opinion based upon a strongly suggestive history or clinically significant symptom-group.

The Determination of Alkalinity.—Personal experience with this method has convinced the author that it does not merit attention as a *clinical* procedure.

Exudates and Transudates.—In general a *transudate* does not coagulate spontaneously unless there be much blood admixed, the specific gravity is 1015 or less and it contains not more than 2.5 per cent. albumin. An *exudate* coagulates promptly, contains 4 per cent. or more of albumin, and shows a specific gravity of 1018+.

* The mass is fixed and recovers its outline when the gentle air current ceases, much as an indented rubber ball recovers its outline after indentation. Just preceding this terminal stage the clot has become so far fixed as to promptly correct the slight dislocation produced by the air current.

CYTODIAGNOSIS.—Widal has given this name to the differential study of the leucocyte content of pleural, peritoneal and cerebro-spinal fluids as they are obtained in operative or diagnostic aspiration. It is a valuable procedure within narrow limits.

The fluid must be centrifugalized, and if immediate attention cannot be given it, any clot may be broken up by shaking in a flask with small glass beads or the fluid may be drawn into a syringe or aspirator flask half full of a solution consisting of calcium oxalate (2 parts) and normal saline solution (1000 parts, *Sahli*). From 10 to 20 c.c. should be obtained if possible. The centrifuge must be run from five to ten minutes according to the speed obtainable and the fluid kept on ice and examined within twenty-four hours. Examinations are best made by staining smear preparations as in the case of the blood. The triacid stain is satisfactory, or Wright's stain, plus one-third its volume of pure methyl alcohol, may be used, to save heating the specimens, though its methylene blue may overstain in the sharply alkaline serum.

Under the microscope we consider erythrocytes, endothelium and tumor cells as well as leucocytes, and the following reasonably dependable conclusions may be drawn:

(*a*) *The presence of large amounts of blood suggests carcinoma, tuberculosis, and the hemorrhagic diseases, but may be present in cardiac, scorbutic, and nephritic cases, though usually accidental.*

(*b*) *In general the predominance of polymorphonuclear forms indicates marked and relatively recent infection.*

(*c*) *A large number of mononuclear cells indicates a less acute process, its later or chronic stages, or the chronic stage of a non-specific exudate. A combination of a considerable quantity of red blood cells and an excess of lymphocytes strongly suggests tuberculosis.*

(*d*) *A large number of endothelial cells unassociated with any considerable number of leucocytes speaks for a transudate.*

(*e*) *Specific tumor cells may, if typically grouped, prove the presence of a malignant growth, but ordinarily can with no certainty be distinguished from endothelial cells. The combination of an excess of endothelial cells and red blood cells strongly suggests tuberculosis.*

Primary Tuberculosis is indicated by a predominance of lymphocytes after a week or ten days; *acute infection, by polymorphonuclear preponderance.* Predominance of endothelial cells in *tesselate* arrangement indicates a transudate. *Secondary tuberculous pleurisies may* show polymorphonuclear predominance or a sediment of necrotic cells and débris. An exudate showing bacteria and 90 per cent or more of polymorphonuclears means that empyema is imminent (Musgrave).*

INOSCOPY.—Jossuet's method of predigesting the coagulum of suspected exudates as preliminary to the examination for *tubercle bacilli* has

* Musgrave highly recommends Wright's stain, 3 parts, methyl alcohol, 1 part. Stain twenty to forty-five minutes, treat with 8 or 10 drops of water, let it stand two or three minutes and wash gently by flooding with water several times, allowing it to stand a few seconds with each washing.

proven of much value in pleuritic cases but is now largely superseded by the "antiformin method." Its findings have less accuracy, however, than animal inoculation and possibly than cytodiagnosis, so that it is the positive factor that should be considered, the failure of the test being no adequate proof of the absence of tuberculosis.

Technic.—The fluid is collected in sterilized flasks and the clot, when formed, thoroughly washed on sterile gauze spread over the mouth of a funnel or other receptacle. The clot is then placed in a flask or flasks in each of which is poured from 20 to 30 c.c. of the solution (pepsin 2 grams, glycerin and strong HCl of each 10 c.c., sodium fluoride 3 grams, distilled water 2000). Digestion will be complete in about two or three hours at body temperature. Centrifugation of the mixture for ten minutes is followed by decantation and the staining of cover-slip preparations to which a bit of egg albumin may be added. Gabbett's method and stain should be used, but the preparation must not be as vigorously decolorized as is usual in sputum mounts, thirty to forty-five seconds being sufficient.

THE ANEMIAS

TYPES OF ANEMIA.—*There are three principal clinical types of anemia*: (1) **simple anemia or chlorosis;** (2) **secondary anemia,** *which as its name implies is that which follows or is associated with other diseases;* (3) **pernicious anemia.**

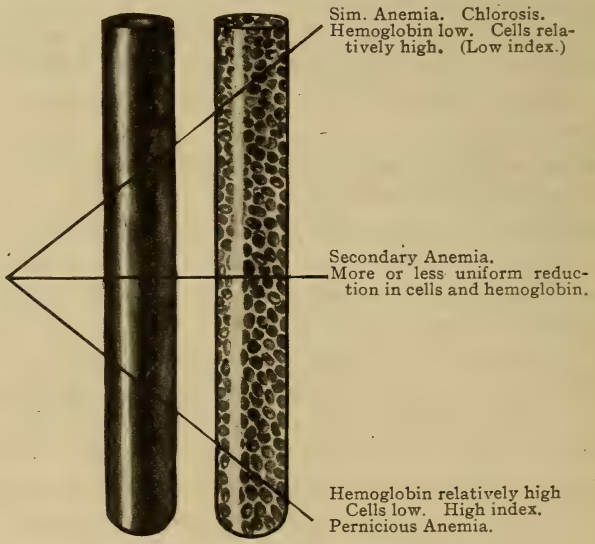

Sim. Anemia. Chlorosis.
Hemoglobin low. Cells relatively high. (Low index.)
Secondary Anemia.
More or less uniform reduction in cells and hemoglobin.
Hemoglobin relatively high
Cells low. High index.
Pernicious Anemia.

FIG. 52.

Hemoglobin-erythrocyte ratios.

Chlorosis is characterized by a predominant hemoglobin loss and a relatively slight diminution in the number of red cells present. That is to say, the individual cell is poor in hemoglobin.

Secondary anemia shows, as a rule, a more even reduction in both elements.

Pernicious anemia is the exact reverse of simple anemia in that the cell decrease predominates and, therefore, the hemoglobin value of the individual cell is high.

GENERAL CONSIDERATIONS.—Color.—In the anemias pallor as affecting the skin and mucous membranes is the rule, but is subject to frequent exceptions. Misleading.

Persons may be pale, yet not anemic, or ruddy and profoundly anemic.

The author recalls many cases of advanced leukemia in which the color was most deceptive. The student and the indoor worker is often pallid, yet not anemic.

The outward signs of anemia should be sought in the face, the conjunctiva, the lips, the mucous membrane, the lobes of the ear, the finger nails, tongue and pharynx.

Wide Variations.—*In chlorosis* there is usually marked pallor and the skin is likely to show a greenish-yellow tint.

In pernicious anemia there is ordinarily very marked pallor and the skin is of a lemon-yellow tint.

In secondary anemia aside from pallor one meets with many variations in color.

In Bright's disease with anemia the skin may be pasty white; or show the peculiar sallowness, brown or fawn color, more or less characteristic of the advanced *chronic parenchymatous* or "mixed" type of the disease.

Interstitial nephritis, even when well established, often yields no such evidence, being found in men of normal complexion or in those who have the ruddy countenance of the high liver.

Syphilis.—Certain cases of congenital and tertiary syphilis with secondary anemia show a peculiar sallowness impossible to describe, but easily recognized by those who have been shown it.

Malignant disease, especially advanced carcinoma of the stomach, is sometimes associated with a somewhat characteristic earthy pallor and in some instances the anemia may be extreme and closely resemble that of the pernicious type.

In diseases of the mitral valve one frequently finds an anemia masked by high color due to chronic congestion.

Muscular Weakness.—In all pronounced anemias one meets with lassitude and muscular weakness, varying, as a rule, with the severity of the disease. It is least marked in mild anemias of the secondary type and most evident in pernicious anemia.

Temperature.—Three-fourths of all cases of pernicious anemia are accompanied by fever, usually of the intermittent type, with an evening exacerbation. It is by no means an infrequent symptom in *severe cases* of chlorosis* or secondary anemia.

Gastrointestinal Disturbances.—Symptoms referable to disturbed digestion, gastric or intestinal, are encountered chiefly in the chlorotic and per-

* It nevertheless should always suggest an active or smouldering tuberculosis in these latter cases and hence a thorough examination of the lungs.

nicious forms of anemia and in the leukemias. *Anorexia, bulimia, nausea, vomiting, constipation and diarrhea* are the common symptoms. Established and advanced *pernicious anemia* is usually associated with exhausting attacks of diarrhea and intercurrent vomiting. Paroxysmal abdominal pain occurs in about 50 per cent. of the cases.

Respiratory System.—*Exertion dyspnea* is more or less pronounced in all severe anemias, but particularly so in marked chlorosis and pernicious anemia.

Circulatory System.—*Palpitation, syncope and precordial oppression* are common. *Edema* of the extremeties is not infrequent in chlorosis and is invariably present in *advanced* leukemia and pernicious anemia. *The pulse is of low tension* (60 to 80 mm. systolic pressure is not uncommon in pernicious anemia); *a curious humming may be heard in the vessels of the neck, and various murmurs are audible over the heart and the great vessels.*

"*Bruit de diable.*"

Myocardial weakness is likely to be pronounced and the heart is often dilated or markedly dilatable if the anemia is decided.

Anemic Murmurs.—*The general characteristics of the murmurs associated with anemia are: (a) Their systolic time. (b) Softness of quality. (c) Predilection for the pulmonary area. (d) Absence of marked and sequential cardiac outline changes. (e) Tendency to disappear as anemia is improved by treatment.*

These murmurs may be heard over any of the valvular areas, but *are most frequent by far in the pulmonary and mitral regions.* A systolic murmur in the subclavian and carotid is a very common feature *in extreme instances.*

Pseudo-"Corrigan pulse."

In certain cases combining anemia and chronic congenital asthenia, and in hyperthyroidism, one may find outward symptoms suggesting aortic regurgitation, viz.: throbbing peripheral vessels, water-hammer pulse, and even a capillary pulse. This condition need not mislead one in the absence of a diastolic murmur* and modified aortic second sound.

Capillary pulse.

The flow of blood is remarkably accelerated in severe anemia and this fact together with the lowered specific gravity of the blood and the *weakened myocardium* would seem adequate causes for the associated murmurs.

Nervous System.—*The symptoms under this head are many and various, being chiefly those in varying degree common to all conditions characterized by impairment or perversion of the blood supply of the tissues.*

Insomnia, drowsiness, vertigo, mental dulness and apathy, extreme irritability, perverted sensation, neuralgia, psychasthenia, hysteria, headache, and even delirium may be encountered, though delirium is not often seen save in pernicious anemia or the last stage of leukemia.

Misleading headache.

Headache may be found in all degrees of severity in simple cases of chlorosis, and one form is peculiarly interesting by reason of its close resemblance to the headache of meningitis.† Such headaches are so severe as to require most

* The author has reported one (terminal stage) case of pernicious anemia in which all these symptoms, together with a loud *diastolic aortic bruit* were present without aortic valve changes adequate to explain the murmur, and a few other such clinical curiosities are to be found in the literature.

† Two cases have come under the author's notice in which a mistaken diagnosis of cerebral meningitis had resulted from this curious condition.

radical measures for their relief, and often tend to recur until the general therapeutic indication, namely, the administration of iron, is fulfilled.

General Nutrition.—*In simple anemia the fat is usually well preserved, giving the patient a plump appearance.* Varies with type of anemia.

In secondary anemia the state of nutrition is dependent upon the primary lesion.

In pernicious anemia, even in its later stage, there is often a wholly misleading appearance of fairly good nutrition, due to the presence of overlying fat, but the skin in these cases has a peculiar velvety feel, and the muscles are sometimes of the consistency of jelly to the touch.

The Eye.—Muscular insufficiencies are common, *retinal hemorrhage frequently occurs in pernicious anemia* and in all severe forms the ophthalmoscope shows a pale fundus. Pale disc.

GENERAL ETIOLOGIC FACTORS.—Age.—*Secondary and pernicious anemias* occur for the most part in those at or above middle age. *Chlorosis* is essentially a disease of the earlier years, most often commencing at or about the age of puberty.

Sex.—Ninety per cent. of all cases of *chlorosis* (simple anemia) occur in young women.

Pernicious anemia is more frequent in the *male*, *secondary anemia* follows the laws of incidence of the primary lesion.

Specific Causes of Anemia.—Little is positively known of the specific pathogenesis of the anemias and the scope and purpose of this volume forbids a discussion of the theories.

General Causes.—In connection with simple anemia one is likely to find defects in food, environment, habits and general sanitation. Boarding-school girls and working girls may suffer alike from insufficient or improperly prepared food or from lack of variety, no less than from poor ventilation, overwork, or lack of fresh air or sunshine. Depressing emotions, such as grief, homesickness or disappointment in love often play their part, and the effect of nearly all of these general factors is shown in Arctic explorers who have in many instances suffered from severe forms of anemia during the Arctic night, when nostalgia, darkness, fatigue, poor ventilation and improper diet all were present. Bad hygiene. Lovesickness and nostalgia.

Secondary Anemia.—This may be associated with the following conditions: (*a*) *Repeated hemorrhages* (hemorrhoids, nose-bleed, uterine hemorrhage, gastric ulcer, hepatic cirrhosis). (*b*) *Malignant* disease, especially carcinoma of the stomach. (*c*) *Syphilis*, leprosy and tuberculosis. (*d*) *Mineral poisoning*, arsenic and lead especially. (*e*) *Malaria.* (*f*) *Bright's disease* in its various forms. (*g*) *Intestinal parasites.* (*h*) *Auto-intoxication.* (*i*) *Any acute infectious disease*, acute rheumatism being the chief.

The foregoing statements as to general symptomatology are applicable to all forms of anemia in varying degree. Considered as arbitrary types, chlorosis stands at one pole, pernicious anemia at the opposite, while secondary anemias of exceptional severity are ordinarily overshadowed and dominated by the symptoms of the causal lesion and may assume varying and at times misleading forms.

A Cause of Chief Importance.—*Many cases of chlorotic and secondary anemia lacking any easily demonstrable primary cause results from hidden foci of chronic intermittent or remittent infection such as are fully discussed elsewhere in this volume.*

CHLOROSIS

Characteristic Feature.—Hemoglobin deficit first reported by Johann Duncan in 1867 (Osler).

Cause unknown.

Etiology.—Although known and recognized before the days of Hippocrates, the specific etiology is unknown. Its onset is associated with the period of sexual development in the female and it would seem that some fault of function in the glands of internal secretion is the chief factor in causation. Its incidence in our own country has decreased strikingly during the past twenty years—a period characterized by greatly improved living conditions, a better understanding of concealed septic foci, and the free exhibition of Blaud's mass in the department stores.

Lessened incidence.

Developmental period.

Age and Sex.—It occurs rarely in young males but is almost exclusively limited to the female sex. Of primary attacks, 99 per cent. occur between the ages of 14 and 25, and chiefly between the 14th and 18th years. Obviously, this latter period corresponds suggestively to that immediately preceding, accompanying, or following the establishment of menstruation. It represents also the age of maximal susceptibility to focal infections. Inasmuch as the disease shows a peculiar tendency to chronicity and recurrence under the usual casual and unsystematic management on the part of patient and physician alike, one encounters frequently cases of chronic or recurrent anemia of the chlorotic type at later ages and occasionally even in middle age.

Recurrent cases.

The Congenital Asthenic.—According to the author's experience nearly all of the cases of pronounced chlorosis occur in individuals of the congenitally asthenic (universally visceroptotic) type. The characteristics of this peculiar type of constitutional inadequacy or inferiority are such as would favor or predispose to the development of chlorosis, whether this be considered as fundamentally the result of disturbance of endocrine function, nutritional deficit, or the result of infections.

Type chiefly affected.

Peculiar vulnerability.

In such individuals there is a peculiar nutritional instability and an unfortunate dependence upon nutritional reserve for the maintenance of health and normal function. The larger number of cases of endocrine disturbance, of gastric neurosis, of cardiovascular abnormalities of the functional type, are associated with the presence of the "drop heart," abdominal visceroptosis, flabby abdominal muscles, a narrow costal angle and the slender bony and muscular structure characteristic of this type. They may be fat or lean.

Leading characteristics.

In these congenital asthenics, particularly, one finds with especial frequency not only focal infections of the usual type but radiographic evidence of larval or obsolete tuberculosis of pulmonary site. It has long been known that this is true also of cases of chlorosis.

The readiness with which disturbance of function occurs in connection

with this constitutional state makes it easy to understand that deficient bone-marrow activity might occur readily in connection with it and indeed both the symptomatology and the etiologic indications accord with the assumption that in the greater number of instances one is dealing with this peculiarly vulnerable and unstable type. Bone-marrow deficiency.

Older Theories of Causation.—Among the numerous and diverse theories which have been advanced in the past, one may note the following:

(*a*) Love-sickness is given as a cause. Of course, this may be and often is encountered in a disease affecting almost exclusively young girls and young women of the age which peculiarly predisposes to this state of mind and body. Dubious etiologic factors.

(*b*) Hypoplasia of the generative organs has also been advanced as a cause and is present no doubt in a certain proportion of the chlorotic cases. However, it would be improper in the light of present knowledge to consider this in itself a direct cause or a constant associated etiologic factor.

(*c*) Tuberculosis, cardiovascular defects, heredity, psychoneurasthenic states, constipation and autointoxication, for the most part represent effects rather than causes and defects peculiarly likely to be encountered in connection with the congenitally asthenic type.

(*d*) Over-work, bad food, unhygienic environment, worry and mental depression, and even certain specific occupations have been advanced as casuative factors. Doubtless each and every one may be considered a potential contributory element, but certainly no one of them can be put down as a specific factor in etiology. Potential contributory factors.

(*e*) Much stress has been laid by certain writers upon the potency of homesickness as a factor in chlorosis and it is quite true that one encounters a large number of chlorotics amongst the recently arrived immigrants, especially such as come from northern countries as those of Scandinavia. Usually, however, it is unlikely that homesickness plays a part more important in itself than any other state of mental depression and worry; and furthermore, in the class of individuals just mentioned, many other factors favorable to the development of the chlorotic state are present. The larger number of these young female immigrants have come from the farms, from an open country life with coarse wholesome food and abundance of out-door work, and have exchanged these favorable conditions of environment for city living, domestic service, work in public laundries, and other occupations of a relatively unfavorable nature. Nostalgia.

(*f*) There can be little doubt that chronic concealed septic foci may play a part in the production and more especially in the persistance and recurrence of chlorosis, yet the greater number of anemias for which chronic infections of the tonsils, teeth, and other organs are responsible are of the secondary type to be described later and not of the chlorotic type. Focal infections.

Unfavorable Environment.—The betterment of conditions of living and working has undoubtedly resulted in a marked diminution in the incidence of cases of chlorosis as might be expected when one considers the large number of contributory factors in etiology involved in bad housing, whether related

to dwellings or shops, insufficient and improperly prepared food, excessively long working hours with resulting tendency to extreme fatigue, and deprivation of fresh air and sunshine for the indoor dweller or worker. Each and every one of these factors is of vital importance to health in general and especially to the maintenance of good health on the part of those of the congenitally asthenic type of constitution.

Cardinal Symptoms.—The most striking symptoms are lassitude, breathlessness, the characteristic color, and a blood picture characterized by a low color index and the predominance of the small erythrocyte.

Color.—Simple pallor or less frequently a peculiar "greenish-yellow" skin are usually, though not always, prominent signs. Indeed the term "chlorosis," "green sickness," is a misnomer.

Occupation.

The patients appear, as a rule, relatively plump and well nourished, though pale, and their occupation is likely to be that of laundress, shopgirl, factory worker or school girl. *The developmental period is the one of commonest incidence.*

The heart is often dilated moderately and edema of the ankles is a common symptom in severe cases. Vasomotor instability is marked and flushing may be associated with cold extremities, numbness and other symptoms of a like sort. A great many of the severe cases show the "drop heart" fully described elsewhere. *Nearly all chlorotics are congenital asthenics.*

The lungs are sluggish and the breathing is superficial.

Systolic murmurs are commonly encountered over the left base, at the apex or even in the aortic area. Both cardiac murmurs and dilatation respond promptly to effective treatment.

A venous hum ("*bruit de diable*") is often heard over the jugular and a similar sound may at times be heard at the occiput or over the eyeball.

Preservation of fat is the rule, high color not uncommon ("chlorosis rubra"), puffiness of the face may be decided and the eyes may be peculiarly brilliant and show a bluish sclera.

The general symptoms are those described in the preceding pages but are usually moderate in degree.

The therapeutic test for chlorosis is found in the prompt response of its symptoms to the proper administration of iron and relapses are common if patients are not kept under observation for long periods.

Low color index.

Blood Findings.—*The blood macroscopically appears pale and thin and the color index is low. The* hemoglobin *percentage varies widely in the different cases* (12 to 75 *per cent.*), *its average being about* 45 *per cent.* Total blood is increased and a certain grade of hydremia is present.

Erythrocytes.

The red cells are not proportionately decreased, the average being 4,000,000 *per cubic millimeter. A count under* 2,000,000 *is rare, and the average size of the cell is less than normal. Stained specimens show marked achromia and old cases may show polychromasia and marked variation in the size of the cells* (*anisocytosis*). *Marked cell deformity* (*poikilocytosis*), *is rare but in some recurrent cases extreme.*

Achromia marked.

"Blasts."

Nucleated red cells are seldom found and when present are normoblasts.

The leucocytes may be normal, slightly or markedly decreased or increased and relative lymphocytosis is not uncommon. Myelocytes* are rarely found, the eosinophiles are decreased and *the "coagulation time"* is shortened. Blood platelets are slightly markedly increased. Coagulation time.

THE ADDISONIAN PERNICIOUS ANEMIA.

(*"Plastic Pernicious Anemia"*)

Age.—It occurs usually in middle age and is excessively rare in childhood and in old age.

Sex.—In the United States and England males are oftenest attacked. In Germany females are chiefly affected. The world owes to Addison of England its first knowledge of this remarkable and mysterious disease.†

Etiology.—*The actual causes of pernicious anemia are unknown, but its dominant and invariable characteristic is an excessive, intractable, and ultimately fatal hemolysis due doubtless to an unknown toxin.* A mystery.

The blood picture is suggestively like that of the bothriocephalus and pyridin anemias in which known hemolysins are causative. It follows, oftentimes, shock, mental strain, prolonged exposure and fatigue, syphilis, malaria, alcoholism, and in women repeated and excessive childbearing, but any or all of these factors seem to be only contributory.

The present view would seem to be that certain toxins acting in small but potent doses over long periods abnormally stimulate the hemolytic activities of the bone marrow, spleen and lymph glands.

As a result of the continued overstimulation and the persistent hemolysis the erythrogenetic centers themselves become overactive and enlarged and cause that ultimate dominance of the large immature vulnerable erythrocytes and erythroblasts which dominate the blood picture of the disease.

Grawitz lays especial stress upon the well-known shortage of free HCl in the stomach contents of these cases as favoring excessive albuminous putrefaction and diminishing the defenses against bacterial activity.

It would seem possible that the ultimate cause may be found in the selective activity of peculiarly virulent hemolytic streptococci.

The disease is peculiarly insidious in onset and in public clinics the great majority of cases show an erythrocyte count under 2,000,000 at the first examination. Insidious onset.

Symptoms.—These are as described under "General Symptomatology" of the anemias, but patients present as special features or striking signs a more or less characteristic *lemon-yellow color*, together with insidious and slowly developed failure of strength, attaining gradually a most extreme type of muscular weakness in the established case.

* Osler reports a series with an average white count of 8,467 per cu. mm., and Gulland and Goodall a series with average under 5000.

† Addison's first report appeared in the London Med. Gazette, March, 1849. Biermer wrote of the same ailment in 1868, and in Germany it is termed quite wrongly *"die Biermersche anaemie."*

Striking symptoms.

Impaired nutrition, intermittent gastrointestinal disturbance often of great severity and persistence, vertigo, submucous and subcutaneous hemorrhages, extreme pallor of mucous membranes, retinal hemorrhage, severe headaches, tinnitus aurium, dyspnea, optical disturbances, fainting attacks, and slight edema of the extremities are among the prominent manifestations of this obscure ailment, as usually encountered.

The asthenia is strikingly progressive in untreated cases, yet temporary recovery is common and may endure for several years, though the ultimate prognosis is fatal.

Heart.—The heart is weak and dilatable, the pulse rapid and labile in action and anemic murmurs are pronounced.

Fever of the hectic type is a feature of the advanced cases or of the terminal stage.

Hemorrhages in the spinal cord and meninges are not uncommon and misleading pictures may be produced through "patchy" involvement of the posterior or lateral columns singly or combined.

Macroscopic

Blood Findings.—The blood is pale, thin, *slips* quickly off the ear or finger following puncture, and if dropped on a piece of white bibulous paper the red is surrounded by a ring at first almost colorless but later showing a faint gray tint.

"Coagulation time."

The "coagulation time" is increased and bleeding from the puncture of the ear may continue sluggishly for a considerable period, but is seldom or never excessive.

The "total blood volume" is diminished in these cases, the "volume index" is high, the specific gravity decreased. Blood plates and fibrin alike are strikingly diminished.

High color index, and low erythrocyte count.

The color index is high, the red cells are reduced on the average to 1,500,000 per cubic millimeter and 1,000,000 is a common finding.

Nageli has reported a count of 138,000 and Zeigler one of 110,000 per cubic millimeter in a case of the "aplastic" type, described further on as a separate form. The lowest observed by the author was 348,000.

Hemoglobin.

The hemoglobin usually is reduced greatly and may be as low as, or even below, 15 per cent., but in most instances is *relatively* in excess.

Poikilocytosis.

The average diameter of the red cell usually is increased, poikilocytosis (cell deformity) is marked and the "pear-shaped" and "anvil" forms are peculiarly plentiful.

Anisocytosis.

The red cells show great variation in size (**anisocytosis**) and giant non-nucleated and *nucleated red cells* are invariably present, though often found only after careful search and only rarely in great numbers, save in the so-called "blood crises."

Predominance of megaloblasts.

In this so-called "plastic" form, the megaloblasts must be found to outnumber he normoblasts or a positive diagnosis is not then justified. A relative excess of microblasts or of normoblasts usually indicates a severe secondary anemia.

Polychromasia.

Polychromatophilia, i.e., irregularity in staining and consequent lack of uniform color in certain of the stained cells in a blood smear is a marked characteristic of this disease though not peculiar to it.

*The leucocytes are diminished in three-fourths of all cases and this leucopenia may be extreme, the average being less than one-half the normal count.** Leucopenia.

Myelocytes and erythrocytes showing basophile granules with Wright's stain are nearly always present, though in small numbers. Myelocytes and basophiles.

It is usually possible to make an accurate diagnosis of pernicious anemia by the blood findings alone, but now and then cases occur in which the clinical history and physical signs must be invoked.

Bothriocephalus infection and nitro-benzol poisoning present a blood picture almost identical with pernicious anemia or actually indistinguishable from it. Its simulators.

Advanced cases of malignant disease of the stomach may present quite as extreme an anemia, but in this the small cell type usually predominates, both as to nucleated and non-nucleated red cells, the polymorphonuclear leucocytes are normal in number or increased and the lymphocytes relatively or actually diminished in number. A confusing secondary form.

The urine in advanced pernicious anemia is usually dark and shows a considerable increase of urobilin. Urobilin increase.

The frequency with which pyorrhea alveolaris, dental abscesses and the chronic tonsillar and sinus infections occur in certain intractable anemias may prove ultimately of the first importance in establishing the identity of the specific causative agent. Focal infections.

Autopsy Findings in Addisonian Pernicious Anemia.—The autopsy findings throw little light upon the cause of the disease but disclose certain striking pathologic changes more or less peculiar to it.

Fatty degeneration is the dominant process in the parenchymatous viscera and is especially marked in the liver, heart and kidneys. The heart is usually dilated and flabby and contains little or no blood. Its color is a pale yellow.

The red bone marrow is strikingly deepened in color and the yellow marrow of the long bones appears to be transformed into a deep red gelatinous substance often likened to currant jelly. This appearance is usually more striking than that seen in even the severest forms of secondary anemia.

An extraordinarily large deposit of iron in the liver and to a lesser degree in the spleen and kidneys attends the continued destruction of red cells.

A section of the liver thoroughly treated with a weak solution of ferrocyanide of iron and then washed in a weak aqueous solution of hydrochloric acid assumes a strikingly beautiful blue color.

The entire general picture presented by the victim on post-mortem section is one of extreme anemia and deficient blood volume. Even the imperfectly formed blood clots show a pallid red.

* This leucopenia usually affects chiefly the polymorphonuclears both neutrophile and eosinophile and to a lesser degree the large lymphocytes. A *relative* lymphocyte increase is, therefore, usually present.

APLASTIC PERNICIOUS ANEMIA

Sex predominance.

Definition.—*This term is applied to a relatively uncommon pernicious form of anemia, occurring usually, but by no means exclusively, in females between the ages of twenty and forty, in which the blood findings suggest a failure of hyperplastic response on the part of the bone marrow.*

Cause unknown.

Etiology.—The cause of the disease is wholly unknown. It is held, without much substantial justification, that in some instances it represents a terminal transformation of the hyperplastic Addisonian anemia. It may be treated properly as a distinct type, however, for bone-marrow insufficiency and aplasia are the dominating features in its pathogenesis.

Progressive and resistant.

The disease is usually progressively fatal, lacks the clinical signs of excessive hemolysis, is resistant to all therapeutic measures, and is distinctly more hemorrhagic in type than the ordinary form.

Low erythrocyte count.

Blood.—*The erythrocyte count* is seldom much above 2,000,000 and one count as low as 110,000 is reported.

Low or normal index.

The color index *is usually low or about normal. All forms of nucleated reds are absent or extremely scant. Poikilocytosis and anisocytosis are slight or wholly absent and polychromatophilia is not marked. Leucopenia and relative lymphocytosis are present as in true pernicious anemia but of higher grade. The blood platelets are decreased markedly.*

Absence of poikilocytosis.

The skin seldom shows the pale lemon tint so common in advanced Addisonian anemia. *The urine* is relatively light in color and free from urobilin. *The liver, spleen and lymphatic glands* are not markedly enlarged and even though nucleated cells are originally present they tend to disappear as the disease advances.

Autopsy reveals an entire absence of hyperplastic changes in the bone marrow and a greater or less degree of actual aplasia, the bone marrow in some instances consisting of yellowish fat.

SPLENIC ANEMIA

Not a Clinical Entity.—This condition has not yet made itself a place in medicine as a clinical entity and its features do not seem to be sufficiently characteristic to constitute a separate disease but rather to stimulate us to seek a more accurate terminology.

Only a syndrome.

Anemia with splenic enlargement would seem to be the more correct term. So far as the actual blood picture is concerned, any form of anemia may be simulated, the marked secondary type being that commonly shown.

We must regard the condition for the present as an interesting symptom-complex, lacking the glandular enlargement of Hodgkin's disease or the peculiar blood changes of leukemia, and adding to the ordinary symptoms of anemia that of splenic hypertrophy, commonly associated with hepatic enlargement. A large number of cases of this general description prove to be due to syphilis or to malignant growth.

Three syndromes under this general heading are worthy of discussion.

SPLENOMEGALY WITH HEPATIC CIRRHOSIS AND ANEMIA.—*This symptom-complex unites anemia, hepatic cirrhosis and splenomegaly.*

This disease was long ago known by the reasonably accurate and descriptive name "splenomegaly with hepatic cirrhosis." We owe to Banti an excellent and orderly description of the condition.

So-called "Banti's disease."

First Stage.—At any age, but most frequently between thirty and fifty a painless splenic tumor develops and gradually increases in size, until in form, area, and consistence it may closely resemble the spleen of a myelogenous leukemia. Coincidently there develops an anemia of the simple chlorotic or mild secondary type with low "*color index*" and more or less enlargement and induration of the liver.

Primary stage.

Splenomegaly.

Second Stage.—*Icterus* appears and dyspeptic symptoms may become prominent.

"Transition stage."

Third Stage.—After a few months the icterus and anemia become intensified but show no decided departure from the picture of severe chlorosis or of a secondary anemia of the ordinary type; epistaxis, hematemesis, purpura or other hemorrhages may appear, cachexia is marked and, later, *ascites* is evident. A fatal issue may result from progressive inanition or from gastric or intestinal hemorrhage.

"Ascitic stage."

Nearly all of the cases observed by the author, whether at home or abroad, have proven ultimately to be either syphilitic or due to malignant growth, and those proven luetic have presented, for the greater part, the typical picture as drawn by Banti.

SPLENOMEGALY OF THE GAUCHER TYPE.—*This extremely chronic and excessively rare disease of infancy and early childhood combines in itself the peculiar pigmentation of Addison's disease with splenomegaly, subsequent enlargement of the liver, and anemia.*

Peculiar pigmentation.

It is familial but probably not hereditary, occurring usually in several members of the same generation (females are chiefly affected), and is characterized anatomically by the presence of masses of peculiar hyaline cells of large size and probable endothelial or reticular origin, (40μ) in the spleen, liver, bone marrow and lymph glands.

Familial.

The blood picture is neither characteristic nor consistent. It may be that of mild chlorotic anemia (the usual form) with leucopenia, or present the blood findings of mild secondary anemia or those of a severe but atypical pernicious form with megaloblasts and myelocytes.

A tendency to hemorrhages may be present.

By some authorities it is stated that leucopenia is constant throughout and a chlorotic blood picture invariably present.

Bastard blood picture.

The course of the disease is extremely protracted, the general health is but very gradually affected and death occurs after several or many years through some intercurrent disease.

Though a disease of childhood and early youth, it often escapes diagnosis for many years.

CONGENITAL HEMOLYTIC JAUNDICE.—This is an extremely chronic, strikingly hereditary, familial disease, of great interest and rarity,

Familial.

characterized by extraordinary vulnerability of the red blood cells, decided anemia, splenomegaly, moderate hepatic enlargement, pronounced icterus and *urobilinurea*. All symptoms of an actual obstructive jaundice are lacking in the stools and urine.

Hemolysis.

The destruction of red corpuscles is extraordinary in many cases, the blood picture being in the rarer instances that of a pernicious anemia. Their resistance to hypotonic sodium chloride solution is so greatly diminished as to constitute an important factor in diagnosis.

An interesting case was reported recently from the author's former clinic by Richards and Johnston.*

LEUKEMIA (*"White Blood"*)

Historic Note.—Until 1841 this disease was regarded as a purulent inflammation of the blood. John Hughes Bennett gave the first account of the disease in the year 1845.

Pathogenesis.—The latest investigations strengthen the theory that we shall find ultimately that an infection plays the chief rôle. Leukemia is undoubtedly a primary affection of the bone marrow and the lymphoid structures, the essential change being one of excessive and wholly unregulated myelocytic or lymphocytic hyperplasia, leading *ultimately* to the dominance in the blood picture of types of white cells which normally are wholly absent or appear only in small numbers in the blood stream. We know practically nothing of the *early* stages of the disease.

Varieties.—*There are two forms of leukemia, the* **myeloid** *and the* **lymphatic.** *In the first form the blood assumes the so-called myeloid type, in the second, the lymphoid.*

Although several cases of acute myelogenous leukemia have been reported, we may for the present regard it as essentially a chronic disease.

Lymphatic leukemia, *on the other hand, may be either acute, subacute or chronic. Transmutation may occur in rare instances.*

Important Distinction.—*It should be clearly understood that the existence or non-existence of leukemia is not determined merely by the* **number** *of white cells present in the given blood.*

A count of 90,000 leucocytes may be but a leucocytosis and a count of 25,000 might represent a leukemia. The difference between the two is distinctly qualitative, not quantitative. It is true nevertheless that in most cases of leukemia the leucocyte count is very high at the time of first recognition.

* Cases showing an apparently clearly defined blood picture of pernicious anemia, but showing decided splenic enlargement and slight jaundice, should always be specially investigated as to familial tendency and the osmotic resistance of the red cells should be tested.

MYELOID LEUKEMIA

Definition.—*A chronic, progressive and fatal disease characterized by a combination of profound anemia of the pernicious type with an extraordinary increase of leucocytes of both the common and the unusual types, which findings, together with splenomegaly, dominate the clinical picture.*

Etiology.—*Age.*—It may occur at any period, but for the most part is encountered in middle age.

Sex.—Males are chiefly affected, predominantly so, in the author's experience.

Other etiologic factors are not well understood. Certain rare cases of streptococcic sepsis have presented a picture suggestively similar but there remains no definite etiologic factor up to the present time.

SYMPTOMS.—The general symptoms are those of severe anemia as already described at the beginning of this section. The disease is not likely to be detected until well advanced,* and the general symptoms are then those of a profound anemia with an especial tendency to hemorrhage and serous effusions.

Insidious in development.

Dyspnea is marked; ultimate loss of flesh usually extreme, and both liver and spleen are enlarged, the latter to an enormous degree in certain cases, and *always* to a marked degree.† It is astonishing that so great an enlargement frequently may be unattended by serious discomfort.

Enormous spleens.

In certain cases there is pain and tenderness over the long bones and disorders of sight and hearing are not uncommon.

Blood Findings.—*The red cell count is markedly reduced and the white cells are greatly increased in number, the average count being* 400,000 *in established and well-advanced cases, the maximum sometimes exceeding* 1,000,000 *cells. The characteristically important white cell of leukemia is the* **myelocyte** *and from* 20 to 50 per cent. of the leucocytes present are of the myelocytic type.

Striking picture.

Characteristic myelocytic dominance.

The eosinophiles are also present in unusual numbers, showing both an actual and relative increase. *Both the lymphocytes and polymorphonuclear cells*, though absolutely increased, are relatively diminished.

Megaloblasts, normoblasts and microblasts are present in quantity and their nuclei frequently show mitotic changes.

One of the most striking though not invariable symptoms of the later stages of the disease is the tendency to hemorrhage from mucous membranes.

Hemorrhagic tendency.

These are often large, persistently recurrent, and demand that every patient should be duly warned and instructed concerning measures for their relief or control. They often prove a terminal event, or death may result from asthenia or from either gradual or sudden cardiac failure, often associated with general dropsy. *Severe recurrent unexplained hemorrhages demand an examination of the blood.*

* Not only may strength be well preserved over long periods, but the outward appearance of health as well. Even a gigantic spleen may be wholly unnoticed by the victim.

† In one case shown by the author to the Minnesota State Medical Society in 1903, the splenic margin actually rested against the anterior-superior spine of the right side.

LYMPHATIC LEUKEMIA

Lymphocytosis absolute.

Essential Features.—*The essential features of lymphatic leukemia are two in number*: (*a*) *Enlargement of the lymphatic glands.* (*b*) *An absolute monocytosis in which either the large or small cell type may predominate.*

The cause is unknown; chronic cases endure for years, but acute cases may *terminate* in a month or six weeks.

Lymphomata.

SYMPTOMS.—*Any or all groups of lymphatic glands may be enlarged. The tumors are, as a rule, separate and movable, free from active inflammatory changes and show little or no tendency to suppuration. They vary greatly in size but seldom exceed the dimensions of an egg. Usually all accessible glands are affected in some degree.*

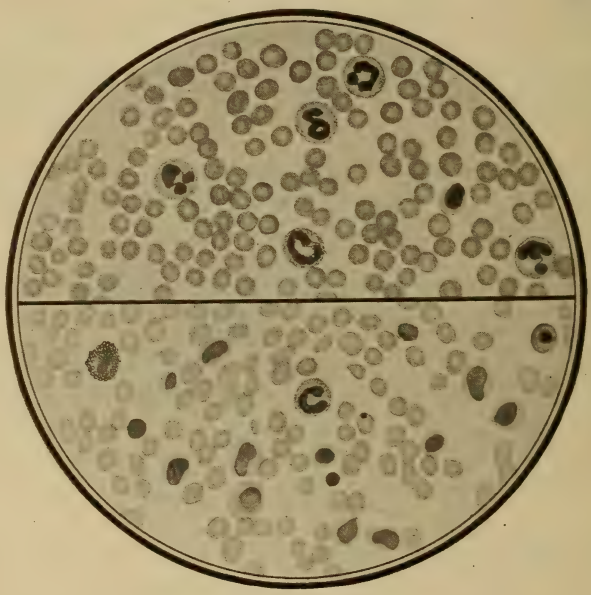


FIG. 53.—Lower section shows a lymphatic leukemia. Upper section normal blood showing leucocytosis. These types of cells should be carefully noted.

Predominant localization.

The cervical chains are ordinarily most prominent, but predominance of enlargement may be found in the axillary or inguinal groups and nodes normally inconspicuous may form large masses.

In the rarer cases the glandular chains affected are not accessible, yet decided involvement of the retroperitoneal or bronchial groups may exist.

Consistent picture.

The spleen is moderately or decidedly enlarged, but the glandular symptoms dominate the picture.

Fever is present in variable degree in all acute cases and the later stages of the chronic form.

A terrible complication.

Pressure symptoms may be marked and in three of the author's cases death occurred horribly from actual slow suffocation due to the pressure of mediastinal growths.

Insidious Onset.—In this form of leukemia, as in the myeloid form, the condition is usually well advanced before the patient seeks his physician, complaining of progressive weakness, easily induced fatigue and, perhaps, of glandular swellings.

Some pallor is usually manifest and these patients often carry a peculiarly harassed expression.

The spleen is usually easily palpable but seldom descends more than one or two finger-breadths below the costal margin.

The liver may be greatly enlarged and markedly indurated but usually the increase in size is moderate.

Fever.—A moderate daily rise of temperature is not uncommon even in the chronic cases.

BLOOD FINDINGS.—*The blood picture contrasts strikingly with that of myeloid leukemia in the comparative lack of diversity of form and type in the blood cells.*

Save in the rarest instances, the lymphocyte absolutely dominates the leucocytic field.

Nucleated red cells are rare as compared with the myelogenous leukemias, though in the acute type of the disease they may be present in considerable numbers and the same is true of certain of the leukemias of children. If nucleated reds are present, the normoblast predominates in most instances.

Very rarely an extreme degree of anemia proper co-exists with the leukemia and the blood picture more nearly approaches that of pernicious anemia so far as the erythrocytes are concerned.

The leucocyte increase, while striking, is seldom as large as in leukemia of the myelogenous type.

The average lies between 200,000 and 250,000 cells, the maximum being about 1,000,000, but in a case observed by the author the count reached 2,133,000 for the single examination possible and was probably a *preagonal increase*, as it occurred but twenty-four hours before death. As before stated, the remarkable predominance of the lymphocytic type (average 90 per cent.) is striking and the cells show many atypical forms. The myelocytes are relatively rare, eosinophiles are diminished and neither the mast cell nor the basophile are common.

A remarkable case.

ACUTE LYMPHATIC LEUKEMIA.—This rare ailment is sufficiently definite in its clinical characteristics to merit a special description.

As in the case of the chronic form, post-mortem section reveals general proliferation of lymphoid tissues of somewhat less extent and degree, as would be expected in a lymphatic leukemia of short duration.

Suggests An Acute Infection.—The onset is usually abrupt and well-defined, rarely insidious. A chill may or may not initiate the febrile stage. Very often an antecedent or existing severe stomatitis, *tonsillitis*, or pharyngitis occurs and may prove most misleading, though in the future we may find the fact helpful in determining the true etiology of the leukemic states.

Onset usually abrupt.

Suggestive coincidences.

A Misleading Occurrence.—When, as occasionally happens, there is no

change from the normal in the blood count for a period of several days error is very likely to occur.

Fever is persistent and may assume a distinctly septic type late in the course of the disease. Indeed a definite streptococcic infection may occur in certain cases. This has been regarded as secondary but recent work with the hemolytic streptococci would suggest that such a conclusion may be erroneous.

Hemorrhage from the nose or gums may initiate the attack and constitutes an important complication of the disease. It may be most persistent and intractable and even prove the terminal event. The condition of the mouth and teeth may closely simulate scurvy.

Enlargement of accessible glands may be wholly lacking or very decided.

Splenic enlargement is present but only of slight degree in cases of short duration and *symptoms of profound anemia rapidly develop with progressive cardiac weakness.*

The blood picture is identical with that of the chronic form, the small lymphocyte usually, but by no means always, predominating. Early counts may prove negative.

It is asserted that many of the cases termed acute lymphatic leukemia are actually instances of acute myeloid leukemia, the mononuclears representing Naegeli's myeloblasts.

The differentiation of these cells from the lymphocyte offers great difficulties, even to the expert hematologist, and is of slight clinical importance.

Prognosis.—The disease is invariably fatal and may terminate within a few days or be prolonged for two or three months.

DIFFERENTIAL DIAGNOSIS OF THE ANEMIAS.—*The enormous value of blood examinations is evident if one considers the insuperable difficulties surrounding the purely clinical differentiation of the conditions described, as compared with the ease with which each disease may be identified, usually, by hematologic methods.*

Depends upon blood-findings.

As regards anemia in any of its three chief forms, no confusion is possible save in rare instances, and the two chief varieties of chronic leukemia are quite as sharply contrasted.

The glandular enlargements of Hodgkin's disease, tuberculosis and syphilis seldom offer the slightest difficulty if the blood findings are invoked, nor can the various enlargements of the spleen be confused with the splenomegaly of myelogenous or lymphatic leukemia.

Sources of Confusion.—Certain cases of profound secondary anemia, particularly those associated with carcinoma of the stomach or *primary* atrophy of the gastric tubules (*not merely the simple achylia gastrica*) may at times present a picture almost indistinguishable from that of pernicious anemia though the small red cell, both nucleated and non-nucleated, is the predominant form in almost every instance. To this may be added bothriocephalus anemias, which symptomatically, and in the blood changes, may be identical with pernicious anemia proper, and separable only through

Relatively common.

the discovery of the true etiologic factor. *A careful examination of the stools is indicated in all anemias.*

It is probable that in the anemias of true gastric atrophy of the type mentioned, the blood findings are always those of the profound secondary type.

Subdivisions and Transition Forms.—It is quite possible that further subdivisions of leukemia may be made in the future, and indeed many cases of variation in the relative predominance of cell types and apparent transition are constantly being placed on record.*

Von Jaksch has reported a "multiple periostitis with splenomegaly and myelocytic anemia, multiple arthritis, profuse sweats *and a like and even greater tendency to the hemorrhages such as so frequently complicate the later stages of myelogenous leukemia."*

CHLOROMA.—*In this rare disease the blood findings may be indistinguishable from those of lymphatic leukemia, but the clinical symptoms are strikingly different save that its* **course** *closely parallels lymphatic leukemia of the acute type.*

"Green cancer."

Tumors.—The curious greenish tumors of chloroma are strikingly unlike anything occurring in connection with lymphatic leukemia.

At autopsy they are found widely disseminated throughout the body, but their tendency to appear at points of prominence on its surface makes the diagnosis simple *if the existence of such a rare disease is recalled to memory.* Indeed they are especially numerous over the bones of the skull and face.

This curious ailment is invariably fatal and attacks, almost exclusively, male children and adolescents.

STILL'S SYNDROME.—*Still has reported a syndrome, the main feature of which is an anemia associated with* **infantile arthritis** *with enlargement of the lymph glands and spleen, but lacking the blood findings of leukemia and presenting usually the history of rachitis.*

MYELOMA.—In this rare and obscure ailment a period of neuralgia-like bone pain may be followed by painful metastatic swellings usually affecting chiefly the ribs or bones of the cranium and sometimes producing spontaneous fractures.

Diagnosis depends upon the presence of the Bence-Jones proteid in the urine and the result of roentgenographic studies.

A useless term.

LEUKANEMIA.—This term has been widely and loosely used to cover a clinical picture in which the symptoms of leukemia and of pernicious anemia are confusingly blended.

It has been wrongly used also to cover cases which were undoubtedly myeloid leukemias or clearly pernicious anemias in which unimportant departures from the type had occurred.†

* The author has observed a case of apparently typical Hodgkin's disease become in all respects lymphatic leukemia and many reports of myeloid leukemias under X-ray treatment have shown an apparent transition to the lymphatic type.

† As Cabot says, "most reported cases are clearly leukemic and nearly all of the remainder are pernicious anemias."

If the term is to be retained it should be confined to cases of the type originally reported under it by Leube and Arneth. *The original case pursued the course and had all the attributes of a rapidly progressive and fatal acute infection.*

The blood picture was that of a pernicious anemia of the Addisonian type to which was added a large increase of leucocytes with many neutrophile myelocytes.

PROGNOSIS IN THE ANEMIAS.—In anemia of the chlorotic type *the prognosis is always good unless it be a part of the clinical picture of gastric ulcer or incipient tuberculosis, or attributable to recurrent septic infection arising from obscure foci.*

Secondary anemias are wholly dependent as to *cure* upon the possibility of removing the primary cause.

Pernicious anemia is almost invariably fatal, though long periods of apparent recovery may be noted in cases of the plastic type.*

Leukemia offers a bad prognosis, though of late apparent cures have been common. It is probable that these cases will also prove fatal after a period of apparent arrest.

THE POLYCYTHEMIAS

POLYCYTHEMIA.—(*Erythrocytosis*).—A more or less marked increase of erythrocytes above the normal count is observed in a great variety of conditions, some important, others trivial, and, curiously enough the permanency of such a condition still remains unproven.

Not uncommon.

It usually represents nothing more than blood concentration or stasis, and in high altitudes is perfectly accounted for by the assumption that nature meets an actual need associated with the rare air and the dyspnea first produced in a non-habituated person.† This assumption is borne out by the secondary partial reduction noticed in those who take up permanent residence.

High altitudes.

A similar slight increase may follow baths, hot or cold, massage, violent exercise and the administration of certain drugs or may be associated with the process of digestion, blood regeneration, vomiting, profuse sweating, the removal of exudates, profuse diarrheas and, it is said, with myxedema and acute yellow atrophy.

Multiple causes.

Its presence in connection with circulatory stasis, such as may occur in organic heart disease, emphysema, stenotic dyspnea and similar conditions affecting the circulation is readily understood and in extreme cases the count may reach 7,000,000 per cubic millimeter. In congenital heart disease

Stasis polycythemia.

* The longest duration of such a period of false cure personally observed was five years, a second period of about one year followed a second stay in the hospital, succeeded by a rapidly fatal relapse. One case of complete cure has occured in the author's practice. The blood being *wholly normal* ten years after apparent recovery.

† An immediate primary apparent increase such as occured in the military observers and fighters of the aviation corps during the "Great War" is caused probably by cardiovascular embarrassment and irregularities of distribution, violent and abrupt transitions from sea level to 14,000 ft. altitude being common events of the day's work.

of extreme cyanotic types the erythrocyte count may exceed 9,000,000 per cubic millimeter of blood and hyperplasia of the red marrow is found in such cases.

In the dyspneic states due to acquired heart disease there is probably no *excessive* formation of red cells, but rather an irregular distribution throughout the circulation.

Polycythemia rubra.

ERYTHROCYTOSIS MEGALOSPLENICA.—(*Erythremia*).—**This peculiar disease or syndrome of unknown etiology comprises:** (*a*) *Marked increase of the red cells.* (*b*) *A peculiar "red cyanosis."* (*c*) *Enlargement of the spleen.* (*d*) *Frequently, hepatic enlargement.* (*e*) *Commonly, albuminuria.* (*f*) *Hyperviscosity of the blood.* (*g*) *Rapid coagulation.* (*h*) *Great increase in the total blood volume.*

A recent discovery.

The disease was first noted by Vaquez in 1892, again reported by Saundby and Russell* and by McKeen† in 1901, and in 1903 Osler's report of cases and masterly description brought the condition into prominence.

It is rare, but not to the extent formerly assumed. The author has encountered four cases in his private consulting practice during the past few years.

Hemoglobin values of 165, and even 220 (by Palmer's method), have been reported, but in one of Cabot's cases it was but 85 with a count of 8,484,000 reds and 15,000 whites, and in another 105 with a count of 11,352,000 reds. Moderate or absent leucocytosis is the rule. The highest erythrocyte count reported is 14,800,000 per cubic millimeter.

The color index is low, the coagulation time variable, and the behavior of the erythrocytes to salt solution is normal.

The specific gravity of the blood is high; that of the serum, normal; and the freezing point shows no abnormal variation.

Sight diagnosis.

Symptoms.—*The diagnosis can be made at sight with reasonable accuracy by one who has carefully observed a typical case previously.*

Dusky red.

The face and ears exhibit a peculiar dusky *redness* seen in no other disease. *It is furthermore quite distinct from ordinary cyanosis, however intensive, in that the red dominates the color field as far as the face is concerned.*

The extremities are markedly cyanotic and its depth and degree is greatly intensified by exposure to cold and the dependent position.

Splenomegaly.

Decided enlargement of the spleen is present almost invariably and may attain large proportions. It is smooth, indurated and seldom or never fixed by adhesions.‡ All of the author's cases showed marked enlargement.

The liver is usually decidedly enlarged.

Arterial hypertension common.

Albumin and casts *have been present in four of the six cases occurring in the author's practice and in all the blood pressure was distinctly high. In all cardiac hypertrophy and dilatation were manifest.*

* Lancet, 1901, Vol. 1.

† Boston Medical and Surgical Journal, June 20, 1901.

‡ In a case hereafter referred to as under observation for eight years past the primarily gigantic spleen is greatly shrunken and the liver has assumed almost a normal outline. The last blood count showed over eight million erythrocytes.

Many cases are reported which have lacked circulatory disturbances of this nature; and, doubtless, a large number of the cases on record are unworthy of inclusion. It is certain, however, that the typical cyanosis with splenomegaly and erythrocytosis may exist without high blood pressure. *Internal hemorrhages and thromboses are common terminal events.*

Prognosis.—No well-authenticated cases of cure have been placed upon record. But one of the four cases of the author is living after eight years of observation and his improvement has been maintained only by repeated free phlebotomy and the use of benzol.*

In reporting cases the chief clinical factors must be demanded inasmuch as a polycythemia of marked degree may co-exist with cyanosis under other conditions. The peculiar *cyanosis* with its accompanying brick-red, mottled facial color is most striking.

CERTAIN OBSCURE DISEASES ASSOCIATED WITH ANEMIA

HODGKIN'S DISEASE.—(*"Pseudo-leukemia," "lymphomatosis granulomatosa," "lympho-sarcomatosis," "lymphadenosis," "anemia lymphatica," "adenie," "lymphadenie," "malignant granuloma," "infectious granuloma."*)

Definition.—*An infectious granulomatous process of uncertain etiology, chronic, progressive and fatal, chiefly affecting male adults under forty years of age; characterized by peculiar histologic changes in and marked enlargement of the lymph glands; associated with splenic enlargement and anemia of varying degree, but lacking the blood changes of lymphatic leukemia.*

The number of names given above indicate the dubious nature of the ailment and the diverse pathologic processes with which it has been confused.

Historic Note.—The disease was first reported by Malpighi in 1669, who did not recognize it as a distinct disease. Craigie, in 1828, differentiated the anatomical character of the glands from those of carcinoma and tuberculosis, but to Dr. Hodgkin (1832) we owe our first definite description of the disease as a clinical entity. It did not, however, attain general recognition until the sixth decade of the last century.

Bunting's micro-organism.

Etiology.—C. H. Bunting has recently isolated and cultivated a Gram-positive diphtheroid bacillus, *corynebacterium granulomatis maligni*, which, when grown in pure culture and injected into apes, produces an enlargement of glands and histologic changes similar to, if not identical with, those of Hodgkin's disease in man.

Fraenkel and Much have reported Gram-positive granular non-acid-fast bacilli, and some observers have considered these as degenerated tubercle bacilli, but on the whole, little evidence has been adduced to support the older supposition of Sternburg that the disease is of tuberculous origin.

The *etiology* therefore remains unknown.

Usually striking.

Glandular Enlargement.—Dorothy M. Reed reports the following histologic picture as characteristic of Hodgkin's disease: (*a*) Swollen endothelial

* No knowledge of the actual duration of this disease is available for the reason that none have been seen in the early stages and watched throughout the course of the disease.

cells lying upon the fibrils of the reticulum; (*b*) Large epithelioid giant cells lymphocytes and eosinophiles occupying interstices of the stroma. The essential clinical change is one of enlargement of the lymph glands involving primarily limited areas, but tending to extend widely. The cervical and in-

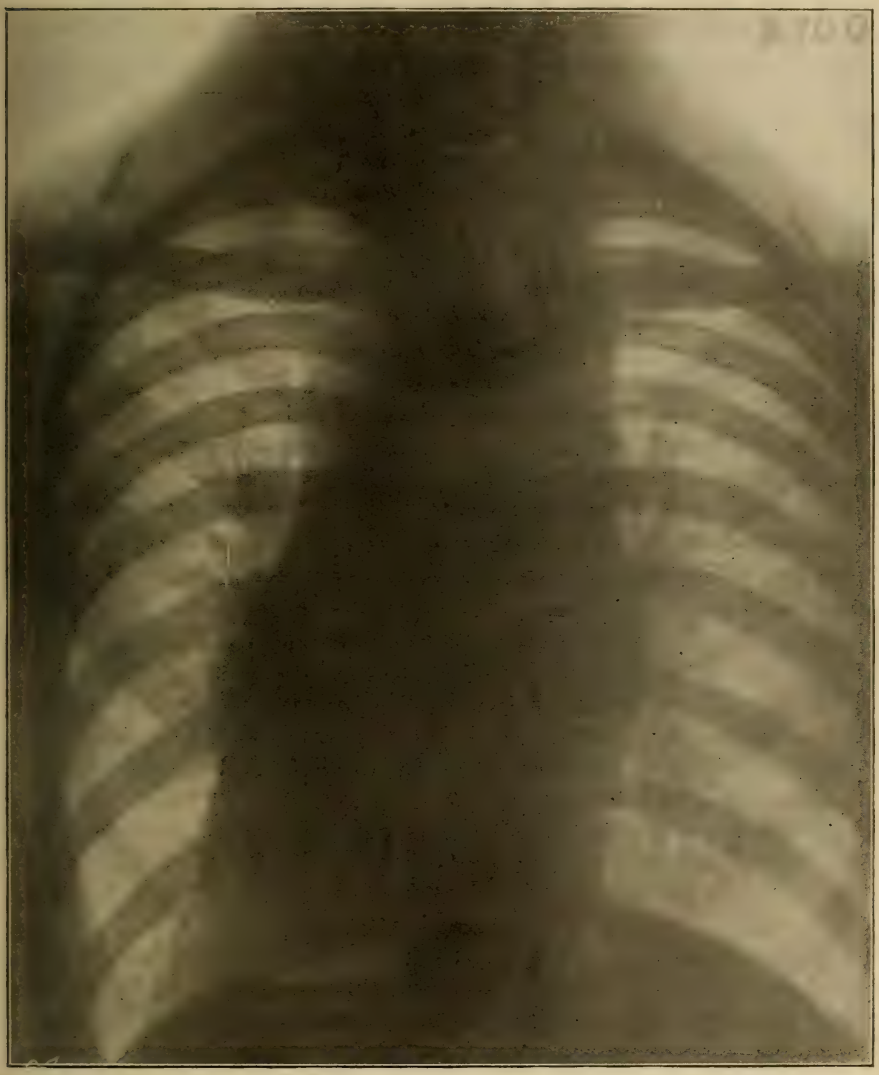


FIG. 54.—Hodgkin's disease, enlarged glands filling the mediastinum. (*Dr. Frank S. Bissell.*)

guinal glands are usually the first affected, the former giving to the disease its most marked characteristic. The tissues of the neck may entirely lose their normal contour, and the glands there and elsewhere tend to steadily enlarge without marked tenderness or symptoms of inflammatory change. They tend to maintain their individual outline until late in the disease,

and the skin usually remains unaffected and unattached to the subjacent tumors. When periadenitis occurs in the later stages lobulate tumors are formed which may lose the firmness characteristic of the earlier period, become softened and, rarely, may undergo ulceration. The deep glands above the trachea, those of the larger bronchi, of the mediastinum and retroperitoneal area become involved and in some instances form large tumors and greatly complicate the case. The spleen is enlarged in 75 per cent. of the cases. In some cases the internal lymphadenoid tissues alone are involved, rendering positive diagnosis extremely difficult.

A source of error.

The Blood.—There are no changes in the blood peculiar to this disease, the picture being one of secondary or of chlorotic anemia. *The blood index* is in fact usually low as in the chlorotic type and the number of red cells seldom falls far below 2,000,000 per cubic millimeter. In cases which develop marked mediastinal involvement and cyanosis a stasis-polycythemia may obscure the blood picture. In most cases a moderate or decided leucocyte increase is observed, especially during periods of increased fever when it may reach a high figure (50,000 or more).

A more or less decided eosinophilia is usually demonstrable.

Pseudopodia-like elements representing megalokaryocytic protoplasmic masses are also reported by Bunting as appearing in the blood.

In certain profoundly cachectic and asthenic cases the polymorphonuclears are decreased and a *relative* lymphocytosis results.

Debated point.

Whether an actual lymphocytosis is at all common is a contested point. Morawitz asserts its frequency and its value as a differential point in the separation of true Hodgkin's from the other granulomata. It is probable that such an increase is usually only apparent and is actual'y relative.

Important data.

Bunting believes that an increase in blood plates and transitional leucocytes constitutes a constant feature and it is true certainly that these features are encountered with sufficient regularity to make them diagnostically suggestive.

Acute Hodgkin's Disease.—Certain cases develop so rapidly as to be properly called acute, and may run their course to a fatal termination in a few weeks. The symptoms are, however, not different from those of the chronic type, save that there is a certain concentration of all clinical phenomena.

The Hodgkin's Collar.

Symptoms of the Ordinary Form.—*The onset* is insidious, save in acute cases, and the enlarged glands, usually in the cervical region, but sometimes in the groin and axilla, may first attract attention.

The glandular enlargement may primarily be either unilateral or bilateral, but extends and tends to become general. The changes in the neck and the peculiar outline produced have given rise to the descriptive term, "the Hodgkin's collar."

As the disease progresses, though often only after several months, a considerable or high degree of secondary anemia and more or less progressive emaciation appear.

If, as is not infrequent, the mediastinal glands take on a rapid growth, the pressure symptoms may be extreme and will precisely resemble those seen

in any other mediastinal tumors, pain, cough and dyspnea, paroxysmal or continuous, being the chief symptoms.

Fever is present in the advanced cases and shows a decided tendency to the relapsing form. Indeed its behaviour may constitute a point of differential value.

*Splenic enlargement is readily made out in three-fourths of all cases and occasionally becomes excessive. The liver is less frequently affected.**

Differential Diagnosis.—*From tuberculous adenitis* it is usually readily differentiated by the age of the patient (which is generally above thirty and almost invariably above twenty); by the absence of decided inflammatory symptoms in and around the glands during the earlier periods; by the absence of a history of tuberculosis past or present; by the excision, if necessary, of a small gland and its examination; together with the fact that tuberculosis is more generally localized and unilateral, and that its tumors never attain a large size without marked evidence of inflammation and caseation.

From leukemia it is absolutely differentiated in most instances by the blood examination.

Aleukemic lymphadenosis lacks the blood picture of Hodgkin's disease.

Syphilis must be differentiated by the history, the presence or absence of scars, the result of a Wassermann or luetin test, and the difference in the character and extent of the glandular swelling, which in this disease, though more or less general, does not often attain the large size found in Hodgkin's disease. Tentative antiluetic treatment is often advisable in clinically atypical cases.

Lymphosarcomatosis spreads rapidly to all other glands in the area affected but does not invade the other adjacent organs.

True sarcoma tends to spread to other tissues.

Simple lymphoma is localized and lacks the predominating symptoms of Hodgkin's disease.

In all the conditions named the blood picture described above is lacking.

It must be admitted that certain of the generalized granulomata of tuberculous or syphilitic origin cannot always be differentiated clinically from Hodgkin's disease.

Unusual Symptoms.—An enormous number of symptoms might be named in connection with the development of glandular masses in the abdomen, among these being bronzing which results probably from pressure upon the solar plexus. One has only to bear in mind that in this disease large tumors may develop in remote regions producing exactly the same effects as would any other tumors in the same location. Bronzing possible.

Prognosis.—The disease is almost invariably fatal, its course occupying usually one or two years. Misleading periods of improvement followed by relapse are extremely common and this applies to both the general and local manifestations of the disease. Progressively fatal.

* Marked splenic enlargement is due usually to secondary growths: that of the liver to the same cause or to fatty degeneration.

PURPURA

Definition.—A morbid state characterized in its graver forms by a low blood-platelet count, prolonged bleeding time, deficient contractility of the blood clot, usually without delayed coagulation time, by hemorrhages, subcutaneous, submucous, or, in rare instances, an outpouring of blood into the serous cavities, with or without fever and other constitutional symptoms.

Deficiency of blood platelets, and the consequent deficiency or lack of platelet thrombi, may account for the persistent or unduly prolonged bleeding noted, in the severer forms of purpura, following slight wounds.

The cause of the platelet deficiency is wholly unknown, nor can we determine whether in any given case the unknown etiologic factor operates to destroy them when formed, inhibit their production from the megacaryocytes or destroy the parent cell in the bone marrow.

The platelet count has a practical value in this ailment inasmuch as a count below 60,000 per cmm. predisposes to or initiates hemorrhage (Minot) and with a decided rise in the count the danger of hemorrhage disappears.

In severe purpura the platelets may almost wholly disappear (5000 or less per cmm. of blood).

Comment.—So little understood is this disease that an arbitrary classification serves the purpose of clinical convenience and little more. *It seems to bear a close resemblance to certain forms of urticaria, to scurvy and to hemophilia, and possesses but two constant and invariable symptoms, viz.: subcutaneous hemorrhages, and imperfect thrombus formation.*

Cases may be grouped under four general heads.

1. **Complicating purpuras,** *viz., those occurring in connection with (a) acute infections, (b) chronic diseases associated with cachexia.*

Significance

When purpura occurs in association with acute infection it ordinarily indicates an exceptionally severe type of the primary disease. One may encounter it in typhoid fever, smallpox, malignant endocarditis, septicemia, pyemia, whooping cough and measles as well as in the more virulent of the tropical diseases. In cerebro-spinal meningitis and typhus fever it represents the usual exanthem.

In the chronic diseases it is seen most frequently in connection with scurvy, pernicious anemia, idiopathic plastic anemia and leukemia, not infrequently in tuberculosis, Bright's disease, Hodgkin's disease, syphilis, and various cardiac lesions, particularly those associated with marked degeneration of the myocardium. In epilepsy it is not uncommon and sometimes points the way to a correct diagnosis in cases of an obscure nocturnal type. In such instances it is usually slight and transient, often showing only on the face as an indistinct capillary streaking which may last for a few hours or a few days.

"Hysterical" purpura.

In hysteria, the same form may be present, or the rarer forms, among which the most curious is the cruciform purpura in which, it is said, the hem-

orrhages appear at points corresponding to the wounds of the Saviour on the Cross. *It may be seen at once that in epilepsy and whooping cough mechanical strain plays a great part.* Cruciform purpura.

True purpura would seem to be a more or less direct expression of an infection and might better be considered wholly apart from the conditions mentioned in the paragraph preceding. True purpura.

2. Arthritic Purpuras.—In the three forms of purpura coming under this class there is a joint involvement distinctly rheumatic in type. Under this we have: (*a*) *Simple purpura.* (*b*) *Peliosis rheumatica* (Schönlein's disease). (*c*) *Henoch's purpura.* This classification need not be confusing if the following points are borne in mind. *In all, the essential lesion is hemorrhage; all are accompanied by fever and by joint lesions.* Differentiation possible.

Simple purpura *is of a mild type and short duration.*

Peliosis rheumatica *is much more severe, with higher fever, more marked joint involvement, sore throat usually at the onset and not only marked purpura, but frequently, pemphigoid spots, purpuric edema and marked urticarial manifestations of either the ordinary or giant variety. The distinctive feature, however, is the involvement of joints to a marked degree.*

Henoch's purpura *differs from the two already named, primarily, in the fact that gastrointestinal symptoms, such as nausea, vomiting, diarrhea, and hemorrhage from mucous membranes, are the predominating features, while the arthritis is usually slight. In all other respects it may closely resemble the other forms.*

3. Purpura Hemorrhagica (*Morbus Werlhofii*).—*This terrible disease may be rapidly fatal, and is characterized by severe and often intractable hemorrhage from any or all mucous membranes, associated with high fever.*

It will be seen from the foregoing description that though the basic symptom is the same in each and every variety, the differences presented are such as to permit of clinical differentiation in most instances.

One cannot but feel that an infection must lie at the root of purpura, but nothing of a specific nature has so far been discovered.

4. **Purpura Fulminans.**—In this form the disease runs a rapidly fatal course and may terminate without the appearance of joint lesions, those of the skin being the only striking indications of the nature of the ailment.

HEMOPHILIA

Definition.—*A morbid state, hereditary and characterized by an extraordinary tendency to spontaneous or induced hemorrhage, and an especial familial tendency to attack the young males and spare the females who, however, are the chief sources of its transmission to succeeding generations.*

Etiology.—The actual cause is unknown, but deficiency of fibrin ferment (thrombin) or of one of its necessary constituents (thrombokinase, or perhaps, prothrombin), is proven. A peculiar thinness and fragility of the vessel walls, a neuropathic tendency and abnormal alkalinity of the blood, together with many other, presumably baseless, assumptions, have been advanced as factors in causation.

Age and Sex.—It affects chiefly male children under ten years of age, first appearing in early childhood,* cases being rare in the later years of life, and usually less severe in their nature.

In those affected who live to maturity the tendency seems to diminish gradually.

The oft-repeated claims that women of hemophilic stock may themselves be bleeders seem to be unsupported by substantial evidence. The claim for occasional conduction through the male seems still less valid.

Heredity.—Although cases occur in which this element apparently is lacking, the disease furnishes one of the most interesting known examples of atavism in relation to disease. It affects male children through maternal heredity, the mother or the girls of a family seldom showing any hemophilic tendencies, yet passing it on to their male children. In one family in Switzerland the disease can be traced back over 300 years. It may even skip a generation or two only to return later on.

Atavism.

Symptoms.—*Intractable nose-bleed is most common, but hemorrhage may occur from the lips, gums, throat, stomach, urethra, lungs, bowels or, in the female, from the uterine mucous membrane, though, strangely enough, menstrual flow is not always excessive nor is labor extraordinarily perilous, in those rare instances of hemophilia in the female ordinarily showing extraordinary hemorrhage in response to slight wounds, abrasions or even pressure.*

Extraordinary features.

It is fortunate that the disease is relatively rare, as information upon the subject is sometimes withheld by members of these foredoomed families, and slight wounds or bruises, even the puncture of the ear or finger for blood examination, the drawing of a tooth, the application of a leech or a vaccination, may bring about a fatal hemorrhage.

Beware of "bleeders."

More extraordinary still is the fact that the slightest traumatism or even light and unconscious pressure or friction may excite what apparently may be spontaneous hemorrhage from mucous membranes or even the free skin (lobe of ear, tip of finger).

"Spontaneous" hemorrhage.

Coagulation Time.—This is greatly prolonged, a firm clot never forming under an hour if blood from a vein, unmixed with the tissue juices, is used. Blood from a mere skin wound or puncture gives unreliable and misleading results because of the unavoidable admixture of such tissue juices which contain cephalin.

Extraordinary delay.

The mortality is extremely heavy, and pseudo-rheumatic affections are common, as are ecchymoses and hematomata, these showing its close resemblance to purpura and scorbutus.

Prognosis.—A majority of male children of affected families die under ten years of age, though light cases occur in which, after the age of puberty, the tendency is largely or wholly lost.

* Infants are usually free from its manifestations even though they may be profoundly affected later. Nevertheless Nacke of Kirchheim, the physician of the famous Mampe family which has been under medical observation since 1827, states that he could tell from the hemorrhage following the cutting and *tying* of the cord whether or not that child would prove a "bleeder."

SCORBUTUS ("*Scurvy*")

Definition.—A morbid state peculiarly dependent in most instances upon the diet* (vitamin deficiency), characterized by profound weakness, anemia, apathy, and a marked tendency to sponginess of, hemorrhage from, and severe inflammation and ulceration of, the gums. An avitaminosis.

Etiology.—The actual cause is unknown, but the conditions of its development are well understood. They are: (1) *A diet from which fresh vegetables, fruits and meats are excluded.* (2) Bad air, overcrowding, overwork, deficient light. Known factors.

Its occasional presence in children even in our own country must not be forgotten.

Up to within the last century scurvy was the scourge of seamen and armies. During the Crimean war there were 23,000 cases in the French army alone. Military and naval expeditions had sometimes to be abandoned on account of outbreaks of the disease and East Indian ships frequently lost half of their crews in one voyage. In one of the early Arctic expeditions 50 per cent. of the members died of the disease. In a recent Antarctic expedition the effects of the factors mentioned above were clearly shown, though a better food supply prevented a general epidemic. Once a scourge.

A considerable number of cases occurred in the Russian troops during their war with Japan; it was present to some extent in the Allied Armies during the last great war, and exists at the present writing in famine stricken Russia and doubtless in some of the lesser Balkan States. Essentially, however, scurvy belongs to generations past.†

Symptoms.—*The leading symptoms are spongy, bleeding, and often ulcerating gums, purpuric symptoms, areas of brawny induration especially in the lower extremities, progressive circulatory weakness often with right heart dilatation, and subperiosteal hemorrhages especially in children.* Peculiar syndrome.

In children the swelling is commonly over the outer aspect of the femur; and in every marked case is hard, tense, and exquisitely tender (see "Barlow's Disease," following). Epiphyseal suppuration may occur and hemorrhages into the joints and retina are not uncommon. Retinal hemorrhages are not uncommon. Physical weakness is extreme and there is pronounced and progressive mental depression and usually edema of the ankles.

BARLOW'S DISEASE.—*Occurring in infants*, scurvy is known as *Barlow's disease*, having been described by Dr. Thomas Barlow in 1878.‡ Up to that time it had been regarded wrongly as "acute rachitis" and was so

* In an absolutely typical and remarkably complete adult case recently observed by the author absolutely no connection with diet could be traced nor could the man's condition be materially improved by the usual therapeutic measures.

It may have represented one of the rare examples of atypical purpura exactly simulating scorbutus. Many cases of purpura strongly resemble scurvy and the latter sometimes lacks the most striking features of the disease.

† The theories asserted as causative factors: (*a*) sodium excess, (*b*) potassium deficit, (*c*) ptomain activity, (*d*) acidosis, (*e*) deficient phosphorus compounds, and (*f*) specific infection, have one and all failed to stand.

‡ Dr. Cheadle, a colleague, had previously suggested that scurvy played a part.

named by Moeller in 1857. As before stated, the most aggressive symptom in children is the subperiosteal extravasation. The child lies motionless, shrinks from the touch, and presents what is really a characteristic swelling, the author having seen, as a student, in Dr. Barlow's and Mr. Edmund Owen's wards an immediate diagnosis on sight and touch in many instances.

Age incidence.

The disease is rarely seen in breast-fed children and occurs usually *from the sixth to the sixteenth month.*

It derives its general interest from the fact that the other symptoms of scurvy as seen in the adult may be entirely lacking.

The gums.

In children possessing teeth, mild scorbutic changes in the gums may be manifest but in very young babes these are wholly absent.

The usual symptoms of the hemorrhagic diathesis may be marked and in a considerable proportion hematuria may be an early symptom.

Posture.

Suggestive Symptoms.—Unwillingness to move the legs though the reflexes are wholly normal and the attitude of the babe, which lies with the thighs drawn up, cries out if handled and, manifestly, is comfortable only when in this position.

It may be shown usually that the lower end of the femur is exquisitely tender or this may be absent primarily and extreme pain on movement at the hip or knee be the chief symptom. This is followed by a firm, tense, extremely tender swelling which becomes bilateral and involves primarily and chiefly the upper and lower portions of the femur and upper third of the tibia but does not extend to the epiphyseal junctions. Other bones may be affected and even actual epiphyseal separation may result.

Misleading appearance

Similar subperiosteal blood extravasations may affect the ribs anteriorly and cause what appears to be a marked recession of the sternum and costal cartilages.

The diagnosis should be possible before such extreme symptoms appear.

DISEASES OF THE GLANDS OF INTERNAL SECRETION

ADDISON'S DISEASE ("*Morbus Addisonii*")

Historic Note.—This was first reported by Dr. Addison, of Guy's Hospital, in 1855, and in his monograph he attributed the ailment to a disease of the suprarenal capsules.

Definition.—*A disease characterized by progressive weakness and apathy, marked gastrointestinal disorders in the later stages, and a peculiar pigmentation of the skin and mucous membrane.*

Etiology.—The exact nature of the primary causative factors remains as yet undetermined. An antecedent tuberculosis seems the most important. Various diseases may precede it, but their direct connection in causation is unproven. Poor food, insanitation, and excessive mental or physical labor, are often associated factors, but may be only coincident.

Congenital Hypoadrenia.—Of late the impression that in cases of Addison's disease the chromaffin system is both anatomically and functionally inadequate from birth, has been strengthened. It would appear probable that in such cases the glands are peculiarly vulnerable to certain infections and especially to tuberculosis.

Males are affected much more frequently than females (60 to 70 per cent.). It is most common in middle age, yet cases have been reported in infants.

*Nearly 90 per cent. of these cases show demonstrable lesions of the suprarenal capsule, and in 80 per cent. of these instances the lesion is said to be clearly tuberculous. In a relatively small percentage a lesion of the capsule is not evident, and, furthermore, the suprarenals may be removed by operation or be congenitally absent without of necessity producing the disease.**

Etiologic paradox.

Morbid Anatomy and Pathology.—Any one of several conditions may be found at autopsy. These are, atrophy, simple or due to chronic interstitial inflammation, malignant disease of the capsule, extravasation of blood, inflammation or pressure affecting the semilunar ganglia and in a vast majority of cases tuberculous infection of the suprarenal itself. *It seems probable that Addison's original theory, that the disease was due to a loss or impairment of adrenal function, is correct.*

Impaired adrenal function

The involvement of the semilunar ganglia may materially affect the secretions of these glands and contribute to the symptom-complex. This view seems on the whole much more rational than that advanced by some observers, which holds the nervous system entirely responsible for the disease.

The Chromaffin System.—In the medullary substance of the suprarenals nests of cells occur which stain brown with chromic acid. These, the chromaffin cells, may be found in the solar plexus, in the ganglia of the sympathetic trunks and along the sympathetic nerves. Furthermore, such cells exist at the hilus of the kidney itself, at the point of origin of the left coronary artery, at the root of the mesenteric artery and in the left stellate ganglion.

* It is true, nevertheless, that experimental epinephrectomy in animals produces some of the most important symptoms of Addison's disease.

In the suprarenal gland the chromaffin cells constitute physiologically a system largely independent of its "inter-renal" fellow—a distinction which is emphasized in certain lower animals by an entire and permanent anatomic separation.

Deficit.

Adrenalin is the active constituent secretion of chromaffin tissue and it is probable that impaired function of the chromaffin system may account for at least *many* of the symptoms of Addison's disease.

Among those thus explainable are low blood pressure, subnormal body-temperature, progressive muscular weakness, the tendency to lymphocytic predominance in the blood picture, the lowered sugar content of the blood and the pigmentation itself, though the cortical system may play a part in the production of the cardinal symptom last named.*

SYMPTOMS.—*The disease is characteristically insidious in its approach and is rarely suspected until pigmentation becomes well-marked. The gastro-intestinal disturbance as well as the loss of strength tends to increase and may assume the form of crises, yet there are cases showing long periods of latency and relative immunity from vomiting, nausea and diarrhea.*

Blood.

A slight secondary anemia is usually present, but is not an important factor. *Lymphocytosis* is now held to be an almost constant finding and the polymorphonuclear neutrophil leucocytes are both relatively and absolutely diminished. *The total leucocyte count, however, does not often exceed normal figures.* An eosinophilic increase may often be demonstrated but is not constant.

The body-temperature is subnormal and the blood pressure is invariably low, dropping in some cases to 50 mm. (systolic).

As the disease progresses, attacks of syncope, vertigo and palpitation may be troublesome and sight and hearing may be impaired. Vomiting may become persistent, and indeed uncontrollable, sometimes ending fatally, but usually subsiding after several days. A history of active tuberculosis, past or present, in some other part of the body is often obtainable.

The pigmentation of the skin *is very variable in its intensity,* **the typical case** *showing a deep bronze color that is quite unmistakable, though the early pigmentation may be little more than a brownish-yellow or definite brown, and, rarely, the case may die before this deepens to the typical shade.*

Pigment distribution.

It shows certain interesting and important characteristics as regards distribution, in that it is most marked in the portions of the body exposed to light, such as the face and hands, and to those exposed to pressure or friction from clothing, being emphasized in the axilla, groin, under the breasts, or at the waist line and knees. Areas of pigment atrophy often appear, making a sharp contrast and the *mucous membranes of the lip and buccal surfaces* especially are likely to show patchy pigmentation.

Mucous membranes.

The heart invariably shows atrophy at autopsy.

* The interested reader should refer to Falta's masterly description of Addison's disease for an adequate description. "The Ductless Glandular Diseases," Blakiston, 1915.

An extended discussion of hyper- and hypo-adrenia is to be found in Tice's "Practice of Medicine," vol. viii, 1921, p. 131, and Oxford Medicine, vol. iii, p. 783.

Diagnosis.—The diagnosis is ordinarily easy in view of the grouping of low blood pressure, progressive loss of strength, marked gastrointestinal disorders and the peculiar pigmentation as above described. In those rare cases in which all characteristic skin discoloration is lacking, the autopsy alone suffices to positively confirm a tentative diagnosis. Misleading variations.

Differential Diagnosis.—Verminous bronzing is rarely seen except in the clinics of the great medical centers, being due to the constant presence of vermin, attended by scratching, which results in a more or less profuse pigmentation; in most instances the attending signs would be lacking as would certainly be the patchy mucous membrane pigmentation.* "Vagabond's disease."

Cirrhosis of the Liver.—In a few instances hepatic cirrhosis in its last stages may produce a very dark pigmentation, but in such instances there is almost always abundant evidence that the color is due to an actual jaundice. In no such case in the author's experience has any doubt arisen. Jaundice.

Argyria (chronic silver poisoning) is excessively rare at the present day, and the color is quite different from the pigmentation of Addison's disease, being more distinctly gray, and when excessive, showing a distinct bluish-black tint; moreover, it is ordinarily limited to the face and hands, and the associated symptoms of the latter disease are absent.

Parry's disease† and many other conditions are accompanied by pigmentation and peculiar patchy discoloration of the skin, *i.e.*, malignant disease involving the abdominal organs, certain cases of tuberculosis, of chronic nephritis, etc., but it does not seem that the difficulties in diagnosis are so great as to permit us to accept the dictum of Leube, to the effect that one "should forego any diagnosis of affections of the adrenal bodies."

The tuberculin reactions whether positive or negative are of slight value in view of the deceptive frequency of the former and the uncertainty of response in any such profound cachexia as is characteristic of the disease. Tuberculin test.

The strongest emphasis should be placed upon the absence of patchy pigmentation of the mucous membranes in the many simulators of Addison's disease.

Status Lymphaticus.—Certain evidences of this condition are not infrequently encountered and thymus hypertrophy has been reported.

Prognosis.—The disease is almost invariably fatal, usually within one or two years, and the last case observed by the author died after an illness of only a few months' duration. In rare instances life is prolonged for more than a decade and a few cures have been reported in cases where syphilis was possibly the primary factor. Variable.

STATUS THYMICOLYMPHATICUS, (*"Lymphatism" "Status Lymphaticus," "Constitutio Lymphatica"*).

Definition.—*A morbid state characterized by coincident hyperplasia of the lymph nodes, spleen, thymus, red bone marrow and, in certain instances, hypoplasia of the heart and arteries. A decided tendency to sudden death exists in this condition.*

* In such cases as have been observed by the author, the likeness was only sufficient to draw a passing reference.

† Exophthalmic goitre.

Historical Note.—The discussion of this curious condition is really a revival of ancient knowledge. Nearly three centuries ago *the association of an enlarged thymus with sudden death* is said to have been noticed by Plater, and *thymic asthma* was described by Kopp in 1830. This he regarded as a pressure asthma. Later discussions occurred, but the subject was again dropped to be renewed during the past few years.

Probable cause.

Etiology.—Little is known of the etiological factors, but the disease is one of youth and early childhood. It is probably associated with functional inadequacy of the chromaffin system and this belief receives clinical support in its not infrequent appearance in cases of Addison's disease and the fact that the blood picture is practically identical in these two conditions.

As to the functions of the thymus itself little of importance is known although many theories have been advanced and much conflicting testimony adduced.

Important complex.

Symptoms.—The symptoms upon which, as a whole or in part, a diagnosis must be based are essentially the following: *a pale, pasty, rachitic or tuberculous aspect, enlargement of the spleen and of the superficial glands, usually associated with tonsillar hypertrophy, adenoids and dulness over the upper sternum.*

The *thyroid* may be slightly enlarged, and there may be evidence of involvement of the *mesenteric glands.*

Disease of youth.

"Thymus death."

The chief clinical importance of the disease lies in its relationship to early and sudden deaths in the young or even the adult, both as occurring *without apparent cause* and in connection with operative or therapeutic procedures, the so-called "*thymus death.*"

This assumes considerable importance to both the physician and surgeon, cases having been reported as following adenoid operations, anesthesia, administration of antitoxin, convalescence from various infections, and croup.

Paltauf ascribes to this condition many of the cases of otherwise inexplicable sudden death in children. The condition merits further study and observation, and its symptomatology should be kept clearly in mind.

Attacks of causeless suffocative dyspnea and cyanosis occurring in babes or young children should always suggest the condition as a possibility and demand a most careful investigation including roentgenography and careful percussion.

A dull area over the manubrium whose lower border rises with extension of the head and falls with flexion is particularly suggestive and any dyspnea induced by throwing the head back may be regarded as a suspicious symptom.

MYXEDEMA AND CRETINISM

"Athyrea."

Definition.—*A chronic depressive disorder of cellular metabolism associated with more or less marked functional insufficiency affecting alike vegetative, psychic, circulatory and trophic spheres, associated with and resulting from structural changes and impaired function on the part of the thyroid gland.*

Sporadic or endemic.

Cretinism is the term applied to athyrea in the immature organism and may be either sporadic or endemic, the latter form appearing chiefly in regions where goiter is prevalent.

Age incidence

Signs of the disease usually appear at or about the fifth or sixth month but may develop earlier or much later. Some authorities attempt to distinguish two forms, the "infantile" and "juvenile."

Classification.—Although lacking in fundamental differences, three clinical groups may be distinguished:

First.—Cretinism *or myxedematous changes in the infant or young child.*

Second.—Myxedema adultorum, *i.e., as it appears in the adult.*

Third.—Operative myxedema, *which covers the changes attending the complete removal of the thyroid gland.*

Gland atrophy not constant.

Etiology.—That the loss of function which is usually but not invariably associated with *atrophy of the thyroid gland* is directly responsible for myxedematous changes has been well established by both clinical and experimental observations.

Heredity marked.

The disease attacks women five times more often than men, is peculiarly frequent in certain countries and districts, and in the case of cretinism seems to be markedly hereditary; the offspring of people who have removed from regions where cretinism is endemic showing a marked tendency to the disease whatever the nature of their new environment.

Predisposing conditions.

Congenital syphilis, a tubercular taint and rachitis seem to favor the development of the disease in children and infants. In these the disease is ordinarily directly due to congenital atrophy or deficient function, but does not make its appearance for several months after birth, as a rule, and sometimes not for many years.

The cretin.

SYMPTOMS OF SPORADIC CRETINISM.—The appearance of the affected child is characteristic and unmistakable.

Characteristic physiognomy.

The face is senile, coarse and stupid, the lips and eyelids thickened and overgrown, the tongue is large and thick and often protrudes from the mouth, the root of the broad, flattened nose is depressed, the head is large, clumsy and rests upon a thick, short neck.

Arrested development.

The stature is dwarfed, and a child of fourteen or fifteen appears often to be two or three years old.

The legs are bowed, the abdomen prominent, the joints thick and clumsy, the gait awkward, dragging, laborious, and marked lordosis is often present.

Depressed function.

The skin is sallow, the hair often thin and brittle, the skin usually dry, though it may be greasy, and signs of arrested mental development are as striking as the physical appearance.

Open fontanels.

The fontanels remain open and muscular weakness is usually marked.

Delayed dentition.

In infants the dentition is markedly delayed and, in all children affected, there is likely to be a great delay in the epiphyseal closure.

It is impossible to discuss fully in this volume a condition concerning which so little is positively known or to deal with the many conditions which may or may not represent mere variants. Certain cases seem to represent a form of infantilism.

The head enlarges but the growth of the facial bones is retarded, and myxedematous changes in the skin are usually marked.

Sexual organs.

Sexual development is distinctly retarded and imperfect, and the sexual instinct is almost wholly lacking in the female and but slight in the male.

Test medication.

Respiration is slowed, the temperature lowered and such patients do not sweat under the usual test doses of pilocarpin or show glycosuria following the administration of adrenalin.

ENDEMIC CRETINISM.—This form of athyrea or hypothyrea occurs chiefly in regions where goiter is prevalent among the adults of the population and differs materially from sporadic cretinism not only in the frequent absence of some of the extreme features (half-cretins) but also in its less marked and constant response to thyroid medication.

FIG. 55.—Cretinism. (*Courtesy of Dr. Henry Jackson.*)

Striking when typical.

Typical Symptoms of Myxedema in the Adult.—*The skin is dry, harsh and pasty, the hair lusterless and brittle, the face peculiarly moon-shaped and lacking in expression.*

The features are clumsy and coarse, the nostrils broad, the lips thickened and the tongue enlarged. Thought, motion and speech are slow and clumsy. The whole body is increased in bulk, and the superficial tissues have an appearance like edema, yet show no pitting on pressure. Large pads of firm inelastic tissue usually appear above the clavicles and on the extensor surface of the forearm and wrist, and the hands and feet are "pudgy" and clumsy.

Headache may or may not be present. Irritability is usually marked and actual delirium, hallucination and even true insanity may develop.

A bluish red color in the lips, nose and cheeks may be present and the palpebral fissures are often narrowed. Low blood pressure, readily excited exertion-dyspnea, *low body temperature* with more or less persistent or easily induced chilliness, are usually manifest.

Duration.—*The course of the disease may extend over a period of from ten to twenty years, the patient dying of intercurrent disease.*

Acute myxedema.

Osler reports what is apparently a case of acute myxedema and in some instances it seems to have been associated with the development of exophthalmic goiter.

The author has reported in 1905 a case in which very decided symptoms of myxedema coexisted with those of acromegaly.*

* See Transactions of the Association of American Physicians, 1905 (see also reference under "Acromegaly").

Operative myxedema does not differ materially from that described above. This condition is rare because it necessitates for its development the complete or almost complete removal of the thyroid gland.

Diagnosis of Myxedema.—*In the adult this must depend upon the peculiar physiognomy, the dry skin, marked edematous aspect of the body, and the failure of the bulky pads of tissue to pit on pressure, combined with the speech and mental state of the patient.*

Carbohydrate tolerance is greatly increased, although, in general, metabolism is distinctly depressed; 250–300 *grams of glucose producing no alimentary glycosuria, a fact of considerable importance in certain doubtful, illy-developed cases.*

Therapeutic test.

The improvement of all symptoms under the administration of thyroid gland offers conclusive proof of the nature of the disease, but loss of bulk alone is not sufficient evidence.

Masked Myxedema.—*Where one case of myxedema is recognized, a hundred cases of thyroid insufficiency escape our notice and it should be clearly understood that fragmentary clinical pictures fully justify and indeed demand test doses of the specific remedy.*

Extremely common.

Some of the most striking cases of improvement observed by the author have represented patients whose obscure symptoms barely suggested dysthyroidism as a possibility.

It is impossible to formulate any clean-cut group of symptoms which would lead us to a correct diagnosis in these cases of masked hypothyroidism. In many, one's suspicion is excited by the occurrence of a fragmentary myxedema picture made up of any one or several of the following symptoms: dryness of the skin, the presence of the so-called myxedema pads particularly at the back of the wrists or in the supraclavicular region, inactivity of the sweat glands, a slowing up of the mental processes, and the like.

Osler properly warns the clinician against laying undue stress upon the supraclavicular pads alone. As a matter of fact, this condition is very commonly seen in any stout person otherwise in perfect health and may be absent in decided thyroid insufficiencies.

Insufficient evidence.

But in many instances the therapeutic test, namely, the administration of thyroid gland, is productive of the happiest results even when no clinical picture is discernible. If the administration of this remedy is carefully and intelligently handled one need not fear the results, but every patient should be carefully watched least some larval hyperthyroidism be roused into activity.

HYPERTHYROIDISM

(*"Exophthalmic Goiter," "Parry's Disease"*)

Definition.—*A disease which, when fully and typically developed, is characterized by abnormally increased metabolism, prominence of the eyeballs, rapid pulse, enlargement of the thyroid gland and fine tremor associated with marked nervous manifestations.*

Atypical Forms.—*It may be, and often is, but imperfectly developed as a clinical picture, even when assuming an extreme and rapidly fatal form and may present an acute, subacute, chronic, or larval type, the last two being the forms oftenest encountered.*

Neither Basedow's nor Graves' disease.

The names of Basedow and Graves have apparently been improperly used in connection with this disease, and it should be called Parry's disease, as he described it quite fully in 1825, indeed having made notes of a case as early as 1786, half a century before the description of the two former appeared (Osler).

Hyperthyrea.

ETIOLOGY.—The disease is due chiefly to hyperthyrosis (increased thyroid activity), the exact cause of which is unknown.

It is the exact opposite of myxedema with respect to the vegetative functions, which are exalted to a marked degree.

It is probable, as suggested by Falta, that the symptomatic expressions of hyperthyrosis vary with and are dependent upon the participation and response of other sources of internal secretions in the individual case.

Age and Sex.—It is rare in men, quite common in women, and appears usually between the ages of twenty and forty, though rarely under the age of fifteen.

Emotional Crises.—It seems to be well established that violent or depressing emotion, even though transient, may produce it and in some instances it has seemed to the author that there was a marked sexual element. It can no longer be regarded as a disease of the nervous system.

Not a "nervous disease."

SYMPTOMS.—Acute cases *occur which, with or without marked exophthalmos or excessive or even marked tremor, may terminate fatally after a few days of violent and intractable gastrointestinal crises and profound and progressive cardiac weakness, i.e., precisely the same phenomena that may bring a chronic case to a fatal end.* Curious abortive or incomplete transient acute attacks also occur with or without the complete, classical, clinical picture.*

Types of extreme importance.

Ordinarily, the onset of the disease is gradual and its progress slow, its tendency being toward increase in the severity of all symptoms, but with marked periods of improvement if under observation and treatment.

Thyroid Enlargement.—It usually involves both lobes, but not equally, and may be unilateral or, at first, almost wholly absent. Venous bruit and loud systolic or double murmurs are commonly heard, there is usually a palpable thrill and often a visible expansile pulsation.

Exophthalmos.—This protrusion of the eyeballs varies greatly in degree, and it is said may become so great as to actually dislocate the eye.† The change is almost invariably bilateral, the vision is seldom affected, but

* A rapidly fatal case of this type seen recently by the author showed a relatively slight unilateral enlargement of the thyroid of sudden onset, following by several days an accident which resulted in a broken thigh.

Tachycardia was marked, vasomotor relaxation extreme, intractable vomiting dominated the clinical picture and the heart rapidly weakened, dilated enormously, and yielded no response to stimulation.

† No such extreme cases have ever been observed by the author.

much stress has been laid upon three so-called *signs* in connection with *exophthalmos.*

These are: (1) *The failure of the lid to follow promptly the downward movement of the eyeball* ("*Graefe's sign*"). (2) *Great lengthening of the time intervals of involuntary winking. These ordinarily represent but fifteen or twenty seconds but in this disease may be minutes apart if exophthalmos is marked* ("*Stelwag's sign*"). (3) *Deficient convergence of the eyes* ("*Moebius's sign*"). (4) *A tremor of the lids may be noted when the eyes are slowly and softly closed which has been dignified of late by the name of Rosenbach's phenomenon, but is neither limited to exophthalmic goiter nor new to medicine.* (5) *A lifting of the upper lids when the patient's gaze is fixed intently upon some object* ("*Kocher's sign*"). Over-rated signs.

All are somewhat superfluous clinically. The characteristic associated appearances, in any considerable grade of exophthalmos, are the peculiar stare and the rim of white which appears between the corneal margin and both lower and upper lids ("*Dalrymple's sign*"), but these are not necessarily due to either palpebral spasm or lid retraction.

Tests of Importance.—Of late several tests have been proposed for the detection of larval hyperthyroidism. It is well known that the basal metabolism is greatly increased, but the special apparatus needed and the time necessary for the completion of the tests and the special delicacy of the technic employed, render it quite unavailable for the average practitioner.

Hence, simpler tests are greatly to be desired. Of these, several have appeared during the last few years, the most readily applied being those of Goetsch and Loewi.

It must be remembered that neither of these have proven infallible, yet both may be considered useful, justifiable, and of considerable value.

Goetsch's Tests.—Goetsch has devised two tests; one, a skin reaction; the other, a general one.

The Skin Test.—Goetsch states that if 1 minim of a 1:4000 solution of adrenalin is injected *intradermically* "the central large area of blanching" results, this being "surrounded by a peripheral zone of reddening due to neighboring secondary vasodilation. In the blanched area a characteristic 'goose flesh' is often seen due to the contraction of the 'erector pili' muscles, which are of the smooth variety and under sympathetic control. The reaction lasts for one and one-half to two and one-quarter hours as compared with one-half to three-quarters of an hour in a normal individual, in whom, furthermore, the area of blanching is much less definite and the peripheral zone of red is usually entirely absent."

In practically all cases of hyperthyroidism, Goetsch states that the reaction is more marked and of longer duration than in a normal individual.

It should be added that of late much stress is laid on the behavior of the color of the areola produced in this test, which, in victims of hyperthyroidism, though fading to a lavender color remains for nearly or quite four hours after the time of the original injection.

The Hypodermic Test (Goetsch).—Seven and one-half minims (0.5 c.c.)

of a 1:1000 solution of adrenalin chloride is injected *hypodermically* in the deltoid region. If hyperthyroidism is present there is an "early rise of blood pressure, and pulse variation of from 10 to 50, and normally proportional to the degree of toxicity present." Among the associated symptoms produced are "asthenia, tremor, throbbing, vasomotor changes, apprehension and nervousness."

Note.—It is obvious that the second of these tests should be used with some discretion and that both must contain possibilities of error.

With respect to the hypodermic test, it would appear that the conditions under which it is performed should be controlled carefully, this being especially true if we are to draw any conclusions of importance from the rise in blood pressure of the lesser grades. Every practising physician knows how readily decided increases in systolic pressure occur and there would seem to be a considerable opportunity for error with respect to this manifestation.

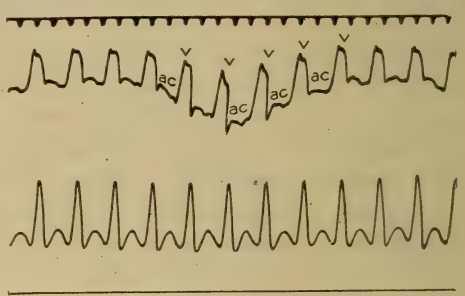


FIG. 56.—Tachycardia as frequently seen in exophthalmic goiter. Upper tracing, jugular; lower, radial. Pulse rate 132. Time ⅕ sec. Note respiratory curve in jugular tracing, and resemblance of pulse to that of aortic regurgitation. (Tracing by Dr. R. E. Morris, made with his modification of Mackenzie's ink-polygraph.)

Loewi's Test.—This consists in the instillation of a 1:1000 solution of adrenalin into the eye; dilatation of the pupil following if hyperthyroidism is present.

May for a time exist alone.

Rapid Pulse.—This is often the first symptom to attract attention. Pulse frequency is greatly affected by exertion and psychic excitation alike (labile pulse); regularity or irregularity may be present and the frequency may vary from 90 to an uncountable rate, as is frequently seen in fatal cases. Peripheral pulsation is marked, the abdominal aorta may throb violently and a capillary pulse or even a venous pulse is often present. High "*pulse* pressure" is common in well-marked cases.

Cardiac dilatation common.

Bruits.—All sorts of *bruits* may be heard at the base and over the gland or the vessels of the neck, usually associated with palpable thrill in well-marked cases, together with visible pulsation of the gland itself. A systolic murmur is seldom lacking in the mitral area *and the heart is not only readily dilatable, but often dilated.**

* Some of the most rapid and extreme dilatations observed by the author have been associated with acute hyperthyroidism and several have been post-operative complications. Residual post-operative dilatation of a decided degree is common and, too often, disregarded and far more rapid and satisfactory post-operative results would be obtained if the cases showing marked cardiac involvement were treated medically for a short period before surgery is attempted. At present, even in successfully operated cases, a period of several months (6–12) is usually necessary to the development of maximal improvement following surgical procedure.

Such cases usually are perfectly responsive to rest and digitalis, though in some, but not all, of the extremely acute forms the drug seems impotent.

Goiter heart may sometimes be due to pressure on the vagus, ("Rose's dyspneic form").

The glandular *bruits* are sometimes auto-audible, or to be heard at a distance. The subjective sensation of throbbing is often most harassing. The heart action may be violent, often tumultuous, and its area of visible pulsation greatly extended.

Tremor.—A fine involuntary tremor usually attacks the head and extremities, rarely the eyelids, lips or tongue, and is of great importance, as constituting one of the earliest and most constant symptoms of the disease.

"Formes frustes" important.

Tremor and rapid heart action may be present, separately or in combination, without exophthalmos or demonstrable goiter.

Decided exophthalmos is absent in at least 60 *per cent. of the cases.*

Blood Picture.—Aside from a decided tendency to a *relative* lymphocytosis or, more correctly, a mononucleosis the blood shows nothing of interest. This is increased during exacerbations and may be experimentally produced by thyroid feeding.

Subsidiary and Complicating Symptoms.—A vast number of symptoms may be encountered in connection with the course of the disease. Ordinarily extreme nervousness, insomnia, subjective sensations of throbbing, flashes of heat, excessive perspiration, general or local, excessive flow of saliva or, more rarely, dryness of the mouth, cardiac distress and mental irritability are those most marked. Less common are the marked gastrointestinal disturbances, complicating myxedemas, angio-neurotic edema, pigmentation or leukoderma of the skin.

Nervous symptoms marked.

Serious mental depression may occur or instability in the psychic sphere may manifest itself in ways most diverse and capricious. Irritability or actual choler may rapidly alternate with gaiety and exaltation.* Melancholia has been the common form observed, though acute mania sometimes occurs.

Mental disturbances.

Emaciation is often extreme in advanced cases; yet on the other hand, one frequently meets with those of the well-nourished type.

The disease is ordinarily chronic, lasting for several years; it is, moreover, frequently curable under medical treatment or may disappear and reappear

The author has thrice seen an acute firm enlargement of the thyroid gland associated *with marked nervous symptoms, tremor and tachycardia*, which lasted but three or four days.†

May be trivial.

Mere swelling of the thyroid gland, whether inflammatory or non-inflammatory, suppurative or non-suppurative, is not unusual, and moderate enlargement of the thyroid is so common in young girls at the age of puberty and in young women at the time of their first pregnancy as hardly to deserve notice. The surgical conditions of the thyroid cannot be considered here.

Usually unimportant.

* In one case coming under the author's observation an insanity developed temporarily, in an ancient *virgin*, with delusions of so unfortunate a nature as to involve wholly innocent parties in a serious scandal, the later development of the case making all clear.

† The last case two weeks.

ACROMEGALY (*"Acromegalia, Marie's Disease"*)

Pituitary gland affected.

Definition.—A chronic nutritional disease, supposedly attributable to overactivity of the anterior lobe (glandular) of the hypophysis, and characterized by progressive enlargement or overgrowth of certain portions of the body, chiefly and primarily affecting the bones of the hands, feet and face, essentially chronic and progressive in its course and tending to a fatal issue after the lapse of many years.

Historical Note.—It was first described by Marie in 1886, having existed for thousands of years without recognition as a clinical entity, though presenting one of the most striking pictures known to medicine.

Fig. 57.—Acromegaly showing enlarged and clumsy bones (case of L. G.).

Autopsy finding.

Etiology.—The cause of the disease is unknown, though the fact that in nearly every case that has come to autopsy the pituitary gland has shown definite changes, usually adenomatous or adenocarcinomatous, points to that curious structure as the probable source of the disease.

Physiologic partition.

The pituitary gland is a secreting organ as regards its anterior portion, whereas its posterior lobe has evidently a nervous function. As its duct has become atrophied in the process of structural development, the secretion has become an internal one taken up by lymphatic absorption. But little is definitely known with respect to its functions despite the excellent work of many workers during the past few years.

Center for growth.

There is much to support the belief that it is the main center for body growth and that it bears some curious but little understood relation to the thyroid and suprarenal glands.

A relation between gigantism and acromegaly has been suggested and is probable. It would seem possible that as suggested by Brissaud and Meige, if the hyperpituitarism occurs in childhood, gigantism results, but if delayed until ossification of the epiphysis is complete, acromegalic changes occur.* The disease occurs more frequently in women than in men, usually begins in early adult life, almost never after the age of forty, and no recognized preëxisting disease has been definitely connected with it.

Sex and age.

* This is denied by Falta and others who insist that hyperpituitarism alone does not produce gigantism and that acromegaly may occur while the epiphyseal junctures are still open.

SYMPTOMS.—If well-developed, it is essentially a case for street-car diagnosis, being easily recognized by its outward signs. The face is elliptical in form, the superciliary ridges being prominent, the head often large and massive, the lower jaw prognathic, the features enlarged and coarse also by reason of the marked thickening of the greasy integument, in which appear deep creases, especially marked across the forehead.

Street-car diagnosis.

Fig. 58 Acromegaly.—Showing predominant hypertrophy of great toe (case of L.G.)

In many cases even the lids are thick and coarse, the hands and feet are enormously enlarged, the fingers are spatulate and clumsy, and upon examination the increase is found to depend upon the bony overgrowth, a finding easily verified by the X-ray. This growth may extend for a considerable distance upward along the bones of the forearm and leg, and sooner or later tends to involve the clavicles, scapulæ, ribs and spinal column, at which time the patient is likely to assume a peculiar stoop, due to kyphosis of the spine. The X-ray may reveal an enlargement of the *sella turcica*.

Striking symptoms.

Sella turcica.

There are no constant symptoms, as far as the internal organs are concerned, and decided subjective symptoms, such as headache, mental irritability, malaise, joint pains, disturbance of vision or drowsiness, may be absent until the later periods of the disease.

The tendency is toward a fatal termination after a long term of years, in some cases covering two decades, and the symptoms are subject to many variations and exceptions.

Prognosis.

In one case which, fortunately, has been under the author's close observation for nearly twenty years, most interesting variations have been observed.* In this case the predominant facial hypertrophy in the bones of the face appeared in the *upper* jaw; the bones of the feet were but slightly hypertrophied as compared with those of the hand, yet after a

An unusual case

* Transactions of the Association of American Physicians, 1905.

term of years marked localized overgrowth appeared in both great toes. Another, then rare, phenomenon was a very marked co-existing myxedema, which promptly vanished each time that a course of thyroid medication was applied, finally disappearing altogether at a time when a previously existing marked *enlargement* of the thyroid gland slowly subsided. In this case also there was from the beginning a chronic synovitis of the knee-joints, persisting and so weakening the ligaments as to permit displacement of the patella and render the patient liable to dangerous falls. At the present writing this individual still lives, but grows gradually weaker and is subject to attacks of vertigo and occasional syncope.

Differential Diagnosis.—The difficulties attending the diagnosis of acromegaly relate almost wholly to its early or relatively early recognition, and this must usually depend upon the testimony of the patient, or those in daily contact with him, relative to changes in physiognomy, often best revealed by a comparison of photographs taken at different ages, and to a definite enlargement of the head, hands or feet necessitating changes in the size of the hat, gloves or shoes, respectively.

Fig. 59.—Hand. Case of pulmonary osteoarthropathy.

The marked enlargement of the *sella turcica*, readily demonstrated in most of the advanced and decided cases with actual tumor, may be lacking in certain cases. Acromegaly, it is said, may be mistaken for myxedema, leontiasis ossea, osteitis deformans, arthritis deformans, and pulmonary hypertrophic osteoarthropathy. There is hardly a shadow of an excuse for any error along these lines.

Osteitis deformans is rather a matter of deformity than overgrowth, and in it the cranial bones are chiefly affected rather than the facial bones as in acromegaly. Furthermore, the shape of the head in osteitis deformans is characteristic, its broadened cranial portions contrasted with the narrower maxillary region, a condition usually reversed in acromegaly.

Leontiasis ossea shows merely bony prominences on the skull and face, and lacks every characteristic of acromegaly.

In pulmonary hypertrophic osteoarthropathy enlargement of the hands and feet exists, but is confined chiefly to the articulations. The face is not affected, and chronic pulmonary disturbance of some sort, primary or secondary, is an invariable accompaniment.

Syringomyelia causes enlargement of the extremities but is readily differentiated by the associated symptoms.

Arthritis deformans certainly could not be mistaken for acromegaly under any conditions.

The demonstration of an enlarged *sella turcica* by the X-ray, a procedure already mentioned, is a valuable and sometimes crucial test.

Gigantism.—There are two chief divisions of giants, viz., the *acromegalic* and the *eunuchoid*. In the latter the genitals are distinctly of the infantile type and the bones long and slender.

It is assumed to be the result of hyperpituitarism existing prior to uniting of the epiphyses.

Hypopituitarism.—The evidences of this condition vary with the age period at which the pituitary insufficiency occurs.

Its chief effects express themselves in the retardation or actual limitation of growth, underdevelopment and, in adults, impairment of sexual function, obesity and in the female amenorrhea.

Dwarfism in some forms is probably an expression of the condition and in young children a syndrome embodying adiposity and arrested sexual development may be associated with pituitary tremor.

INFANTILISM.—Closely related to myxedema and cretinism is the curious condition termed infantilism of the Lorain type or those described by Gilford as ateliosis and progeria.

The former type (Lorain) represents merely man or woman in miniature. In the latter (Gilford) there is an asexual type representing delayed development and a sexual form represented by the ordinary traveling showman's dwarf, the delayed development in this case yielding along sexual lines at puberty.

Progeria covers cases of infantilism associated with premature senility, outward and structural.

PANCREATIC INFANTILISM.—This condition, characterized by a marked amelioration under the administration of pancreatic extract, is described by Byrom Bramwell.* In this case the developmental arrest was complete for seven or eight years, but in the course of nine months' treatment there was a gain of 8½ pounds in weight, and nearly 2 inches in growth. The patient was eighteen years of age, his developmental arrest occurring at eleven or twelve. The various classifications of infantilism are unsatisfactory and probably do not rest as yet upon a very substantial basis.

EXOGENOUS OBESITY.—Obesity may be general or local, and its cause may be excessive food consumption, or defective employment of normal oxidation or elimination.

In view of the discomfort caused by the excessive accumulation of fat, its tendency to limit proper exercise and cause impairment of the functions of vital organs, such as the heart, one may fairly consider the condition as a morbid state tending directly to shorten life. The general conformation of the body should be given consideration, and *large bones*, unusual muscular development and a *relatively* small waist measurement are factors that materially modify the estimation of individual longevity. This means that *excessive fat* is the condition most feared.

Shortens life.

Lines to be drawn.

There are two distinct types of the obese, namely, the anemic and the plethoric, with little to choose between them, so far as life expectancy is

* Clinical Studies, Vol. 1, part 2, Jan., 1903.

concerned. The excessively obese are peculiarly liable to disturbances of the secretory organs, heart disease, asthma, diabetes, gall-stones, gout, apoplexy. Furthermore, they succumb readily to severe acute infections, and are bad subjects for major operations.

ENDOGENOUS OBESITY.—In certain cases of obesity the combustive capacity of the individual is subnormal.

General Comment.—As is well known, certain races or tribes deliberately cultivate obesity in their women, making the pudgy outlines of the Hottentot Venus their ideal of feminine beauty. In all races 90 per cent. of the cases of excessive obesity occur in women; furthermore, the condition is often congenital. Cases are reported in which a baby thirteen months old weighed 75 pounds, a child four years old weighed 256 pounds. In some of these less pronounced cases the excessive fat of childhood disappeared largely in adult years. Cases of excessive weight have been reported that tax one's credulity to the utmost. Daniel Lambert is said to have weighed 730 pounds. A case was reported in Baltimore in which the weight was 850 pounds, and one from North California is said to have reached 1000 pounds. These cases are ordinarily short-lived, and the fact that few excessively fat people attain advanced age cannot fail to strike even the lay observer.

Adiposis dolorosa.

In a rare and curious disease known as "adiposis dolorosa," more likely to affect women than men and falling almost wholly between the age periods of forty-five and sixty, the excessive accumulation of fat is associated with asthenia, headache, pain, tenderness of tissues, and mental disturbances.

The accumulation of fat in this disease ordinarily occurs in the form of bunches or nodules, though later becoming widely distributed in most instances. Its cause is possibly a disturbance of internal secretion but nothing certain is known. Degeneration of the ultimate nerve filaments has been reported and in the absence of the typical findings with respect to fat deposit its differentiation from an alcoholic polyneuritis may be difficult or impossible.

It is wholly probable that "*adiposis dolorosa*" as an entity will disappear soon from medical literature.

CERTAIN DISEASES OF UNKNOWN CAUSATION

OSTEITIS DEFORMANS. (*"Paget's Disease"*).—**Definition.**—A rare disease characterized by kyphosis of the upper spine, a broad-based thorax, lozenge-shaped abdomen, marked enlargement of the cranial portion of the head and enlargement and deformity of the long bones, due to a rarefying osteitis. The disease is extremely rare and of unknown etiology and needs no further description.

LEONTIASIS OSSEA.—A disease characterized by hyperostosis of the bones of the cranium, rarely those of the face, in some instances combined with localized hypertrophy of the soft tissues.

MICROMEGALY.—This disease is the opposite of acromegaly, is excessively rare, and of unknown causation.

PULMONARY HYPERTROPHIC OSTEOARTHROPATHY.—(*Bamberger's Disease*).—This ailment, associated almost invariably with chronic pulmonary disease, is characterized by an enlargement of the hands and feet, the distal portions of the long bones, joints, and terminal phalanges chiefly being affected. The finger-nails become brittle and show longitudinal striation.

Achondroplasia.—Victims of this curious developmental abnormality are seen frequently on our streets active, intelligent, wearing a facial aspect reasonably well suited to their actual age, but seldom exceeding in height that of a well developed child of ten, and almost invariably wearing a "pug" nose as evidence of precocious ossification.

Active and intelligent

Pug-nosed dwarfs.

The genitals develop normally to full adult proportions.

The skull is so enlarged as to *suggest hydrocephalus, the four fingers of the hand are of nearly equal length,* and if, as happens often, the mid- and ring-fingers stand apart, the two pairs of fingers with the thumb form the "trident hand." The trunk is nearly or quite of adult proportions but the arms and legs are extremely short. These cases are readily distinguishable from the cretinic dwarfs on the one hand and those of pituitary disease (Fröhlich's syndrome), with their obesity and infantilism, on the other.

A great variety of cases of excessive fat accumulation may be placed under this head, but their exact classification is unsatisfactory and the condition may arise from various disturbances of the internal secretions. Among these, the obesity of hypothyroidism is especially important, as is that following castration and the menopause.

URINALYSIS AND DISEASES OF THE KIDNEY

General Considerations.—*The normal activity of the kidney depends upon a proper performance of function on the part of the other related organs, as well as upon the structural and functional integrity of the kidney itself.*

Relationships marked.

Its remarkable intrinsic nervous mechanism makes it difficult to apply rigid rules governing its activity, but it may be said that the *quantity of urine and inorganic* salts depends upon osmotic glomerular filtration as affected by blood pressure and volume, whereas the output of *urea and its congeners* is determined by the peculiar selective action of the cells of the convoluted tubules.

Filtration.

Selective cell action.

Therefore, anything affecting the renal structure or circulation, directly or indirectly, must alter the amount and constitution of the urine.

Value of Urinary Examination.—*No other secretion or excretion of the body furnishes greater or more varied information. Any substances which are taken into the blood stream are excreted ultimately in large measure by the kidneys, and through a critical chemic and microscopic examination of the urine we can measure variations in nutrition and waste, and obtain positive, suggestive or corroborative data with relation to many diseases, both local and general.*

Wide scope.

Poisoning (coal-tar products, morphia, lead, mercury, chloral, etc.), Bright's disease, diabetes, spermatorrhea, cystitis, pyelitis, gonorrhea,

oxaluria, gout, the presence of fever, a failing heart, typhoid fever, pneumonia, septicemia, urinary and general tuberculosis, filaria sanguinis hominis, dyspepsia, chronic appendicitis, hysteria and partial or fully established obstructive jaundice may be mentioned as some of the conditions, either directly diagnosticated, or suggested, by urinary signs.

The ordinary examination of the urine is largely qualitative, but certain necessary quantitative methods involve only a small amount of apparatus and a negligible loss of time, and all vital procedures may be carried out by simple methods whose margin of error is not so great as to vitiate clinical results.

Obtain 24 hours' urine.

The First Step in the Urinary Examination.—*The total urine for twenty-four hours best represents the functional activity of the kidney as affected by changes in its structure or diseases of related organs.*

Collection and preservation.

It should be collected in clean bottles, the night urine separate from that of the day, and if it cannot be kept cool, a preservative should be added. Chloroform is the best agent readily available and is readily removed by heat. *Camphor, thymol, chloral and formalin give rise to misleading reactions.*

Toluol is an admirable preservative and, as it floats upon the surface, need not be removed, the urine needed for examination being withdrawn by a pipette as needed.

Procedure.—The urine passed on the first morning is disregarded, all voided for the remainder of that day saved, thoroughly mixed, and kept in a cool place. That passed after retiring and that portion voided upon arising the *following* morning are separately collected.*

In certain important renal diseases, notably in advanced interstitial nephritis and certain cases of cardiac insufficiency, the normal ratio of day to night excretion is greatly modified or reversed.

Night vs. Day.

Ordinarily, the day urine is three or four times greater in volume than that of the night, hence any marked departure in the direction of equalization or of reversal of preponderance is suggestive.

Best.

Single Specimens.—Of the individual specimens, that passed late in the day, preferably after a full meal and exercise, is most likely to contain albumin or sugar, and, *except in suppurative disease of the kidney or bladder, that least likely to show abnormalities is the early morning urine.*

Worst.

Diverse factors.

Normal Variation.—*The normal amount* of urine varies from 750 to 2000 c.c., and this variation may be temporary or permanent, depending upon exercise, the amount of perspiration, the kind and amount of water ingested, or the nervous condition of the individual. The smaller amount represents usually either diminished ingestion or increased loss of fluid through other channels, such as free perspiration during hot weather, or a diarrhea. Women drink little water and usually show a small daily total, and children secrete *relatively more* though *absolutely less* than adults.

Women and children.

Transient and trivial.

POLYURIA.—Temporary vs. Persistent.—An increased flow may be temporary or permanent, purely functional (psychic, emotional), or, due to

* This insures at least one fresh specimen should decomposition occur in either of the others, and a mixing of the three gives the twenty-four hours' total.

local or general organic disease. The ingestion of large quantities of water or such diuretic substances as beer or gin, the use of certain diuretic drugs, hysteria, migraine, and atmospheric humidity with *low* temperature, markedly, though temporarily, increase the amount.

Persistent polyuria usually indicates actual disease, and is encountered in certain neuroses (*urina spastica*), as well as in chorea, pyelitis, amyloid kidney, interstitial nephritis, convalescence from acute nephritis, absorption of exudates, and diabetes mellitus or insipidus. Usually important.

Diabetes mellitus and certain rare cases of interstitial nephritis violate the accepted rule that increased flow of urine results in or is associated with a lowered specific gravity. In diabetes insipidus the total urine shows increased total solids, though the individual voidings yield diametrically opposite findings.* Polyuria and specific gravity.

OLIGURIA.—*Pathologic diminution* is usually associated with severe acute congestion, various forms of acute nephritis, chronic parenchymatous nephritis in its active stage, or with circulatory diseases and conditions producing passive congestion. Among these are cardiac weakness from any source, cirrhosis of the liver, chronic emphysema, or pressure due to abdominal growths or a pregnant uterus. Associated conditions.

Relation of Oliguria to Color and Specific Gravity.—A marked oliguria accompanies acute infections associated with toxemia and high body temperature, profuse diarrheas or hemorrhages, shock or collapse.

In nearly all instances a reduction in the total quantity of urine is associated with an increase in coloring matter and a heightened specific gravity of the individual specimen. This last may be present from various causes in the individual voidings, even though the total excretion of solids is markedly diminished.

ANURIA.—*Complete suppression of urine may result from many causes and, though always serious, may or may not prove critical.*

It may occur in nephritis, renal tumor, phosphorus poisoning, shock or collapse, *hysteria*, ureteral calculus, cholera, and as a sequel to nephrectomy. If but one kidney is functionally potent, pressure upon the ureter of the other organ may produce complete anuria. Etiologic factors.

It is sometimes directly obstructive and, in cases not due to actual renal disease, has been known to exist for two weeks or more without producing uremic manifestations. Unusual duration.

Frequency of Micturition and Dysuria.—*Increased frequency associated with pain usually indicates disturbance of the renal pelvis, bladder, prostate or urethra, rather than disease of the kidney itself.* Painful.

It may be due to simple concentration and high acidity or, more frequently, urethritis or cystitis. Relatively or absolutely painless increased frequency may accompany any increased urinary flow, normal or abnormal, or certain stages of active disease, acute or chronic, of either the pelvis or the parenchyma of the kidney. Painless.

* Ogden reports such an instance—"The Clinical Examination of the Urine," 1903. The author has never encountered one though the relatively high specific gravity of the scant urine of terminal cardiac incompensation in such cases is a common finding.

An important sign.

Habitual rising at night to void urine may be due to urethral stricture, an enlarged or inflamed prostate, gravel, renal or cystic stone, diabetes mellitus or insipidus, malignant or tuberculous diseases of the genito-urinary tract, interstitial nephritis, *insomnia*, and, *rarely*, to mere habit. Nearly all patients exhibiting the polyuria of interstitial nephritis or the frequent night micturition of prostatic disease believe the frequent voiding to be merely the result of "habit."

THE COLOR.—Fresh normal urine varies from pale yellow to yellowish-red, and all urines may be classified as pale, normal, high-colored, or dark.

Specific gravity and reaction.

Deep color is usually associated with relatively high specific gravity and marked acidity; pallor with low specific gravity and lessened acidity or alkalinity, diabetes mellitus furnishing an exception to the rule.

Indeed, so striking is the heavy weight of diabetic urine in connection with its light color, that a provisional diagnosis is often suggested when lifting the tube or bottle.

Fever urines are usually high-colored, scant and of high specific gravity, though fever may of course exist in exhausting and wasting diseases with a relatively pale color and low specific gravity.

Brown, Black, Smoky, or Red Urines.—(*a*) **Blood.**—*Color red, reddish brown, smoky, brown, or black.*

Black.

(*b*) **Melanemia** (*Melanotic Sarcoma*).—*The urine becomes black on standing, but does not reduce Fehling's solution.*

(*c*) **Alcaptonuria.**—*Urine becomes black on standing and reduces Fehling's solution.*

Green-black.

(*d*) **Poisoning by Coal-tar Products.**—The greenish-black color is due to hydroquinon and follows the excessive use of drugs like carbolic acid, naphthalin, guaiacol, resorcin, salol, creolin and lysol.

Claret-color.

(*e*) **Hematoporphyrin.**—Color of port wine or Bordeaux.

Yellow foam.

(*f*) **Bile.**—*Greenish, yellowish-green, greenish-brown or deep brown urines may contain bile pigment*, in which event the foam produced by shaking the liquid is yellow. Urine containing bile may, when freshly passed, be reddish brown, but oxidation of the brown bilirubin converts it into biliverdin and produces the greenish tinge. *A deep green urine results from the administration of methylene blue.*

Yellowish-green, greenish-brown, reddish-brown.

Deep green.

Blue urine usually indicates a great excess of indoxyl products, as in ileus, cholera and typhus, but is rare. **Orange and reddish-brown** urines suggest rhubarb, senna, or chrysophanic acid; **yellow** urines, santonin.

INDICAN.—(*Indoxyl-potassium-sulphate*).—*This normal urinary chromogen and tryptophan derivative results from albuminous putrefaction in the presence of bacteria; and, if found in the urine in excess, suggests intestinal stasis or obstruction, septic processes or the excessive ingestion of red meat.*

Of some diagnostic value.

C. E. Simon reports an excess in hypochlorhydria, anachlorhydria and the hyperchlorhydria of certain gastric ulcers, and an excess is found in typhoid fever, appendicitis (chronic or acute), cancer of the stomach, peritonitis, chronic gastritis and similar conditions, reaching its maximum in stasis and ileus.

*Obstruction in the lower colon or simple constipation does not materially increase it.**

Test (*Jaffé-Stokvis*).—(*a*) *Take equal parts of the urine and strong hydrochloric acid.* (*b*) *Add two or three drops of a saturated solution of sodium or calcium hypochlorite or of common saltpeter. Shake. Add* 1 *c.c. of chloroform, shake thoroughly and repeatedly and set aside. The chloroform then shows a depth of color varying with the amount of indican present. Potassium iodide if present may yield an intense carmin, and codein a reddish-purple.*

Establish a rough standard.

The observer should establish a normal standard by repeated observations using always the same quantities of urine and reagents. A more accurate test involves the use of a small amount (not an excess) of lead acetate sol. (20 per cent.) which removes the urinary pigments in the precipitate.

Standardized color solution.

Quantitative Tests (*Strauss's Method*).—This involves the use of a standard color solution obtained by dissolving 1 mg. of C. P. indigotin in 1000 c.c. of chloroform. This should be carefully sealed and kept in the dark.

Take of urine 20 c.c., add 5 c.c. of lead acetate sol. (20 per cent.) which precipitates the urochrome, filter. Mix 10 c.c. of Obermayer's reagent† with 10 c.c. of filtrate (= 8 c.c. urine), add 5 c.c. of chloroform, cork, and shake gently for two minutes. Remove chloroform, add another 5 c.c. of same and so continue until no color is extracted. Of the chloroform used take 2 c.c. in test-tube, add chloroform *guttatim* until the color corresponds with that of the standard solution in a control tube of equal caliber, both being held against a white background.

The total number of cubic centimeters of chloroform used both in extraction and dilution represents the amount containing 1 mg. of indigotin.

By multiplying the total amount of chloroform used for extraction by the amount used to dilute to a standard color and dividing by two which corresponds to the amount subjected to this dilution we obtain the amount necessary for complete standardization of the total chloroform used.

Recto-vesical fistula.

INDOL (Excreted as indoxyl potassium sulphate).—*When found in substance in the urine this suggests recto-vesical fistula;* otherwise, the same statements and tests apply as are given under "Indican."

Test for Indol in Substance.—*Cholera red reaction.* Add to the urine a few drops of dilute sodium nitrite solution and gently pour it down the side of a tube containing sulphuric acid. *Indol yields a purple color at the point of contact, a diffuse pink on shaking and blue-green on neutralization with sodium hydrate.*‡

* Winternitz considers the absence of an indican increase in ovarian disease of differential value in cases where doubt arises as between this disease and a chronic appendicitis, in the presence of symptoms equally explainable by either condition.

† Obermayer's reagent. C. P. HCl 1000, ferric chloride 2. This forms a permanent fuming yellow mixture. Accurate quantitative determination is not essential.

‡ This is simple and sufficiently delicate, but other tests, such as that of Ehrlich, will show 1 part of indol in 400,000 parts of urine.

Diagnostic importance.

SKATOL (Excreted as skatol-carbonic-acid).—This substance is said to be especially abundant in cases of tuberculous ulceration of the intestines, gastric or intestinal carcinoma and pneumonia, and is not increased in the other diseases showing an excess of indoxyl (F. C. Wood).

Rosenbach's Test.—*Add nitric acid, guttatim, to boiling urine. A Burgundy-red* color with a bluish-red foam on shaking indicates skatol. A reddish or brownish red is usually due to urobilin.

Diagnostic value trivial.

UROERYTHRIN.—*This normal urinary pigment is increased in febrile states, gout, in various dyspeptic conditions and after excesses in food or alcoholics. It gives an orange color to the urine and a rose tint to uratic sediments.*

Test.—Add C. P. sulphuric acid to the urine guttatim and a carmine-red color reaction appears, or some pink urates dried or filtered may yield a bright green color with a drop of sodium hydrate solution.

MELANIN.—This substance produces a brownish-black urine or one darkening thus *from above downward upon exposure to the air* (melanogen). This color is intensified by the addition of nitric acid, bromine water, chromic acid or ferric chloride.

Significance.—It is the pigment of melanotic sarcoma and carcinoma (see also "Alcaptone" which also produces dark urines).

UROBILIN.—*This, or rather its chromogen, urobilinogen, in very small amounts is a normal constituent of urine, is derived from bilirubin by a process of reduction within the intestine and is probably the stercobilin of the feces.*

In excess it suggests an acute infection, such as sepsis, scarlatina, pneumonia, acute rheumatism, malaria, typhoid or erysipelas, as well as lead colic, hepatic cirrhosis, *hemolytic pernicious anemia, congenital hemolytic jaundice* or internal hemorrhage. It may accompany passive congestion, as in a weak heart, the ingestion of such substances as antipyrin and antifebrin, chloroform inhalation or the injection of tuberculin (Wood).

It may be also the forerunner of, or alternate with, jaundice, constituting the *urobilin icterus*, most frequent in atrophic cirrhosis, carcinoma and pneumonia. It will readily be seen that it assists but slightly in exact clinical diagnosis save in relation to hemolytic processes.

Of slight diagnostic value.

Chemic Test.—To the reddish or reddish-brown urine, which upon shaking usually shows a brownish or even yellow foam, one adds ammonia freely and follows with a few drops of a 1 per cent. solution of zinc chloride. Urobilin is indicated by a green fluorescence.

Spectroscopic Test.—Dilute urine if necessary, add a few drops of tincture of iodine to each 10 c.c. of urine, and the spectroscope will show a broad absorption band between the green and blue if urobilin itself, not its chromogen, be present.

THE OXYACIDS.—These have the same significance as indoxyl and have no special clinical importance.

ALCAPTONE.—This rare substance, a derivative of the amino-acids, has no clinical importance aside from the fact that it produces a urine which, like that containing melanin, slowly darkens on standing or, rapidly, following the addition of an alkali. It reduces Fehling's *solution*, though not affecting

the fermentation, phenyl-hydrazin or bismuth sugar tests, and yields a negative polariscopic test. See also "Melanin" and "Hematoporphyrin," both of which produce dark urine.

HYDROCHINON AND PYROCATECHIN.—Both of these substances produce a urine which darkens on standing, but are of no other clinical importance.

UROCHROME AND HEMATOPORPHYRIN.—These constant normal urinary pigments are often associated with urobilin and uroerythrin. The first is the chief yellow or amber pigment, the second scant and red. (Urobilin yields a yellowish brown and uroerythrin a pink color to the urine.)

A useful procedure.

Urochrome is readily precipitated by lead acetate solution, leaving a clear urine well adapted to spectroscopic examination.

Urochromogen Test of Weiss.—This simple test consists in dividing a small amount of urine into two portions, reserving one for control, and adding to the test specimen a few (2 or 3) drops of potassium permanganate solutions, 1 : 1000.

A canary-yellow color constitutes a positive reaction and is said to be of special importance in relation to prognosis in pulmonary tuberculosis.*

Hematoporphyrin is an iron- and albumin-free derivative of hemoglobin and is often excreted in excess in chronic users of trional, sulphonal, tetronal, and the like, the urine being by transmitted light Burgundy red, and, by reflected light, dark brown or black. It is probably derived from hematin.

Salkowski's Test.—Treat 30 c.c. of urine with a mixture consisting of equal parts of a solution of barium chloride (10 per cent.) and cold saturated solution of barium hydrate. Wash the resulting precipitate with water, again with absolute alcohol, and shake it up repeatedly with a warm solution representing 10 c.c. of alcohol and 6 to 8 drops of HCl.

If the test be positive, a red-violet color results and the solution yields to the spectroscope the characteristic double bands of acid hematoporphyrin, viz.—a narrow dark band in the orange and a broader one between the yellow and the green in contact with a lighter band in the yellow.

CREATININ.—This derivative of the ingested meats follows the same laws as urea as to the variation in excretion, both in health and disease.

Test.—It is easily recognized by the *intense* red color produced by adding a little saturated solution of picric acid and a few drops of sodium hydrate (*Jaffé's test*) to the urine.

Various other substances of no clinical importance but related to the foregoing organic compounds are omitted.

* M. E. Cowen has recently reported the results of testing 832 cases at the Cresson Sanatorium and believes that "only in the presence of an actual destructive process does the positive reaction appear. He would apply it, therefore, both in prognosis and in the management of the individual case. The author has had no personal experience with the test, but if its name is truly descriptive it should have much the same incidence, significance and value in this disease as would positive diazo-reactions, *i.e.*, according to the author's experience it would indicate *active mixed infections*.

Chylurea, pyurea and lipurea.

MILKY URINE.—Such an appearance is usually due to pus or chyle. Pus forms a sediment and its characteristic cells are easily detected by the proper chemical and microscopic tests.

Free fat upon the surface is almost invariably from an unclean bottle, the local use of some unguent, or, rarely, to extreme fatty degeneration of the kidney. Fat cells are characteristically refractile under a shifting focus and stain black with osmic acid and red with Sudan III. It cannot be denied that a true lipurea occurs as a rare finding in many diseases or even in an alimentary form.

Chyluria.—This rare condition is almost pathognomonic of the presence of *Filaria sanguinis hominis*. The urine appears usually like skim milk though often tinged with pink. The typical large fat globules are absent, blood and lymph cells present, and the fat cells appear under the microscope as fine mote-like particles. The urine resembles a fine emulsion. A "cream" layer may rise on standing and the parasite itself be found in the fluid.

Tests for Fats.—The microscopic or macroscopic examination suffices in many cases.

Chemic Test.—Add potassium hydrate and ether, shake, decant and evaporate the supernatant fluid. The fat is taken up, held in solution and remains after evaporation.

Fibrinuria.—In rare instances, as in some cases of villous growth in the bladder, the urine jellies on standing, or, if less fibrin be present, a sticky sediment may adhere to the bottom of the glass.

The nubecula.

TRANSPARENCY.—*Normal, undecomposed urine is usually perfectly clear save for a slight mucous nubecula floating in its upper portion.*

Physiologic sediment.

Even in freshly passed urine, however, earthy phosphates or the amorphous urates sometimes cause a physiologic sediment, *the phosphates being associated with feeble acidity or alkalinity;* the urates with concentration and acidity.*

Persistent opacity.

Heating increases the precipitation of phosphates and redissolves urates. Persistent opacity or turbidity is pathologic in undecomposed urines and may be caused by pus, blood, fat or bacteria.

ODOR.—The peculiar odor is intensified in sharply acid, concentrated urine and distinctly and characteristically modified in alkaline urine; the volatile alkali (ammonia) being easily detected if present, as are indol and skatol in recto-vesical fistulæ, the odor of hydrogen sulphide in hydrothionurea, bitter almonds in nitro-benzol poisoning, etc.

Fixed vs. volatile alkali.

REACTION.—The reaction may be acid, alkaline or amphoteric, *i.e.*, doubly reactive.† *If alkaline, the physician should determine by repeated tests whether the condition is temporary or permanent* and the alkali *fixed* (potassium or sodium), or *volatile* (ammonia). Any ammoniacal urine to which a few

* Many such urines are really neutral, as shown by a phenolphthalein solution, though turning red litmus-paper blue, possibly through the release of CO_2 from the bases (F. C. Wood).

† Presumably because of the co-existence of the *acid* monosodium phosphate and the *alkaline* disodium phosphate.

drops of potassium or sodium hydrate solution has been added yields when boiling the odor of ammonia. So also, immersed red litmus-paper resumes its original color when dried in the air.

Physiologic alkalinity.

The "Alkaline Tide."—A physiologic alkaline or neutral reaction often appears two or three hours after a meal especially in vegetarians, in which case the alkali is fixed, not volatile, and the condition unimportant.

Volatile alkali.

Persistent, non-dietetic alkalinity is abnormal, and urine ammoniacal when passed indicates usually some disease of the bladder or prostate with or without renal involvement.

Effect of diet.

An exclusive meat diet tends to produce acidity; an exclusive vegetable diet, alkalinity; but quantitative determination of the acidity or alkalinity is clinically useless.

When encountered.

Excessive Acidity.—This may be encountered in fevers, in acute rheumatism to a marked degree and in gout, diabetes, leukemia, scurvy, and decided cardiac incompensation. *As a rule high acidity and scanty urine go together.*

SPECIFIC GRAVITY.—For ordinary purposes a clinical urinometer of the usual type, properly graduated for definite temperature (usually 17.5°C.) is sufficient. In testing freshly passed urines, for every 3°C. above the standard temperature for the individual instrument one point should be added to the right-hand figure of the specific gravity. If more accurate results are desired or very small quantities of urine are to be dealt with, as in urethral catheterization, the hydrometer of Saxe, one of the pycnometers, or the Westphal balance may be readily obtained.

Instrument.

Temperature.

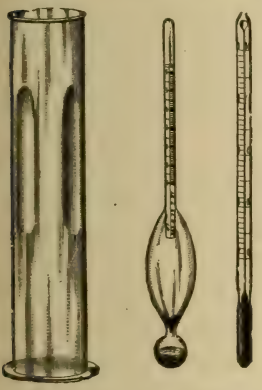


FIG. 60.—Urinometer, thermometer and specific-gravity tube on foot.

Technic.—*In ordinary clinical work the following precautions are necessary: (a) The urine must be allowed to cool or proper allowance made. (b) All air bubbles must be removed with filter-paper. (c) The urinometer must be perfectly dry and (d) introduced with a little spin to prevent it from adhering to the side of the receptacle. (e) The specific gravity should be read from the meniscus or true surface, the observer's eye being at that level; false readings result if one reads from above, because the fluid rises slightly along the urinometer stem. (f) If the amount of urine is small, approximate results may be obtained by diluting sufficiently to float the instrument and multiplying the two right-hand figures of the specific gravity by 2, 3 or 4 according to the amount of distilled water used.*

Details important.

Factors Determining Specific Gravity.—*The normal figures vary from* 1.012 *to* 1.024, *the average lying between* 1.018 *and* 1.022. *Hysteria and the use of diuretic fluids or drugs may reduce it to* 1.002 *and in disease it varies from* 1.002 *to* 1.060.

Wide variations.

In general, *high specific gravity* points to large hemorrhages, profuse perspiration, diarrhea, fever, and *diabetes mellitus.*

Low specific gravity suggests chronic Bright's disease (particularly interstitial nephritis), diabetes insipidus and hysteria.

Exceptions to rule.

As a rule, a low specific gravity accompanies increased excretion, and a high specific gravity, diminished excretion, but there are many exceptions to this rule, many grave diseases being associated with scant urine and deficient elimination of solids, whereas diabetes mellitus may show an enormously increased excretion with the highest readings and diabetes insipidus an increased amount and low specific gravity *with an actual excess of solids in the twenty-four hours' urine.*

URINARY SOLIDS

Organic vs. inorganic.

Normal Constituents.—*Normal urine in the healthy adult of average weight and on a mixed diet should contain about* 60 *grams of solids.* Of these about 35 grams are organic and 25 grams inorganic. By multiplying the two right-hand figures of the specific gravity by 2.2337, one obtains roughly the total urinary solids in 1 liter (1000 c.c.) of urine. [*Example:* Sp. Gr. 1020. $20 \times 2.337 = 46.74$ grams per 1000 c.c.] Of this *about* one-half is urea, which should be separately estimated.

Rough methods.

The principal inorganic substances are: HCl (9.35 grams), phosphoric acid (2.5), sulphuric acid (2.5), nitric acid (1.0), oxalic acid (0.01 to 0.02), sodium (8.0), potassium (3.0), ammonia (0.7), magnesia (0.5), lime (0.3), iron (0.001 to 0.002).

The organic substances are, in grams, urea (30.0), creatinin (1.0), uric acid (0.7), hippuric acid (0.7). Traces of a large number of organic substances, including the purin bodies, make up the balance for the organic group.

The pathologic substances are the albumins, blood, bile pigment, bile acids, indoxyl, acetone, diacetic acid, cystin, leucin, tyrosin, carbohydrates, phenol, creosol, skatol, urobilin, cholesterin, lecithin, diamins, melanin, fats, fatty acids, lactic acid, beta-oxybutyric acid.

Of these, indoxyl, acetone, lactic acid, fatty acids, phenol, creosol, skatol, urobilin and even albuminous bodies may be present in clinically unrecognizable or negligible traces in normal urine.

TOTAL NITROGEN.—A Direct Index of Protein Metabolism.—Before considering urea and uric acid the derivation and importance of the total nitrogen excretion should be considered.

Urinary nitrogen.

While the total nitrogenous output of the human body involves a consideration of that of the feces, lungs and skin as well as the urinary nitrogen, the fact that this last represents, in health and on a mixed diet, from 92 *to* 93 *per cent. of the total makes the urinary examination the primary clinical consideration.*

Variations.

Unfortunately the same ratio does not hold true in disease and in the case of special dietaries, for the reason that in pathologic states the total amount of nitrogen is divided among other nitrogen-containing compounds in various degrees.

The healthy adult on a mixed diet secretes 0.2 gram of nitrogen per

kilo of body weight, viz., from 10 to 15 grams of *nitrogen* per day and under such conditions about 86 per cent. of the urinary nitrogen is urea nitrogen, though this ratio is not absolutely constant. Eight per cent. is derived from ammonia, creatinin, the purin bodies and the pigments, while 6 per cent. represents hippuric acid and unknown substances.

Ratio to body weight.
Urea.

The total nitrogen excretion measures the variations, not merely of nitrogenus intake, but of body waste and hence the metabolic processes.

Nitrogen Retention.—*Though excretion is increased by nitrogenous diet,* an excess may nevertheless be retained in part for several days, a fact which has not yet been explained.

Increased Excretion.—This occurs in acute infectious fevers (excepting acute yellow atrophy of the liver), malignant growths, chronic infections, pernicious anemia, exophthalmic goiter, scorbutus, the leukemias, resorption of exudates, diabetes insipidus, phosphorus poisoning and, indeed, in practically all diseases *associated with marked emaciation, malnutrition or excessive toxic metabolism.*

Diminished Excretion.—This occurs in nephritis, diseases of the liver and in convalescence from acute diseases, though in the first named, if associated with excessive albuminuria, the urine may show an apparent increase if the albumin is not removed. In nephritis especially, the stomach, intestines and skin may take on vicarious activity and the feces will show increased nitrogen content.

Not practicable.

For accurate work, careful measurements of the nitrogen intake and that of the feces as well as the urinary nitrogen is necessary, a time-consuming process illy-adapted to clinical work. (See "Index of Urea Excretion.")

Individual specimens of urine are no more than suggestive with respect to the nitrogen excretion. It must always be determined from the twenty-four hours' total urine. For a full description of all the elaborate chemical processes involved, the reader is referred to the special works dealing with physiological chemistry, but the most commonly used method of quantitative "urinary nitrogen" estimation is given briefly in the following paragraphs.

ESTIMATION OF TOTAL NITROGEN (*Kjeldahl's Method*).—This method depends upon: (*a*) The conversion of urinary nitrogen into ammonia. (*b*) The transformation of ammonia into ammonium sulphate. (*c*) The liberation of the ammonia by sodium hydrate. (*d*) Distillation into a decinormal sulphuric acid solution. (*e*) Determination of the amount of combined ammonia by titration of the decinormal acid solution with a decinormal alkaline solution, using some such indicator as rosalic acid, alizarin red, or methyl orange. (*f*) Estimation of total nitrogen on basis of 0.001401 gram of nitrogen for each cubic centimeter of combined acid and ammonia as indicated by the preceding titration.

Test.—*First Step.*—Using the apparatus shown in Fig. 61 and placing it under a proper fume-conduction hood, one measures into a Kjeldahl flask of 800 c.c. capacity, 5 c.c. of urine; 10 c.c. of concentrated sulphuric acid and 1 gram of copper sulphate.

Avoid fumes.

Oxidation processes.

Second.—*Heat* over low flame until white fumes indicate the giving off of sulphuric acid.

Conversion into ammonium sulphate.

Third.—Add 5 grams of potassium sulphate and, shaking carefully from time to time, boil for from thirty to forty minutes or until the yellow color has entirely given place to bluish green.

Fourth.—Set aside to cool.

Fifth.—Add distilled water sufficient to make total quantity of liquid in flask about 250 c.c.

Prevent "bumping."

Sixth.—Add a few bits of zinc, pumice stone or a little talcum powder to prevent "bumping" during the succeeding stages.

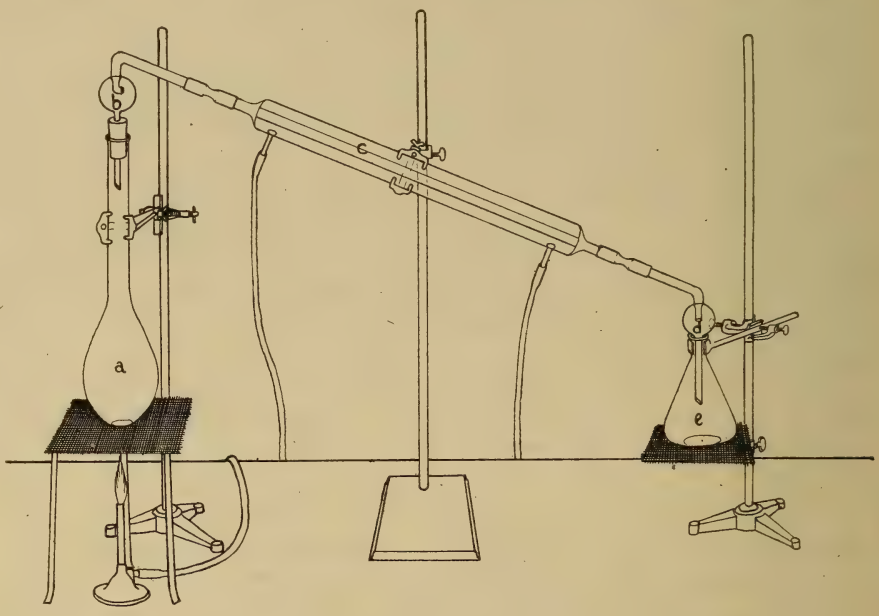


FIG. 61.—Kjeldahl's nitrogen apparatus. (*Webster, "Diagnostic Methods."*)

Liberate ammonia.

Seventh.—Add 50 c.c. of 40 per cent. sodium hydrate, being careful to avoid having it touch neck of flask.

Eighth.—Shake; connect by means of a Fresenius bulb with a Liebig condenser.

Distil and recombine ammonia.

Ninth.—Place in an Erlenmeyer flask 50 c.c. of a decinormal sulphuric acid solution and rapidly connect the outlet tube with the bulb and condenser.

Tenth.—*Heat slowly and carefully* to boiling and maintain slow boil for twenty to thirty minutes or until about 150 c.c. of test mixture have passed over, or until outlet tube fails to color red litmus-paper blue, *i.e.*, until distillation of ammonia is complete.

Ammonia bound by acid.

Eleventh.—Disconnect distilling flask and rinse both connecting and outlet tubes into acid solution.

Titrate to measure amount combined.

Twelfth.—Titrate acid solution with decinormal sodium hydrate, using for an indicator rosalic acid, methyl orange, or alizarin red.

The acid and alkaline solutions being standardized and of equal strength,

the number of cubic centimeters of sodium hydrate solution required indicates the number of uncombined acid molecules, and by deducting this from 50—the number of cubic centimeters of decinormal acid solution originally used—one finds the number representing combined ammonia, each cubic centimeter of which is equivalent to 0.001401 gram of nitrogen.

Hence the difference between the number of cubic centimeters of decinormal sodium hydrate solution used in the last titration and the number of cubic centimeters decinormal acid solutions originally used, multiplied by 0.001401 gram gives the amount of nitrogen in 5 c.c. of the urine, and multiplying by 20 one obtains the total nitrogen percentage and readily calculates it for the mixed twenty-four hours' output of which the original 5 c.c. must be a part.*

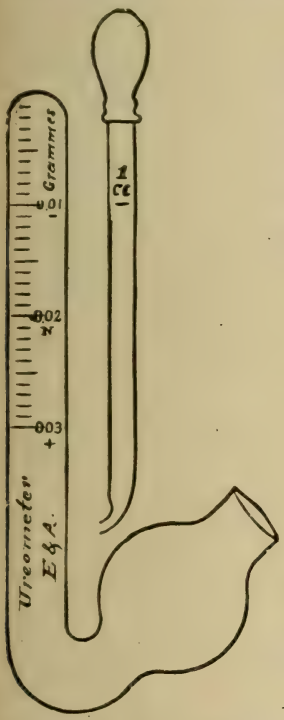


FIG. 62.—The Doremus ureometer.

UREA.—*In the normal individual on a mixed diet the urea output varies from 20 to 35 grams in twenty-four hours. Under such conditions it follows in nearly every particular the laws of total nitrogen excretion already given.*

Periodic variations.

Women excrete less than men, and in vegetarians or poorly fed patients, or those on a milk diet, the amount may not exceed 15 grams a day. Urea nitrogen represents approximately 86 per cent. of the total nitrogen† (protein-free diet 60 per cent., vegetable diet 79 per cent., rich proteid diet 87 per cent.).

Retention Periods.—There are periods of retention lasting several days, and these may be increased by certain drugs, such as salicylic acid, caffein, and quinin. Urea excretion is greatly increased in many conditions associated with marked tissue waste such as fevers, severe advanced diabetes, pernicious anemia and leukemia.

In diseases of the liver the urea excretion is more greatly diminished than is the total nitrogen, leucin and tyrosin being coincidently increased, or, in acute processes, an increased ammonia excretion replaces the leucin and tyrosin.

In view of the recent work of Folin and others, one cannot consider the mere estimation of urea a substitute for total nitrogen determinations in questions of protein metabolism, but it still remains of value in relation to renal disease.

Urea in Bright's Disease.—*In Bright's disease, save the early stage of parenchymatous nephritis, and especially in the interstitial form, urea excretion is usually diminished and also shows marked periodic or irregular variations.*

* Synopsis is taken from the full description contained in Webster's excellent book, "Diagnostic Methods."

† And may reach 92 or 93 per cent.

Sudden drops.

It is supposed that retained urea in such instances is responsible for the periods of diuresis or diarrhea so commonly seen. A sudden drop in urea is often noted in nephritis of all kinds, and is a danger signal that cannot be disregarded.

Early chronic nephritis.

In many of the author's cases presenting intractable neuralgias the attacks coincided with periods of low urea excretion. In others indeterminate but troublesome gastric symptoms, marked psychasthenia and hypochondria and arterial hypertension were found to coincide with the same condition and all these cases were promptly relieved by treatment directed to secure the better elimination of urinary solids and proper dietetic restrictions.

Nephritis *sine albuminuria.*

In eight such instances no structural disease of the kidney was then clinically demonstrable, but all have since shown typical interstitial nephritis.

Estimation important.

The author would strongly differ from, and, indeed, hold the exact reverse of, the statement made by certain writers that in renal disease urea determinations are less important than the finding of casts and albumin.

*With relation to the functional activity of the kidney even the rough estimation of urea, under dietetic control, is one of the simplest and most important of all clinical tests, and the most convenient method of quantitative testing is that of Doremus which is sufficiently accurate for ordinary clinical purposes.**

Instrument.

Description of the Doremus Ureometer.—The Doremus apparatus is much used and consists of a tube carrying a bulb below and so graduated that each division corresponds to 0.001 gram of the urea as represented by the volume of nitrogen evolved at 65°F. There is also a small, curved, nipple-capped pipette holding 1 c.c. of urine.

Test.

The tube is filled with hypobromite solution† and the pipette,‡ filled with urine (*freed from albumin* or *sugar*) up to the cubic centimeter mark, is carefully introduced into the bend as far as it will go, while holding the measuring tube perpendicularly, and the contained urine slowly and completely discharged. After the evolution of gas is complete the number of divisions is read off as milligrams per cubic centimeter or the result multiplied by 100 to obtain the percentage.

A Fascinating and Accurate Procedure.—The method of determination of renal function described below should be known to all students and practitioners of medicine and the author has felt constrained in this instance to give much space to a procedure both time-consuming and exacting.

It will be noted with satisfaction that the simpler phenolsulphonephthalein test (see p. 245) has borne the comparison very well and that *in renal disease the variations from normal are usually violent in the obscurer forms of*

* Of the more elaborate and accurate methods, that of E. K. Marshall is the most recent and most useful. It depends upon the use of the Soya bean ferment *urease*, for the hydrolysis of urea to ammonium carbonate. Though relatively simple, as compared with the older methods, it requires special apparatus, and far more time than the practitioner can afford.

† 100 c.c. of caustic soda dissolved in 250 c.c. of distilled water makes a permanent solution, to 50 c.c. of which 5 c.c. of bromine is added to make test solution when needed.

‡ The instrument can now be obtained with an attached hollow arm with a thumb-screw to hold, measure, and release the amount of urine used.

nephritis. It must be borne in mind that a lack of absolute accuracy in the rough simple older formulæ by no means wholly destroys their clinical value.

This lies in their simplicity and availability, for this test of precision, however fascinating and valuable, is wholly unavailable for the greater number of practitioners of medicine. Like the Wassermann test, it must remain in the hands of the expert and he will apply it chiefly to groups of patients accumulated over several days.

The Index of Urea Excretion.—Most interesting and accurate results are being obtained at present by expert workers in the clinical laboratories, through the use of F. C. McLean's "*index of urea excretion*," which represents a marked improvement of Ambard's process based upon his two "laws," *i.e.*, (1) when the concentration of the urinary urea is constant the amount excreted by the kidneys varies proportionately to the square of the blood-urea concentration; (2) when the blood-urea is constant, renal excretion varies inversely as the square root of the urinary urea concentration. On the basis of these two laws Ambard devised what he calls the "*ureo-secretory-constant*" obtained through a formula known as the "*urea-coefficient.*" His formula (the urea coefficient) is the following:

$$K \text{ (constant)} = \frac{Ur}{\sqrt{D} \times \frac{70}{P} \sqrt{\frac{C}{25}}}$$

Ur = urea per liter of blood.
D = grams of urea in urine during 24 hours.
P = weight of patient in kilos.
C = grams of urea per liter of urine.

It is asserted by him that the "urea-secretory-constant" ("K") varies only from 0.06 to 0.07 in normal individuals, whereas in cases of nephritis the value of "K" is enormously raised, by reason of concentration of urea in the blood as a result of diminished renal elimination.

McLean and Selling greatly increased the accuracy of the process by replacing the hypobromite method of urea estimation used by Ambard, by the exact technic of Folin and of Marshall.

The tedious recourse to logarithms they avoid by the use of a scale rule adapted to the formulas. With relation to the necessity for and value of such a method, and after briefly reviewing the various other tests of renal permeability and adequacy now in use (lactose, phenolsulphonephthalein, etc.) McLean says:*

"In all these tests it has been the urine alone which has been examined, the assumption being made that alterations in the manner or time of elimination are due to kidney factors alone. But this assumption is open to serious objection. The time required for the excretion of considerable amounts of nitrogen or salt, suddenly added to the diet, may, indeed, give some indication of the degree of kidney insufficiency; but the failure to excrete these substances within a specified time is often due to other factors than the ability of the kidneys to carry on their excretory functions. Thus a patient with fever may retain enormous quantities of salt in the body, even though the kidneys are perfectly capable of

*Journ. A. M. A., Vol. LXVI, No. 6, p. 415 (abstract). Italics are the author's.

excreting it, and retention is due to the fact that the excess salt is removed from the blood by the tissues. That, under certain other conditions, a similar retention of nitrogen in the tissues occurs, independently of the ability of the kidneys to excrete nitrogenous substances, is indicated by the recent studies of Foster and Davis.

"Study of the daily nitrogen or salt balance between intake and output is of even less value than study of *suddenly added nitrogen or salt*.

"It has, indeed, long been recognized that determination of the total nitrogen or urea output gives no indication as to the state of the function which provides for the excretion of nitrogen. An individual may excrete normal amounts of urea in the twenty-four hours, and remain in perfect nitrogen equilibrium for weeks or months, and yet be suffering from severe nephritis, with advanced functional changes. Except in the case of those patients with chronic parenchymatous nephritis, who are scarcely able to excrete any salt at all, comparison of the salt output with the intake gives little information. The same objection seems to hold with regard to the use of potassium iodid as a test of the ability of the kidney to excrete salts, since potassium iodid is influenced by the same extrarenal factors which influence sodium chlorid.

"*Studies such as these, of urine alone, can at best give only a general idea of the true renal condition, and can be in no sense a quantitative measurement of the renal activity.*

"Determination of the amount of urea nitrogen, or of total non-protein nitrogen in the blood, however, is not entirely dependable as a measure of excretory activity. The normal concentration of urea in the blood is not fixed, but varies within wide limits in response to variations in nitrogen and fluid intake. As a matter of fact, the larger proportion of individuals with disturbed urea function have concentrations of urea in their blood which are below the upper limits in normal individuals, so that the line of demarcation between normal and abnormal subjects is not sharp.

"*Blood urea figures alone, therefore, may call attention only to the more serious instances of disturbed function. Furthermore, the concentration of urea in the blood even of nephritic individuals is not constant or fixed.* On the contrary, Widal and Javal, in 1904, showed that the concentration of urea in the blood can be made to fluctuate at will in cases of nephritis, by varying the protein intake.

"Either increased urea formation in the body, or disturbance of urea elimination through the kidney, requires a higher level of urea in the blood to provide for a sufficiently rapid rate of excretion to keep the organism in nitrogen equilibrium.

"Changes in the rate of water excretion alter the rate of output of urea, which changes as the concentration of urea in the urine changes, though not directly in proportion to these changes. The rate of elimination also varies according to the weight of the individual, and is constant per kilogram of body weight, when the other conditions remain constant. All of these factors have been considered by Ambard in formulating the laws published by him.

"Data obtained by this method are direct quantitative measurements of one of the most important functions of the kidney, that is, the elimination of nitrogen in the form of urea. Performance of the test requires a shorter time than the use of any test diet, requires fewer analyses, is much more broadly applicable, and may be repeated indefinitely without harm or inconvenience to the patient. *It requires no knowledge of the nitrogen or fluid intake, and is not interfered with by vomiting, refusal of food, diarrhea, delirium or coma.* The blood and urine required for the necessary analyses may be secured within the time of an ordinary consultation, and the analyses can be carried out in any *well-equipped clinical laboratory*. Also of the value of the results obtained, the method is to be preferred to the various modifications of the test diet.

"*The Index of Urea Excretion.*—The index of urea excretion is based on an ideal normal Ambard's coefficient of 0.080. The extremes of variation of Ambard's coefficient in the normal are from about 0.050 to 0.085 or 0.090. Deficiency in urea function results in higher values. These higher values are due to a change in the ratio which exists between the concentration of urea in the blood and the rate of its excretion. Such findings indicate deviation from the normal, but do not measure the extent of the functional change which

is responsible for such deviation. The significance of the abnormal ratio found in any pathologic case, therefore, can be understood only by reference to an arbitrary scale, determined by experience.

"Since it is possible to utilize the laws of Ambard in another form to measure functional changes directly, we have thought it desirable to use a new formula. In the case of the formula as at present used, the ideal normal is determined arbitrarily, by experience, and deviations from the normal in the rate of urea excretion are measured directly in terms of the ideal normal. Ambard has shown experimentally that the rate of excretion of urea under otherwise constant conditions varies directly with the amount of functionating kidney tissue. In observations in human subjects it is not possible to keep conditions constant, but it is possible, by an application of Ambard's laws, to measure the influence of their variations on the rate of excretion of urea. The ideal normal rate of excretion, under the conditions found, at any time, is given a value of 100. The index then expresses, in direct percentage, the rate of excretion found, in terms of the rate of excretion that a normal individual would develop under the same conditions as to concentration in the blood, concentration in the urine, and body weight.

"The index is therefore not a substitution of a new formula, with a new scale of arbitrary values, in place of the Ambard formula, but is a direct measure of an *important excretory function.*

"Since, as we have shown, Ambard's coefficient is not in itself a direct indication of the state of the function which has to do with the excretion of urea, but is merely the ratio between the concentration of urea in the blood and the rate of its excretion, the index expresses facts which can be obtained from Ambard's coefficient only by further calculation.

"Ambard's laws depend on the constant relationship of four variables: (1) the concentration of urea in the blood; (2) the concentration of urea in the urine; (3) the rate of urinary excretion, which together with (2) gives us the rate of urea excretion, and (4) the weight of the individual. We have indicated this constant relationship in our present formula as follows:

$$\text{Index of excretion} = \frac{\text{Rate of urea excretion } \sqrt{\text{Concentration in urine}} \times \text{Constant}}{\text{Weight of individual} \times (\text{Blood urea})^2}$$

"In order to give the index a constant value of 100 for the ideal normal, and in order to determine the numerical value for the constant, certain arbitrary methods of expression of the various factors must be adopted. We have adopted the same methods as those used by Ambard, that is, the expression of rate of urea excretion as grams per twenty-four hours, the concentration in the blood and urine as grams per liter, and the weight of the individual in kilograms. Urea, and not urea nitrogen, is used throughout. It should be distinctly understood that other methods of expression of the various factors would not change their constant relationship, or the index, but would change merely the numerical value of the constant of the formula. When variables which give an Ambard's coefficient of 0.080, the figure chosen as the ideal normal, are substituted in the formula, the constant must have a value of 8.96 to give an index of 100. The mathematical derivation of the new formula is discussed in detail in our previous papers. It reads:

$$\text{Index of Urea Excretion (I)} = \frac{D\sqrt{C} \times 8.96}{Wt \times Ur^2}$$

D = grams urea excreted per 24 hours.
C = grams urea per liter urine.
Ur = grams urea per liter blood.
Wt = body weight of individual, in kilograms.

"The rate of excretion is not actually determined for twenty-four hours, but for a shorter period, usually seventy-two minutes (one-twentieth of twenty-four hours), the calculation being made on a basis of grams per twenty-four hours. *It is important to remember that it is not the actual twenty-four hour excretion of urea that is essential, but the rate of excretion at the time of observation.*

"Substitution in the foregoing formula of values obtained by observation in normal individuals should give values for the index approximating 100. The actual variations in Ambard's laws in normal individuals are magnified by this formula, the variability appearing to be much greater than when the same results are expressed in the form of Ambard's coefficient. When observations are made under the proper conditions, values for the index of less than 80, corresponding to an Ambard's coefficient of 0.090, should never be obtained in normal individuals, and no normal subject should be repeatedly below 100. Values above 100 are the rule, and values up to 180 and 200 may occur. Much higher values may be obtained under certain pathologic conditions, associated with an increase in the rate of urea excretion. It is perhaps unfortunate that such apparently wide divergence occurs among normal persons, and in the same normal individual. This is a result of expressing the laws in this form, and it does not affect the value of the index as a measure of abnormal variations.

Fig. 63.—Ten-inch slide rule (closed) adapted to the calculation of the Index of Urea Excretion.

Example:

Grams urea excreted per 24 hours, $D = 20.0$
Grams urea per liter of urine, $C = 11.0$
Grams urea per liter of blood, $Ur = 0.330$
Body weight, in kilos, $Wt = 55.0$

$$\text{Index} = \frac{D\sqrt{C} \times 8.96}{Wt \times Ur^2} = \frac{20.0\sqrt{11.0} \times 8.96}{55.0 \times (0.330)^2}$$

1. 55.0 on *Wt* scale is set opposite 20.0 on *D* scale, first position.

2. Hair line on runner is moved to 11.0 on *C* scale, second position.

3. Slide is moved so that 3.30 on *Ur* scale is at hair line on runner, third position.

Reading is now made at the arrow which points to scale I and is at 100. Therefore Index, $I = 100$.

"*Methods for Observation.*—Observations can be made at any time, without regard to diet, since application of the laws is independent of the nitrogen intake. It is preferable, though not essential, that the observation should not be made too close to the taking of food, since the most rapid changes in the various factors occur during the height of digestion and absorption. Since observations require neither the introduction of a foreign substance, nor the ingestion of weighed amounts of food, they may be made without regard to vomiting, coma or delirium, and interfere in no way with any plan of treatment that is being carried out. Frequent observations may thus be made without the necessity of interrupting a dietetic treatment with test meals.

"**The essential points in an observation are (1) the collection of urine over a carefully timed period, (2) the collection of blood near the middle of that period, and (3) accurate determination of the urea content of the samples of blood and urine.**

"Our usual routine is as follows: The patient is given from 150 to 200 c.c. of water, in order to insure a free flow of urine. One-half hour later, in order to start with the bladder empty, he voids. The patient is catheterized if necessary, as in urine retention or in the case of coma. The time of voiding is recorded to within one minute. About thirty-six minutes later, from 7 to 10 c.c. of blood are withdrawn from an arm vein into a dry tube containing a few milligrams of potassium oxalate or citrate to prevent clotting. At the end of seventy-two minutes from the first voiding the bladder is again emptied, and the urine carefully measured to within 1 c.c. and used for analysis. The patient takes no food or water during this period. Otherwise no restrictions are placed on him.

"The seventy-two minute period is merely for convenience, as being one twentieth of twenty-four hours. Any other period, preferably not too long, will serve. Ambard preferred a period of thirty-six minutes, but the possibility of error in the collection of the specimen is doubled when the time is halved. If it is desired to carry out a simultaneous phenolsulphonephthalein test, the sixty minute or two hour phthalein period may be used, half of the urine being used for the phenolsulphonephthalein reading, and the other half for the analyses.

"Analyses should be made by the most accurate, convenient and rapid method available for determination of urea in the blood and urine. The method requires more than the usual degree of accuracy in clinical laboratories, as a relatively slight error in analysis may introduce a considerable error into the formula. The urease method, introduced by Marshall, fulfils all of the requirements. This method depends on the breaking down of urea into ammonia and carbon dioxid by the specific enzyme found in the soy bean, and the subsequent determination of the ammonia. We have used the permanent preparation of urease described by Van Slyke and Cullen,* and carry out the determinations in the manner which they prescribe. With careful technic and control of the reagents, this method gives consistently accurate results. It is very simple, very rapid, and requires the minimum of manipulation. *Three analyses are necessary; blood urea, urine urea and urine ammonia.* Urine ammonia is determined in order to correct the urea figure for preformed ammonia, which is also determined in the urea method. Duplicate determinations on blood are advisable, as a higher degree of accuracy is thus obtained. The determinations are run simultaneously in the same apparatus, and require little attention.

TABLE I.—ILLUSTRATION OF INDEPENDENCE OF INDEX AND NITROGEN INTAKE†

Date, 1915	Weight, kg.	Grams nitrogen in 24 hr.					Urea excretion, 72 min. period			
		Intake	Output			Difference	Gm. per liter blood, Ur.	Gm. per liter urine, C.	Gm. per 24 hours, D.	Index of urea excretion I.
			Urine	Stools	Total					
Nov. 5.....	65.0	19.2	16.1	3.15	19.25	−0.05	0.858	9.87	37.5	22.0
Nov. 12.....	64.8	9.6	7.92	1.61	9.53	+0.07	0.499	10.68	12.8	23.2
Nov. 16.....	64.8	9.6	6.6	2.92	9.52	+0.08	0.411	7.06	11.0	24.0
Nov. 30.....	65.4	4.8	3.48	1.37	4.85	−0.05	0.211	4.37	3.32	21.4

"*Calculation of the Index.*—The results from the determinations are calculated to the form in which they are to be expressed in the formula. The weight of the individual, without clothing, should be known to within one kilogram. The four variables are substituted in the formula, and the index calculated. Calculation has been rendered simple by the use of a special calculating device, illustrated in Fig. 63. With this device, which is a modified 10-inch slide-rule, one needs to remember neither the formula nor the mathematical principles involved. With a little practice in reading the scales, calculation becomes purely mechanical, and requires only a few seconds.‡ Without this slide-rule, calculation is most easily performed with an ordinary slide-rule, or by the aid of logarithms.

* Van Slyke, D. D., and Cullen, G. E.: A Permanent Preparation of Urease and Its use in the Determination of Urea, Jour. Biol. Chem., 1914, xix, 211; id., The Journal A. M. A., May 16, 1914, p. 1558. Urease is prepared according to the method of Van Slyke and Cullen by the Arlington Chemical Company of Yonkers, N. Y., and furnished with directions for use.

† Case 1.—C. A. P., man, aged 45. Diagnosis: general arteriosclerosis, chronic interstitial nephritis.

‡ The rule, with directions for use, may be obtained from Keuffel and Esser Co., 127 Fulton Street, New York. The same rule is used for calculation of the chlorid formula described in previous papers.

"*Application of the Index.*—Numerous studies have been made, or are in progress, using the index as a measure of urea function. It is not the purpose of this paper to present cases or conclusions, but certain points regarding the applicability and value of the index are illustrated.

"Table I illustrates the fact that the *index* is independent of nitrogen intake. It also shows clearly that the actual blood urea figure may be very misleading, if taken by itself, as an indication of the ability of the kidneys to excrete nitrogen. In the case illustrated, reduction of nitrogen intake was followed by a corresponding decrease in nitrogen elimination, the patient coming rapidly to equilibrium. The balance between intake and output was maintained at a lower level of urea in the blood, the index remaining constant. Had the blood urea alone been determined at the lowest level, which is lower than the usual normal, one could not have said that there was evidence of impaired renal function. *By application of the index, the actual ability of the kidney to excrete urea is seen to be the same at the lowest level in the blood as at the highest.*

"This case also throws light on the mechanism of so-called nitrogen retention. One can easily see that the increased level of urea in the blood on the higher nitrogen intake is purely compensatory, and is not due to a lack of ability on the part of the kidney to excrete the larger amounts of urea being formed in the body. *The kidney actually does excrete the larger amounts of urea just as well as the smaller amounts, but it requires a higher concentration of urea in the blood to bring this about.* To speak of nitrogen retention on the basis of increased concentration of non-protein nitrogen in the blood is therefore apt to be misleading.

TABLE II.—ILLUSTRATION OF THE COMPARISON BETWEEN THE UREA INDEX AND PERCENTAGE OF PHENOLSULPHONEPHTHALEIN EXCRETED IN TWO HOURS

Subject	Diagnosis	Blood urea, grams per liter	Urea index	Phenolsulphonephthalein, per cent. in two hours
1	Mitral stenosis	0.132	250.0	83.0
2	Parenchymatous nephritis	0.216	139.0	66.0
3	Mitral stenosis	0.196	105.0	65.0
4	Acute nephritis (convalescent)	0.383	92.0	46.0
5	Heart block	0.480	91.0	58.0
4	Acute nephritis (convalescent)	0.395	89.0	51.0
4	Acute nephritis (convalescent)	0.317	68.0	41.0
6	Chronic nephritis	0.388	60.0	45.0
4	Acute nephritis (convalescent)	0.406	56.0	34.0
7	Chronic nephritis	0.446	52.0	41.0
8	Chronic nephritis	0.437	47.0	38.5
7	Chronic nephritis	0.454	44.0	33.0
7	Chronic nephritis	0.546	39.0	42.0
8	Chronic nephritis	0.400	37.0	27.0
4	Acute nephritis (convalescent)	0.634	33.0	25.5
9	Mercuric chlorid poisoning	0.610	19.0	15.0
10	Chronic nephritis	0.488	18.0	25.0
11	Chronic nephritis	0.977	8.1	8.0
11	Chronic nephritis	1.016	7.9	8.5
12	Chronic nephritis	1.850	7.4	7.0
4	Acute nephritis	1.320	7.0	6.5
4	Acute nephritis	0.966	6.1	13.2
13	Chronic nephritis	1.110	4.2	2.5
14	Chronic nephritis	2.147	1.0	Trace
11	Uremia	3.430	0.27	0.0

"*Relation to Phenolsulphonephthalein Output.*—Numerous simultaneous observations on urea and phenolsulphonephthalein elimination make it appear that the mechanism is the same in the two instances. The curves of the percentage of phenolsulphonephthalein excreted in two hours, and of the urea index are parallel and are quite striking. No considerable divergence has been found in any case, with the exception of some patients suffering from heart failure. These subjects were not excreting enough water to carry off the urea formed in the body, but were able to eliminate phenolsulphonephthalein fairly well. In Table II are given figures comparing the results by the two methods. These observations were all in individuals who were excreting water at a rate sufficient to eliminate all the urea being formed in the body. They were either simultaneous observations on phenolsulphonephthalein and urea elimination, or the two observations were made within a short time of each other.

"These figures are in close agreement with those of a similar study published by Widal, Weill and Vallery-Radot, their figures for urea excretion being recalculated in the form of the index of urea excretion. Both studies show a remarkably close parallelism between the index and the percentage of phenolsulphonephthalein excreted in two hours. In the cases with normal or increased excretion of both urea and phenolsulphonephthalein the figures for the index are naturally higher, since the ideal normal index is 100, and higher figures are attained in persons with increased excretion, while the normal phenolsulphonephthalein excretion is about 60 per cent. in two hours, and 100 per cent. can be approached by individuals with increased excretion, but not exceeded. In the lower figures the two tests show in practically every instance the same diminution in rate of excretion of urea and of phenolsulphonephthalein. Both show the failure of the blood urea figure alone to indicate the degree of disturbance in urea elimination, since many cases show blood urea figures below the upper limits in normals, though both the index and phenolsulphonephthalein figures are low.

"These findings confirm the value of the use of phenolsulphonephthalein as a clinical test of the ability of the kidneys to excrete substances belonging in the same class with urea. While a rational basis for the use of phenolsulphonephthalein is thus given, certain factors make the direct determination of urea function of still more value than the phenolsulphonephthalein. In the first place, it is a direct determination of a normal function, of which the phenolsulphonephthalein test is an indirect determination. Interpretation of possible discrepancies between the two methods must recognize this fact. For study of the effects of treatment in a given case, the index is certainly the more delicate indicator, and the line of demarcation between normal and abnormal cases is sharper than in the case of phenolsulphonephthalein. The index has an added advantage in that, as it tends to approach zero, smaller differences are of greater import, and are more easily determined than in the case of phenolsulphonephthalein. Thus the difference between 3 and 6 per cent. phenolsulphonephthalein excreted in two hours is difficult to determine colorimetrically, while it is a very considerable difference in the case of the index. From the standpoint of determining whether the condition is stationary or progressive, the index is therefore much more delicate than determination of the small amounts of phenolsulphonephthalein excreted in cases with advanced impairment of function.

"In addition to the index itself, the analyses necessary in determining it are of direct value. We have learned to attach a certain amount of import to the blood urea figure alone. The index, therefore, adds enormously to the value of an already recognized method, with the addition of practically no technical details. The determination of urea in the urine, the only laboratory work required in addition to the blood urea, demands only a moment's extra time.

"*Neither the phenolsulphonephthalein nor the index, in itself, furnishes positive information as to the cause of the impairment of function.* Indirect information is, however, furnished by the determinations made in arriving at the index. A low index, associated with a diminished excretion of urine, with a high urea content, suggests passive congestion of the kidneys. The same index, occurring with the excretion of normal or increased amounts of urine, with a low urea content, may be characteristic of

certain forms of nephritis. Thus additional information is obtained in the determination of the index.

"*For the present we prefer to study cases of nephritis with both the index and the phenolsulphonephthalein test. Each serves as a check on the other, and the maximum of information is obtained in the simplest way possible.*

"*Practical Value of the Method.*—Applicability of the method as a guide to diagnosis, prognosis and treatment will depend on further clinical reports, but certain facts have become evident:

"An index below 80 is to be considered as abnormal, though not necessarily seriously so. In renal disease an index below 50 is indicative of a considerable degree of impairment of functional ability. The amount of damage to the kidneys, it is believed, is increasingly greater as the index is lower, and tends to approach zero. *But a low index may be only* temporary, as in the passive congestion of heart failure or in acute nephritis, and may return to normal on improvement of the condition which is responsible for impaired function. The actual figure obtained for the index should be of value in prognosis in renal disease, though renal function alone is often not the determining factor in prognosis. In our experience a low index has at times been the first indication of a serious kidney involvement. When the condition is stationary, life may be maintained for some time with a low index. For example, we have seen patients with chronic nephritis survive for several months with an index of from 5 to 8, and we have seen recovery from acute nephritis after a number of weeks, during which time we have obtained the same figures. In another instance, the patient survived for about a month after the index was as low as 1.2. Such figures, in chronic disease, certainly determine a grave prognosis. But, unless the condition is known to be progressive, it is difficult to give a prognosis with regard to the duration of life. Other aspects of the disease must, of course, be considered in attempting a prognosis.

"*Progressive decrease in the index, usually associated with a corresponding increase in the concentration of urea in the blood, is of serious import.* Two cases have recently been under observation in which such a fall in the index was followed by death within a short time. In one case the fall in the index was the first indication of impending uremia, although the first symptoms did not occur until ten days after the discovery of a change in the index.

"Use of the index as a guide to dietetic or other treatment of nephritis must depend on further studies. It is doubtful whether the diminished urea content of the blood which follows a diminished nitrogen intake has any direct beneficial effect. Whether a long-continued rest of the urea function will improve that function remains to be demonstrated. On general principles, an impaired function should not be overtaxed, and a restricted protein intake should be advised in cases with a markedly lowered index of excretion. But brutal restriction of nitrogen intake to below the nitrogen requirement of the body does not seem to be indicated in cases capable of excreting normal amounts of urea, though the blood nitrogen may be high and the index low.

"The influence of diuretic drugs also requires further study. Here the findings will depend on the type of case studied, and it is necessary to have a satisfactory method for grouping them. Study of diuretic drugs, involving also their effect on chlorid function, is in progress.

"*Application of the index is seriously interfered with when water excretion is greatly diminished, as in passive congestion or in some forms of nephritis.* The laws of urea excretion depend on a sufficiently rapid rate of urine excretion, and they fail to apply when the water output is greatly diminished. We make it a general rule not to attempt to apply the index when a rate of urinary outflow equal to at least 500 c.c. in twenty-four hours cannot be attained. Otherwise there are practically no limitations as to its applicability."

URIC ACID (0.2 to 1.0 grams daily).—This is a body of that "purin group" which also includes guanin, adenin, xanthin and hypoxanthin, and the methyl xanthins of tea and coffee, caffein, theophyllin and theobromin.

Exogenous purins are those derived from ingested foodstuffs, especially such as are rich in nuclein. Such are thymus gland (sweetbreads), liver, spleen, pancreas, brain, kidney, and the roe of fish. Rich in purins.

Endogenous purins are products of the cell metabolism within the body.

Nuclein or *nucleo-protein* is the basis for all the purin bodies of the urine, 90 *per cent. of the total being uric acid.*

A purin-rich diet greatly increases the output of exogenous uric acid.

A diet of milk, cheese, eggs, fat and vegetables keeps the purin bodies at a minimum though no adequate dietary can be wholly free from nuclein. Purin-poor.

Uric Acid Sediment.—*As a sediment, uric acid is easily recognized by microscopic examination or by the deposit of a macroscopic substance resembling cayenne pepper.* Cayenne-pepper sediment.

The urinary conditions favoring a uric acid deposit are: (*a*) *Concentrated urine.* (*b*) *High acidity.* (*c*) *Deficiency of salts and pigment.* (*d*) *Excess of the uric acid.*

The amount of uric acid in *centigrams*, contained in each liter of the given urine, may be roughly *approximated* by multiplying the last two numbers of the specific gravity by 2, but as a matter of fact, neither the ratio of uric acid to urea nor to total nitrogen is constant. Any sediment may be tested for uric acid by heating with a drop of dilute nitric acid upon a porcelain plate. *Upon evaporation a reddish residue appears which strikes a beautiful deep red with dilute ammonia.* Rough estimate.

Quantitative Test.—There is no accurate simple test and the complicated exact tests (Hopkins-Wörner, etc.) are not adapted to the practitioner's use in view of the slight clinical value of the procedure. But one *approximate* method will be given and it, like other quantitative tests, must be applied to a portion of the twenty-four hours' urine to show an actual excess of uric acid.

Heintz's Test.—To 200 c.c. of clear urine add 10 c.c. of C. P. HCl and mix thoroughly. After twenty-four hours filter through a dry filter-paper *of known weight.* Dry and reweigh. The difference in weights represents the amount of uric acid in each 200 c.c. of urine.

Clinical Importance.—*The uric acid sediment is not a clinical index of uric acid excretion, and an actual excess of uric acid is much oftener due to a diet rich in nuclein than to other causes.*

In chronic gout there is a diminished excretion of both exogenous and endogenous purins tending to produce an accumulation of these bodies in the blood, and during, and for several days preceding, an acute attack a more or less decided retention of the latter group occurs.* Chronic gout.

During an acute attack the excretion rises rapidly only to fall far below normal a few days later. Acute gout.

In leukemia the amount of the daily uric acid excretion may be treble the normal and "uric acid showers" may at intervals replace sugar in certain cases of diabetes.

* Brugsch and Schittenhelm have found uric acid in the blood of gouty subjects even when the diet was purin-free.

Old theories upset.

Lithemia.—*Certain cases of so-called "lithemia" behave like and probably represent irregular gout, but most of the cases so named are examples of faulty diet or mere chronic indigestion.**

Nevertheless, the recent work upon the physiological chemistry of gout gives to uric acid a renewed importance and indicates that such a condition as the "uric acid diathesis" apart from frank clinical gout may deserve consideration.

On the other hand, it has left us once more in the dark as to the causes of uric acid deposits in the joints of gouty subjects inasmuch as late researches show that in gout the blood is neither saturated with uric acid nor does it lose its alkalinity and thus throw this substance out of solution.

It would seem that in certain individuals the transformation of purin bases into uric acid and the oxidation of uric acid itself are alike deficient.

Ferment deficiency.

Gout and its congeners would seem, therefore, to represent an acquired or congenital ferment insufficiency combined perhaps with a tendency to diminished renal permeability for uric acid especially marked in certain chronic arthritic cases.

The true cause of gout remains obscure. The presence of an uric acid excess in itself is certainly not the determining factor.

URINARY CHLORIDES (10 to 15 grams daily, usual proportion to urea is as 1 : 2).

Rough Test.—Filter the urine if it be not perfectly clear; remove albumin by boiling if necessary (a *trace* does not matter). Prepare a solution of silver nitrate and distilled water (silver, 1 part; distilled water, 8 parts) Add this, drop by drop, to a portion of the suspected urine, *which has been treated with a few drops of nitric acid.*

A curdy precipitate indicates normal chlorides. Milky turbidity or simple cloudiness shows a marked reduction of the chlorides. No precipitate indicates absence of chlorides.

A simple quantitative test may be used if desired, its errors being so slight as not to affect its *clinical* value.

Mohr's Test.—10 c.c. of urine freed from albumin is diluted with 100 c.c. of water, a few drops of potassium chromate solution added (enough to produce a yellow color) and the whole placed in a porcelain capsule. This is then titrated with a standard silver nitrate solution ($AgNO_3$ C. P. 29.06 grams to the liter), 1 c.c. of which should precipitate 1 cg. of sodium chloride; a permanent and diffused orange color ends the titration and multiplying the number of cubic centimeters used by 0.01 gives the amount of chlorides present in 10 c.c. of urine.

The Centrifugal Estimation.—Purdy's method is simple and clinically sufficient.

Test.—Fill one of the graduated tubes with urine to the 10 c.c. mark, add

* It is but a few years since "lithemia" was as common a diagnosis as "heart failure," "liver trouble" and the like.

It was not only a convenient cloak for ignorance but highly popular with patients because of its kinship with that most aristocratic of ailments, "gout."

15 drops of nitric acid to prevent precipitation of the phosphates (more if the specific gravity be high), add (1 to 8) nitrate of silver solution, mix thoroughly, and centrifugalize at high speed for fifteen minutes; normal bulk percentage reading is from 10 to 12 and each division represents 0.123 by weight.

Clinical Application.—*The outpouring of either a serous or fibrinous exudate of slight importance in acute disease may be associated with a marked diminution or total absence of the urinary chlorides. The reappearance of the lost chlorides is one of the clinical evidences of resolution in pneumonia or of the reabsorption of an exudate or transudate.*

Of slight importance.

The absence or marked diminution of chlorides in lobar pneumonia is not *pathognomonic* of that condition even in the absence of meningitis or effusions into the great lymph spaces, inasmuch as the same diminution may occur in many other *sthenic* fevers though usually to a lesser degree.

Lobar pneumonia.

Meningitis.

Their estimation is sometimes of slight value in making a differential diagnosis between meningitis and typhoid fever,* they being markedly reduced in the former, and little affected in the latter disease. They are also greatly reduced in acute rheumatism and their sudden entire disappearance without an extension of the joint involvement suggests pericarditis with effusion.

Acute rheumatism.

In normal individuals a rapid increase of body weight follows the ingestion and retention of the chlorides in quantity and a reduction of weight follows if the substance be withdrawn.

Weight loss or gain.

Chloride excretion is more or less decidedly diminished in various types of renal disease, in most wasting diseases, in starvation or a milk diet, in cancer of the stomach with obstruction, in severe vomiting and in diarrhea.

Renal disease.

Relation to Edema and Albuminuria.—*It is now believed that the albuminuria and edema of Bright's disease is in some measure due to the retention of chlorides in the tissues.*

Water retention is favored by the chemical combinations formed by the chlorides, the relative impermeability of the kidney and a poor circulation, hence it is urged that in both incompensated heart disease and nephritis the amount of sodium chloride ingested should be reduced to a minimum if edema is present. The author believes this to be good advice with respect to persistent edema of the renal or cardiorenal type but would not advise or practice it as a routine procedure or continue it radically over long periods.

THE PHOSPHATES (normal excretion 2.5 to 3 grams).—Urinary phosphoric acid is met with largely in combination with potassium, sodium, and ammonium (⅔), to a less extent with calcium and magnesium. It is derived to some extent from tissue and food nucleins and is increased under a vegetable diet or a *rich* proteid intake.

The term "*phosphaturia*" is probably a misnomer representing merely a condition of reduced urinary acidity which leads to the constant or almost constant presence of the phosphates as a precipitate. It is met with chiefly

Phosphaturia.

* E. S. Wood.

in cases of asthenia or temporarily in nervous overstrain, and may accompany hyperchlorhydria.

A marked increase occurs in severe anemias, leukemia and wasting diseases in general.

A marked decrease is observed in most febrile diseases, with high temperature, acute and chronic rheumatism, acute yellow atrophy of the liver, lead or copper poisoning, Addison's disease and hysteria.

Phosphates are readily recognized by the methods mentioned elsewhere and their quantitative determination is of too little importance to be described here. It should be remembered that a phosphatic sediment occurs frequently in normal individuals and many "*phosphaturias*" are little more than the result of morbid introspection and too careful attention to the appearance of the excreted urine. In ammoniacal urines both the fixed and the volatile alkali combine to form the characteristic crystals so frequently found in the precipitate.

Two varieties.

THE SULPHATES (normal excretion 1.5 to 3 grams).—These exist either as preformed or as ethereal or conjugate sulphates. The former run nearly parallel with urea, while the latter follow indoxyl so closely as to justify but slight attention here. These latter are, however, increased by the ingestion of drugs such as creosol or phenol and phosphorus and occasionally without an indoxyl excess.

THE OXALATES.—"Oxaluria" *is another condition which modern investigation has shelved.*

The diagnosis was based chiefly upon the presence of calcium oxalate crystals in quantity in the urinary sediment. These occur usually as a result of gastro-intestinal disturbances to which the symptoms formerly attributed to them are more properly ascribed. The relation of the crystals to the formation of calculi and their suggestion of disturbed metabolism are the only clinical features of importance.

Clinical value slight.

Dunlop has shown that their presence indicates about 25 mg. of oxalic acid to 1000 c.c. of urine, but this amount does not exceed the maximum normal. Very frequently they are associated with an increase of indoxyl and sometimes with increased ethereal sulphates without increase of indoxyl.

IRON.—This exists in the urine to the amount of 1 mg. but its estimation has no clinical value.

AMMONIA (normal 0.6 to 1.2).—The ratio of nitrogen derived from urinary ammonia to total nitrogen is about 5 to 100 and the variation of the two substances under dietetic influence is almost parallel.

In cirrhosis of the liver ammonia is usually increased as also in hyperchlorhydria, pyloric stenosis and acute gastro-enteritis. It is diminished slightly upon a vegetable diet and the administration of fat not only increases it, but causes a corresponding but later increase in the urinary acetone, and such urine may show beta-oxybutyric and diacetic acids. The methods of estimation are too complicated and time-consuming and the results too attenuated to be of clinical use and they are therefore omitted.

Of slight importance clinically.

ALBUMINURIA

Albumin in Normal Urine.—*Normal urine does not contain a sufficient amount of serum-albumin to respond to certain approved clinical tests if these are properly made; therefore a reaction under such conditions is pathologic.*

Transient Albuminurias

Temporary albuminuria due to exposure to extreme cold, to excessive exertion, profound fatigue and other like causes doubtless occurs, especially in individuals of the asthenic type.

Such urines are *not* normal but temporarily abnormal.

"Accidental albuminuria."

Derived Albumin.—*On the other hand, the presence of albumin does not of itself prove a nephritis, as both pus and blood yield a portion of their albumin to the urine and any hemorrhagic or suppurative lesion within or communicating with the urinary tract may cause albuminuria.*

Misleading findings.

Occasional Absence in Nephritis.—*The absence of albumin in a given specimen does not exclude chronic interstitial nephritis, as in that disease the urine may be albumin-free for long periods, appear only at certain times of the day or occur only in definite relation to some exertion, mental or physical, or a dietetic indiscretion.*

Vital factors.

The mere detection of albumin is but a part of the diagnosis, the arterial tension, tests of renal permeability, and recourse to the microscope playing the larger part.

One of the commonest sources of error arises from contamination of the specimen by leucorrheal discharge or the pus and blood of urethritis, less commonly from fistulous openings draining abscesses of the surrounding structures.

Varieties.—*The term "albumin" means in its clinical sense, serum-albumin usually associated with serum globulin, which acts the same, has essentially the same significance and need not be separately sought.*

NUCLEO-ALBUMIN.—This substance frequently obscures some of the more delicate tests. It is increased after overexertion, in actual inflammation of the urinary tract, especially in purulent accumulations, in leukemia, jaundice and in acute infections with marked toxemia.

Test.—*Nucleo-albumin* is readily detected by diluting the urine with three times its bulk of water, rendering it strongly acid with acetic acid and setting it aside until the substance forms a cloud. It may produce confusion if the physician is satisfied with the heat test for albumin and fails to use the acid layer test as well. In the latter the nucleo-albumin ring lies well above (1 cm.) the zone of contact.

A clinical nuisance

ALBUMOSES.—These, chiefly the secondary (deutero-albumoses), appear in the urine and have some clinical importance because of their association with suppurative processes outside the urinary tract. Their presence, for example, in a pleurisy would *suggest* empyema; in meningitis, a septic rather than a tuberculous process; in a severe intestinal lesion, dysentery or typhoid rather than tuberculous ulceration; in hepatic disease, abscess or suppurative cholecystitis; in tumors, malignant ulceration.

Septic processes.

Combined with a leucocytosis, albumosuria speaks positively for infection

Practical value of test.

by a pyogenic organism or the actual presence of a purulent accumulation or focus.

Albumose appears, however, in many non-septic conditions both febrile and afebrile.

Test.—Secondary albumoses are precipitated by acetic acid saturated with sodium chloride or the biuret *reaction* may be obtained as follows: Remove all albumin by treating the urine with sodium acetate and then with ferric chloride until it produces a deep red color, neutralize carefully with sodium hydrate, boil and filter. Repeat if a potassium ferrocyanide test applied to the fluid shows albumin. Nucleo-albumin and mucin may also need to be first removed if present in quantity. This is readily accomplished by strongly acidulating with acetic acid and filtering after several hours. 50 c.c. of albumin-free urine are then acidulated with HCl (5 c.c.) and phosphotungstic acid solution added until no further precipitate is obtained, gentle heat is then applied until the precipitate shrinks and collects on the bottom of the receptacle. After decanting, the precipitate is repeatedly washed, dilute sodium hydrate added and the solution warmed until the deep blue becomes a light yellow. After cooling, a dilute solution of copper sulphate is run down the side of the tube or beaker and a *rose color* indicates albumose. Practically all cases of so-called *peptonuria* are albumosurias and their significance is exactly the same.

THE BENCE-JONES PROTEIN.—A Clinical Curiosity.—This was formerly supposed to be an albumose, but is as yet a doubtful body. It appears in cases of osteomalacia and multiple myeloma and constitutes one of the clinical curiosities. Its most characteristic feature is its behavior when heat is applied to urine containing it. Commencing as an opacity at 50°C., becomes precipitated at about 58°C., and increases up to 70°C. when it begins to disappear and at boiling point has returned to its first opalescence.

Osteomalacia and myeloma.

The presence of the Bence-Jones protein in the urine indicates, almost invariably, multiple myeloma, characterized by pains in the bones, deformities of those of the trunk and the Bence-Jones protein.

Without discussing the theories relating to the causation of albuminuria, one may say that under certain conditions any or all of the protein bodies found in blood plasma may appear in the urine.

THE SIGNIFICANCE OF ALBUMINURIA.—*Osler aptly covered the whole field in saying, "the presence of albumin in the urine in any form and under any circumstances may be regarded as indicative of changes in the renal or glomerular epithelium, a change, however, which may be transient, slight and unimportant, depending upon the variations in the circulation or upon irritating substances taken with the food, or temporarily present, as in febrile states."*

Altered renal or glomerular epithelium.

We know that transient albuminuria may follow emotional disturbances, violent exhausting exercise, cold baths, the ingestion of excessive amounts of nitrogenous foods or infections of various kinds.

Multitude of etiologic factors.

In young persons it may be irregularly intermittent or occur at definite intervals (*cyclical*), and wholly lack the general and circulatory symptoms of Bright's disease.

Traces of albumin (0.5 to 1 per mille) constitute the "*albuminuria minima*" so frequently discovered in the course of life insurance examinations, especially during severely cold weather. Cold weather cases.

Such cases may entirely clear up, but a considerable proportion later become fully developed cases of chronic nephritis.

Even the true nephritis albuminurias may be regularly or irregularly intermittent and often bear a definite relation to the time of day, exercise and meals. In most cases of this kind the first urine passed in the morning is free from albumin, but thereafter during the day it is present, and increases as evening approaches. A source of serious errors.

Muscular fatigue increases it, though regulated moderate exercise frequently diminishes the albumin output.

Cases in which it is affected by mere changes of posture are known as "*orthostatic albuminurias.*"

In albuminuric children and young adults a delicate bony framework, weak circulation and tendency to vasomotor instability and low arterial tension frequently coexist with impaired general nutrition and, in the author's opinion, the entire clinical picture most often resembles the *asthenia universalis congenita* of Stiller which carries with it the visceroptotic habitus so favorable to circulatory renal disturbance.

In every case the adequacy of the kidney, as proven by the excretion of solids, special tests, the absence of true casts and of secondary vascular changes, must be the guide to prognosis, and inquiry as to past infections, such as scarlatina and diphtheria, is often illuminating.

Unrecognized Chronic Infections.—*Every physician who has followed the recent investigations relative to chronic infections of the tonsils, accessory nasal sinuses, teeth and prostate, or who fully appreciates the frequency of unrecognized tuberculosis and lues, must realize the frequency with which an albuminuria or an actual nephritis rests upon such an etiologic basis.* Septic foci.

TESTS FOR ALBUMIN.—As a result of an extended series of tests undertaken by Dr. P. A. Hoff and the author, covering both normal and abnormal urines, he is convinced that for the general practitioner the three best tests are: (*a*) *the-heat-and-nitric-acid-procedure;* (*b*) *the-heat-test-with-acetic-acid-and-brine;* and (*c*) *the-nitric-acid-layer-test which is practical and accurate if performed with care and a proper knowledge of its disturbing factors, and should be applied invariably as a check.* Three best tests.

Every other test used in our experiments gave confusing results when it became a question of determining the small traces of albumin yielding a haze but no definite precipitate.

In this connection several other tests are mentioned because of their general acceptance by physicians, but are not recommended.

General Considerations.—*The essentials of a clinical test for albumin are:* (1) *Simplicity.* (2) *A reasonable degree of delicacy.* (3) *Decisiveness.*

Simplicity reduces careless or imperfect work to a minimum; if one exceeds a reasonable *delicacy* he finds disturbing reactions in almost every urine, and, finally, a test must be so *decisive* as to make unnecessary the more complicated

tests for disturbing urinary proteids and permit a positive and convincing opinion.

Faulty Technic.—*The-heat-and-nitric-acid test, as usually applied, does not fulfill these requirements.*

Mere boiling.

Merely boiling the urine coagulates both serum-albumin and nucleo-albumin, and precipitates the phosphates.

Nitric acid alone.

Nitric acid used alone and diffused through the urine coagulates serum-albumin, the primary and secondary albumoses, nucleo-albumin and the resins, and, in concentrated urines, precipitates urates if these be present in excess.

Layer test.

The careless application of the layer test (Heller's test) may result in the confusion of the low-lying or, rather, *junction-point* band of albumin with the higher stratum of nucleo-albumin or resins, or the albumin ring may be obscured in dark-colored urines by the pigment developed at the point of contact.

Clouded Urine.—*A failure to thoroughly clear the urine before examination is a frequent source of error.*

If ordinary filtration fails, this is best accomplished by the addition of magnesium oxide or sawdust, *not lead acetate*, and passing the urine through several thicknesses of filter-paper. Bacterial urine always requires such treatment.

To clear resins.

Resins may be present and cause opacity if turpentine, oil of sandalwood, copaiba and the like are taken, and bile pigment may precipitate in icteric urines, but both disappear on shaking up with alcohol.

Dark background indispensable.

Proper Background.—*Another astonishingly frequent and unpardonable source of error lies in the failure to hold the tube toward the light, but always against a background, such as the coat sleeve or a book.*

Clear specimen.

The Proper Application of the Heat-and-nitric-acid Test.—(*a*) *Clear the specimen and set aside a portion of the urine for use as a control and for repetitions of the test.*

Avoid urates and nucleo-albumin.

(*b*) *If the urine be extremely concentrated, dilute it with its own bulk of water; if of low specific gravity, add one-fifth its volume of a saturated solution of sodium chloride.*

Use control tube.

(*c*) *Then fill two perfectly clean and clear test-tubes two-thirds full of the urine and set one aside for subsequent comparison of transparency.*

Boil upper stratum.

(*d*) *Boil the upper portion of the liquid in the remaining tube and directly after boiling add* 1 *or* 2 *drops of strong nitric acid and* 1 *or* 2 *drops more if no persistent cloudiness or precipitation appears. Any such persistent cloudiness or precipitate is serum albumin.*

Add nitric acid to 4 minims.

Stop short.

Caution.—*Do not add acid before boiling nor boil again after the addition of the acid.**

An excellent method.

The Nitric Acid Contact Test (*Heller's Test*).—*Place in a conic glass or test-tube a dram or two of pure nitric acid, and, using a pipette, allow the urine to flow gently down the side of the inclined glass and upon the surface of the acid.*

Alternative procedure.

* The latter procedure especially will often cause the conversion of large amounts of albumin into a soluble acid-albumin as is readily proven by any one interested.

If the mouth of the pipette be placed against the side of the glass, *just above the level of the acid*, and a very gentle flow established, the result is extremely clean-cut and beautiful.* *The tube or glass should be set aside, as two or three minutes may be required to develop the reaction.*

The albumin ring.

Albumin, if present, appears as a band or ring at the junction of the two fluids and is more or less distinct in proportion to the amount of albumin present.

Urinary pigments.

Urinary coloring matters appear near the surface of the acid, but usually at a point just below the albumin ring, if the test has been properly made.

FIG. 64.—Horismascope: adding the reagent. (*Todd.*)

Nucleo-albumin.

If within ten minutes there appears another ring resembling albumin, but at a distinctly higher level (0.5 to 1 *cm.*), *one may assume it to be nucleo-albumin. A relative excess of uric acid or acid urates may produce an obviously crystalline deposit if the specimen be overconcentrated or if the individual from whom the urine was received has fever.*†

Urate ring.

Mucin.

Mucin, even if present in excess, is usually dissolved promptly and is not a disturbing factor.

The heat-and-nitric-acid and the cold-nitric-acid tests *as thus applied* are excellent and quite sufficient for ordinary purposes.

If space permitted, much that is of interest might be said of the *urinary chromogens and pigments*. If one wishes to bring out certain of the coloring matter, the urine should be placed in the glass and a considerable quantity of the acid poured rather briskly down the side. When this is done, the colors are prominently displayed and the albumin ring, if present, is raised to a level that nearly corresponds to that of uric acid and acid urates, when the test is applied by the first method.

Robin's method.

Relative value of test.

Of the two, the heat-and-nitric-acid test is the more definite and positive. As too often carried out, they are most misleading and fallible.

To be shunned.

Faulty Older Method.—It will be remembered that this method consisted in adding nitric acid, drop by drop, *while boiling the urine.*

The following errors may result from such methods: (*a*) *A small amount*

* The use of the "horismascope" (see Fig. 64) yields even better results.

† According to C. E. Simon, the subsequent appearance of a film-like hoarfrost on the sides of the glass indicates that about 25 grams of urea are contained in a liter of the urine under examination. Similarly spangles of this urea nitrate point to 45 grams, and the separation of a dense mass to 50 or more grams to the liter. It must be remembered that such deductions are not of much value unless the specimen be a part of the twenty-four hours, urine.

Soluble acid-albumin.

*of albumin boiled with an excess of acid might form a soluble acid-albumin and escape detection.**

Soluble alkali-albuminate.

(b) In an alkaline or neutral urine with phosphates present in excess, a failure to acidify the specimen might result in the disappearance of any albumin present through the formation of a soluble alkali-albuminate, and neither acid nor alkali albuminates are precipitated by subsequent boiling.

Furthermore, according to Purdy, mucin, globulin and albumoses are precipitated by this method.

The Potassium Ferrocyanide Test.—Into a clean test-tube pour a dram or two of acetic acid (50 per cent)., to this add twice its volume of an aqueous solution of potassium ferrocyanide (1–20). Shake the mixture and overlay with the suspected urine, as is done in the nitric acid contact test. Albumin, if present, appears at once as a band at the junction of the two fluids. Albumoses may give a cloudiness, and hence the urine should always be heated after the ring is obtained, and its behavior noted. *The full amount of acetic acid must be added.*

Disadvantages.—It precipitates albumoses and nucleo-albumin.

Robert's test solution (HNO_3 1 part, saturated solution of magnesium sulphate, 5 parts) may be used as in Heller's test, but is, if anything, too delicate and subject to the same elements of error.

Spiegler's Test.—This is too delicate for clinical work, showing one part of albumin in 250,000 of urine containing abundant chlorides.

The trichloracetic acid test can be made by dropping a crystal into the urine contained in a small test-tube. It is extremely delicate, partially precipitates albumose and, in concentrated urines, urates, as a distinct *upper* ring. These three tests may show albumin in all urines, or so nearly all as to rob the finding of a mere *trace* of albumin of all clinical significance. Nevertheless the author has seldom found a *distinct ring* with trichloracetic acid without confirmatory microscopic findings.

FIG. 65. Esbach's albuminometer.

QUANTITATIVE TESTS FOR ALBUMIN.—Two simple tests suffice for the quantitative estimation of albumin. The first demands the use of *Esbach's albuminometer;* the second requires *a centrifuge.* Both tests are extremely simple in execution, and as no physician's office is complete without a centrifuge and as the Esbach tube is inexpensive and easily obtained, no hardship is involved in their application. Esbach's albuminometer is merely a glass tube graduated and lettered as shown above. This tube is filled to the letter U with urine, if necessary previously diluted until its specific gravity is 1008 or lower, and a solution of picric acid (picric acid, 10; crystalline citric acid,

Esbach's test.

* The author recently saw a case in which a specimen of urine containing 0.25 per cent. of albumin was reported normal after the application of this test in the faulty manner described above.

20; distilled water, 1000) is added until the level of the liquid has reached the letter R. A rubber stopper is then inserted, the tube is inverted several times to thoroughly mix the urine and test solution, and is then set aside for from twenty-four to forty-eight hours. The albumin is precipitated, and its height, measured by the numerals upon the scale, represents in grams the amount of albumin present in 1 liter (1000 c.c.) of the urine. *This method is sufficiently accurate for practical purposes (although peptones, mucins, etc., if present, are precipitated together with the albumin), but is slow and far inferior to Purdy's direct centrifugal method.*

Centrifugal Method.—Fill the graduated tube supplied with the centrifuge to the mark 10 c.c. Add 3 c.c. of a 10 per cent. solution of potassium ferrocyanide and 2 c.c. of acetic acid (50 per cent.). Mix thoroughly, set aside for ten minutes, and *thoroughly revolve in the centrifuge until the supernatant fluid is clear and the albumin evenly deposited* at the bottom of the tube. Each mark represents $\frac{1}{10}$ c.c. and corresponds to a bulk measure of 1 per cent. or $\frac{1}{60}$ of 1 per cent. by weight (Ogden).* The old-fashioned methods

Best clinical method.

FIG. 66.—Electric centrifuge.

FIG. 67.—Purdy's graduated tubes for the centrifuge.

of estimation by boiling the urine in a test-tube, or by judging the amount by the depth of the ring produced by the contact test with nitric acid are useful, but not sufficiently accurate and introduce too much of the personal equation. On the other hand, the very exact methods of the chemical laboratory are laborious, and demand more time than the busy practitioner can give.

* If urates are present in excess the supernatant fluid may be replaced by boiling water after centrifugalization and the process continued a short time longer.

Rough estimation.

E. S. Wood's Approximation Method.—The following classification for rough work is based upon the cold nitric acid test performed as follows: An ordinary wine-glass is half filled with urine, inclined until the liquid nearly overflows and then underlaid with nitric acid, poured in as slowly as possible, until it equals one-third of the urine volume. If then the wineglass be perfectly clean and placed obliquely in front of a dark background, the observer facing the light, one distinguishes:

1. The self-explanatory "*faintest possible trace.*"

2. *A very slight trace, i.e.*, a faint cloud *definitely seen only against the dark background.*

3. *A trace, visible without background,* both from the side *and from above* but not concealing the bottom of the glass when so viewed.

4. *A large trace, i.e., a sharply defined zone,* but not flocculent nor wholly opaque when viewed from above (represents about $\frac{1}{10}$ of 1 per cent.).

5. A $\frac{1}{8}$ of 1 per cent. reaction *i.e.*, a sharp, non-flocculent zone *obscuring the bottom of the glass.*

6. One-fourth of 1 per cent., band flocculent and opaque.

7. One-half of 1 per cent., band dense and flocculent.*

PUS.—*Pus in the urine is readily recognized by chemic or microscopic tests.* As a gross sediment *phosphates* and the *pale urates* may mislead the careless observer, but should never cause confusion. Urates are dissolved by heating. Phosphates disappear if an acid be added, whereas both the foregoing procedures will increase a turbidity due to pus.

Effect of reaction.

Chemic Test.—*Add liquor potassæ to the suspected urine and shake the solution vigorously. The persistence of suspended air bubbles and viscidity indicates pus.*

If the amount be very large or if the liquor potassæ be added to the sediment after decanting the supernatant fluid, a gelatinous mass results. *The microscopic test* is more definite when mere traces of pus are present. The addition of a drop of *very dilute* acetic acid solution to a microscopic preparation clearly brings out the nuclei of pus cells under the microscope.

BLOOD (*Hematuria, Hemoglobinuria*).—*In general, acid urines containing considerable blood are dark or smoky, and alkaline urines bright red.*

Color deceptive.

Oftentimes, however, its presence must be detected even though the amount is insufficient to color the urine, and, moreover, *mere color cannot be depended upon in any case.*

Small quantities.

For the detection of small quantities of blood the *microscopic examination,* chemical tests and the spectroscope are extremely important, as only the coloring matter may be present either primarily, or later as a result of fermentation changes.

A misleading color.

The color due to poisoning by coal-tar products closely simulates the smoky urine of hematuria.

Heller's Test for Blood.—(*a*) Boil the urine in a test-tube. (*b*) Add caustic soda and continue the boiling as long as precipitation continues.

* It will be seen that such distinctions require much practice and the method can hardly be justified at the present day, but from personal experience in the old Harvard laboratory the author can attest the accuracy there attained.

If blood be present, the phosphatic precipitate is brownish-red and the supernatant fluid, a bottle-green. This test is said to detect 1 part of blood to 1000 parts of urine.*

The Guaiacum Test for Blood.—Shake in a test-tube equal parts of old turpentine (or hydrogen peroxide solution) and fresh tincture of guaiacum. Pour this mixture gently down along the side of the tube so as to overlay the urine. If *blood or pus* be present, a blue band appears at the point of junction. If this be due solely to blood, it persists when the temperature of the mixture is raised to the boiling point, whereas, if pus alone be present, the color disappears. As oftentimes both are present in the urine, the reaction may lack precision.† Blood vs. Pus.

Comment.—*It need hardly be said that in all cases of hematuria the microscopic examination* is the usual and proper test. In the rare hemoglobinurias the chemical methods find their place.

The Sources of Hemorrhage.—Aside from acute Bright's disease, severe acute congestion, and the rare cases of so-called "*renal epistaxis*," the commonest sources of blood are urinary calculi and new growths. Renal epistaxis.

Clots occasionally exist if the blood is from the bladder, or small clots may represent the lumen of the ureters.

Blood from the kidney is usually well distributed and lacks clots, and further indications of its source are found in the presence of blood casts and fibrinous casts. Renal blood.

If from the kidney pelvis, the blood cells are thoroughly distributed and associated with the peculiar epithelium of that region, and may be due to calculi, acute inflammations, or varicosities. Associated leucocytes in quantity point to inflammation, whether simple, septic or tuberculous. Pelvic blood.

Bladder hemorrhages suggest ulceration, calculi, tumors or acute inflammation and, rarely, the *filaria sanguinis hominis*, may be found together with a chyluria.

If from the urethra, the first jet of urine contains the blood or pus, though if from the *prostatic* portion, it may appear chiefly at the end of micturition. "First jet" blood.

Accidental blood may occur, especially in the female during menstruation, and must always be borne in mind as a possibility. Menstrual blood.

In hemoglobinuria or methemoglobinuria the blood cells are scant or absent but the spectroscope shows the characteristic bands of oxyhemoglobin, reduced hemoglobin or methemoglobin and the chemical tests reveal the true condition.

The washed out, swollen "phantom" blood cells so often encountered in old specimens may be overlooked and mislead the novice into a false diagnosis of hemoglobinuria. "Phantom" blood cells.

Conditions Associated with Hemoglobinuria.—This condition occurs in poisoning by nitro-benzol, sulphonal, antipyrin, phenacetine, naphthol,

* If the urine be primarily alkaline and its phosphates precipitated one may add some acid normal urine before testing.

† If the slightest trace of copper remains from a previously performed Haines' or Fehling's test in an imperfectly cleaned tube, a false reaction will appear.

mushrooms, in severe tropical malaria, and certain rare cases of syphilis, typhus and scarlatina.

Bile pigment.

BILE.—Bile acids and bile pigment are both found in the urine under certain conditions. *Bile pigment*, if present in considerable quantity, is readily detected by the yellow-tinted foam produced by shaking.

Test.—*The best simple chemic test is made as follows:* Filter the suspected urine several times through the same filter-paper. Drop *fuming nitric acid* upon the wet paper and watch for the characteristic color-play, viz., orange, red, violet, and *green, the last being the essential color.*

Rhombic crystals of bilirubin are readily obtained by shaking up thoroughly with chloroform, decanting and evaporating the chloroform extract.

Simple and effective.

Marechal's Test for Bile Pigment.—Overlay a portion of the suspected urine in a test-tube with an alcoholic solution of iodine (tincture iodine, 10 parts; alcohol, 90 parts). If bile be present, a beautiful green band appears at the junction point.

Excellent test.

Haycraft's Test for Bile Acids.—This is made by dropping a pinch of "flowers of sulphur" upon the surface of the suspected urine. If the *bile acids* are present the powder drops to the bottom of the tube.

This old test has been recently studied by Frankel and Cluzet. They find it accurate, sensitive, and, as applied to the urine, very definite. The reaction depends upon the diminution of surface tension and the presence of bile acids indicates the presence of hepatogenous jaundice, though their absence does not exclude it.

TESTS FOR GLUCOSE IN URINE.—*All clinical tests for glucose in solution depend upon one of the following properties:*

(*a*) *The fact that when glucose is brought into contact with certain oxides, as for example, those of copper and bismuth, it becomes oxidized at their expense, i.e., acts as a reducing agent.*

(*b*) *Its ready fermentability.*

(*c*) *The fact that it is dextrorotatory.*

Relative value.

The Copper Tests.—The well-known *Trommer's test* has been superseded by better and more accurate methods, but *Fehling's* solution is so widely used still as to require a description of the test.

This solution is distinctly inferior to Haines' modification and is so unstable that it must be kept in two parts and mixed whenever needed for use.

Test fluid

Fehling's Test.—*Directions*: (*a*) *Remove albumin by boiling and subsequent filtration.* (*b*) *Pour into a test-tube one finger-breadth each of the following stock solutions, which when mixed should form a deep blue solution.*

Stock Solutions.—*Solution A.*—Dissolve 34.64 grams of pure, dry, powdered copper sulphate in 200 c.c. of warm distilled water and add distilled water to make 500 c.c. of the *light blue solution.*

Solution B.—Dissolve in 300 c.c. of hot water 180 grams of Rochelle salt. Filter. Add of pure caustic soda, 70 grams. Cool, and add distilled water enough to make 500 c.c. of a *colorless solution.* Keep in a dark place.

Test.—Heat test solution to boiling point, add at once 20 to 30 drops of the suspected urine and *boil no longer*, but in the absence of a reaction set

aside for from five to thirty minutes. *A positive reaction proves nothing but the presence of a reducing agent unless the ultimate precipitate is red, not yellow or green.* Coppery red is true color.

Objections to Fehling's Solution.—*Fehling's solution as ordinarily prepared is open to serious objections: (a) It is unstable. (b) An excess of glucose obscures the terminal reaction by becoming caramelized if boiling is prolonged. (c) It cannot be applied directly to ammoniacal urine unless the specimen be specially treated. (d) A large number of substances may reduce its cupric oxide.*

Such are glycuronic and glycosuric acid, alkapton, creatinin, uric acid, and various drugs, such as benzoic acid, chloroform, chloral, glycerin, the salicylates, turpentine, etc.

Hence, if one uses Fehling's solution for qualitative work, he must bear in mind that it is more valuable as a negative than a positive test. Of value in negation.

A urine that does not reduce Fehling's solution is free from glucose, but reduction does not conclusively establish its presence.

Haines' Solution.—This is a much simpler, more stable and permanent copper solution than that of Fehling. A better test.

Formula.—Take of pure copper sulphate 30 grains; of distilled water 1 ounce. Make a perfect solution and add, of pure glycerine ½ ounce; mix thoroughly and add liquor potassæ 5 ounces.

Application of Test.—Boil a few cubic centimeters of the solution gently in a test-tube, add guttatim 6 to 8 drops of the urine, *boil gently for a moment only*. The color reaction is identical with that of Fehling's solution. Avoid overboiling.

All concentrated urines should be diluted before a copper test is made.

This procedure minimizes the risk of misleading reductions due to drugs and other substances.

ALLEN'S TEST.—This is said by Hutchinson and Rainy to have the following advantages: (*a*) Albumin need not be removed. (*b*) Uric acid, creatinin and like substances do not affect the reaction. For albuminous urines.

The test derives its value from the fact that acid solutions of sodium acetate precipitate the interfering substances without removing or affecting any glucose that may be present.

Test.—In a perfectly clean test-tube heat 8 c.c. of urine to the boiling point. Pay no attention to any precipitate (albumin). Add 5 c.c. of the solution of copper sulphate used in making Fehling's solution (solution A, as described). Cool partly. Add 2 c.c. of a saturated solution of sodium acetate that has been rendered faintly acid by acetic acid. Interfering substances are now precipitated. Filter and add to the clear filtrate 5 cc. of the solution B used in making Fehling's solution. Boil twenty seconds.

If sugar is present, the solution becomes opaque and green, and deposits, either immediately or after standing a few minutes, a fine yellow precipitate.

Control Positive Copper Tests.—*The tests already given are placed in the direct order of their convenience and rapidity, but the inverse order of accuracy. Yet error will seldom occur if every sugar reaction is checked by the one accurate test for glucose, namely:* Avoidance of error.

THE FERMENTATION TEST.—*This test depends upon the fact that glucose is a fermentable substance, and practically the only one that urine ever contains.*

Einhorn's tubes.

The Test.—The most convenient apparatus is that of Einhorn. If Einhorn's saccharometer is not to be had, the Doremus ureometer tubes will answer the purpose of proving sugar present or absent.

Each step in the process should be checked by comparison with a normal urine, therefore, two tubes are required.

Technic.—(*a*) *Boil the two specimens for several minutes, to drive off any air they may contain.*

(*b*) *Add to each a pinch of tartaric acid in order to maintain their acidity and prevent ammoniacal decomposition.*

(*c*) *Dissolve in each the same amount of yeast* (about one-sixth of a fresh yeast cake is sufficient). If there be any doubt as to the freshness of the yeast, it is well to test it with a control solution of glucose.

(*d*) *Fill the long limb of the fermentation tube of the saccharometer or of the Doremus ureometer with urine, and place the two specimens side by side in a warm place.*

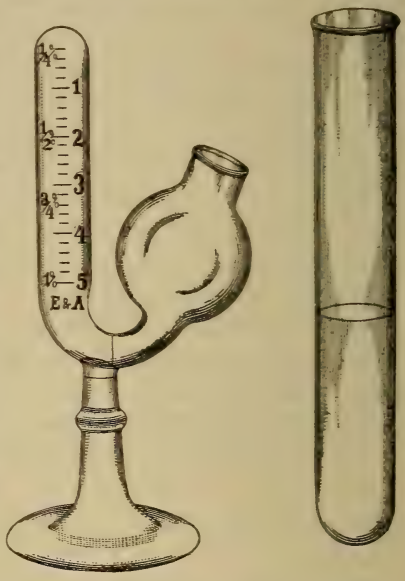


FIG. 67*a*.—Einhorn's saccharometer.

About twenty-four hours are required for complete fermentation and quantitative estimation, but the diagnosis can often be made in a very short time, as the appearance of any considerable amount of gas proves the presence of glucose.

By using Einhorn's saccharometer, the amount of sugar may be determined with sufficient clinical accuracy by the scale measuring the volume of gas produced.

Specific gravity test.

Rougher Method.—Lacking any form of fermentation tube, the test may be made by first taking the specific gravity of the two specimens, to one of which yeast is added. Both are then set aside in a loosely stoppered flask or bottle and in a warm place.

If glucose be present, fermentation occurs, the specific gravity is thereby lowered and each degree of density lost roughly corresponds to 1 grain of glucose to the ounce of urine (0.21 gram to 100 c.c.).

The specific gravity should not be taken the second time until the urine has cooled to the temperature it had when the first estimate was made.

Quick but inaccurate.

QUANTITATIVE ESTIMATION WITH WHITNEY'S REAGENT.—(Much used but not recommended).—In addition to the previously described tests may be mentioned that of Whitney, which, more rapid than the fermentation test, but inaccurate, depends upon the decolorization of an ammonic-cupric-sulphate solution.

Test.—One dram of this solution is heated to the boiling point and the urine is added drop by drop, the mixture being boiled for from three to five seconds after each addition. If no change occurs, the process is continued until 10 or 15 drops have been added. If sugar be present, the blue color begins to fade, and is finally entirely removed, leaving a colorless solution. The amount of sugar present is then estimated by the following table: Technic.

If reduced by minims	It contains to the ounce	Percentage
1	16 or more grains	3.33
2	8 grains	1.67
3	5.33 grains	1.11
4	4 grains	0.83
5	3.20 grains	0.67
6	2.67 grains	0.56
7	2.29 grains	0.48
8	2 grains	0.42
9	1.78 grains	0.37
10	1.60 grains	0.33

If the amount of sugar be large, as indicated by the loss of color following the addition of 1 drop, the urine should be diluted by doubling or trebling its volume by the addition of distilled water, and the result then obtained must be multiplied by 2 or 3, as the case may be. Dilution.

The duration of the boiling period must be neither more than five nor less than three seconds. In the author's hands it has proven far inferior to the fermentation test, yet convenient for rapid rough work. Caution.

THE POLARIMETER.—Direct estimation of the sugars by this means is by far the most satisfactory method and requires no special training, but the instrument is somewhat expensive. The percentage of sugar is read directly from the vernier scale in the clinical instruments, such as Ultzmann's, but it is necessary to decolorize the specimen with lead acetate and magnesium oxide, if necessary, to attain a clear solution. The urine must be albumin-free and the containing tube dry or, if containing water, it should be rinsed out with some of the urine to be tested.

LEVULOSE.—Levulose is abundant in ripe fruits and honey, is absorbed unchanged and occasionally appears in the urine, yet it can be taken in quantity in diabetes without causing a marked excretion, and in ordinary glycosuria may replace cane-sugar or glucose in the diet. Instances of pure levulose diabetes have been reported, but they are exceedingly rare. A glucose substitute for diabetics.

If 100 grams of honey be taken on a fasting stomach by a non-diabetic not more than 10 to 15 per cent. will show levulosuria, but in diseases of the liver, such as cirrhosis, carcinoma and syphilis, the same test may result in a marked excretion and indeed the same substance may appear spontaneously in such cases.

Test.—*The polarimeter shows rotation to the left, but the substance reacts to fermentation and Fehling's solution exactly like glucose.* Levorotatory.

For identification, therefore, one must often use *Seliwanoff's test.* Add to 10 c.c. of urine (showing levo-rotation) a little resorcin, and 2 c.c. of dilute HCl: *a bright red reaction appears on heating if levulose be present.*

Appears post partum.

LACTOSE appears in the urine of women after childbirth (not during pregnancy), and more frequently in those who do not nurse the child (80 per cent. as against 20 per cent.). It is most abundant about the fifth month after delivery and seldom exceeds 0.5 per cent.

Rubner's Test.—*It reduces Fehling's solution* and is fermentable with difficulty, but whenever any sugar reaction appears in women after delivery

Specific test.

Rubner's test should be applied: To 10 c.c. of urine are added 3 grams of lead acetate, the resulting precipitate is filtered off and the filtrate heated until it assumes a brownish color, and again after the addition of ammonia.

The appearance of a brick-red color and a cherry-red precipitate indicates lactose if there be more than 0.3 per cent. Specimens of high specific gravity should be diluted one-half and less amounts require evaporation of the urine and a test of the residue.

PENTOSE.—*After the ingestion of large quantities of fruit, or more rarely after taking excessive amounts of vegetables, coffee, tea or milk, pentose may appear in sufficient amount to reduce Fehling's solution slowly and atypically.*

Non-fermentable.

A very few cases of true pentosuria with otherwise unimportant symptoms have been observed. *It does not yield gas production by fermentation* even though the pentoses are thereby decomposed, *which differentiates it from the more important substance, glucose.*

Bial's Test for Pentose.—*Test Solution.*—One gram of orcin and 30 drops of 10 per cent. ferric chloride solution are added to 500 c.c. of concentrated hydrochloric acid.

Test.—To 10 c.c. of the solution 5 c.c. of the suspected urine is added and the mixture raised to the boiling point but no further. A green precipitate on cooling indicates the presence of pentose.

If the urine has shown glucose this reaction will be imperfect.

F. C. Wood recommends short preliminary fermentation with a *pure culture* of saccharomyces in such cases.

MALTOSE.—Its chief interest lies in its presence in certain cases of pancreatic disease, but the tests are not suitable for the practitioner.

Levorotatory and non-fermentable.

GLYCURONIC ACID.—*This substance may in certain combinations reduce Fehling's solution, but it has no definite clinical significance, does not undergo fermentation with yeast and is levorotatory in acid solution.*

The only specific test for this substance is the complicated one of Neuburg, a description of which may be found in the physiological chemistries.

ACETONE, DIACETIC ACID AND OXYBUTYRIC ACID.—Although oxybutyric acid as the mother substance plays a basic part in acidosis, one need only consider here the clinical recognition of the two former substances. A distinction may be drawn between the diabetes of a severe and that of a mild type by the behavior of the acetone and diacetic acid as affected by diet.

Acidosis.

These substances may be found in conditions other than diabetes and in this ailment are prone to appear if the diet is rigidly and excessively proteid and fatty and disappear when a proper regimen is established, but if they persist in excess in a case of diabetes the case is a bad one and the end not far distant. (See "Diabetes.")

Acetone and diacetic acid have essentially the same significance, but the former precedes the latter and should be sought in diabetic urines if diacetic acid is proven to be absent.

DIACETIC ACID.—Test.—If a few drops of the tincture of ferric chloride be added to the *freshly passed urine* containing diacetic acid, a bordeaux-red appears together with a precipitate of phosphates. Use fresh urine.

For accurate results these should be allowed to settle or be filtered out and the addition continued.* Filtration important.

Heat should be applied and should cause a diminution of intensity in the color on boiling if it be due to diacetic acid.

The presence of diacetic acid indicates the presence of acetone to an amount of at least 0.02 gram in the twenty-four hours' urine, and is seldom absent when the daily acetone excretion reaches 1.5 grams.

Acidosis.—*Diacetic acid in the urine, with or without oxybutyric acid, indicates acidosis.* An ominous symptom.

This represents a condition of the blood inviting fatal coma in cases of diabetes and demanding immediate recourse to alkalies, periods of starvation, the introduction of carbohydrates to tolerance and the withdrawal of fats and diminution of proteids.

Increased diacetic acid excretion with coincident diminution of acetone is said to be an immediate forerunner of diabetic coma.

ACETONE.—Legal's Test.—Distil a few cubic centimeters of urine by using a simple distillation flask with its neck corked and its lateral arm leading into a test-tube or ordinary flask; a bunsen burner or alcohol lamp completing the outfit. The distillate collected in three or four minutes is treated with a few drops of a fresh solution of sodium nitro-prusside, a few drops of acetic acid are added and the mixture rendered alkaline by sodium hydrate. If acetone be present a red appears deepening to carmine and purple-red.

Lieben's Test.—Add to distillate a few drops of Lugol's solution and sodium hydrate. If acetone is present it will form a macroscopic or microscopic sediment of the star-like yellow crystals of iodoform, and on heating the characteristic odor may appear. Simpler method.

The excretion of acetone may promptly follow the withdrawal of carbohydrates in diabetes and in such cases is removed usually by the administration of even relatively small amounts of such foods.

As stated previously, the best, most effective and, in fact, obligatory method of relieving the acidosis *of the diabetic* lies in brief periods of total starvation, administering sodium bicarbonate until the urine is neutral or alkaline, and adjusting a carbohydrate intake to the tolerance of the patient.

Acidosis in Non-diabetics.—*Acidosis may occur in many conditions other than diabetes.*

Among these are advanced gastric or intestinal malignant growths, locomotor ataxia, melancholia, prolonged chloroform anesthesia, severe

* Crofton states that color due to ingested drugs is permanent, whereas that due to diacetic acid soon disappears.

typhoid, senility, terminal cardiac incompensation with general dropsy, and many other conditions.

It should always be sought for and if present corrected in cardiorenal insufficiencies, and in stuporous states or low delirium associated with profound exhaustion or inanition.

The Preservation of Urine for Microscopic Test.—*As previously stated, the preservation of specimens is best accomplished by the addition of a slight excess of chloroform which is easily removed by heat before any tests are applied.*

Chloroform.

Many agents may be used if the sediment alone is to be examined (formalin, chloral hydrate, thymol, boracic acid, etc.), but are likely to cause some confusing reactions in the tests for albumin or sugar.

THE EXAMINATION OF URINARY SEDIMENTS.—The accurate microscopic examination and correct interpretation of urinary sediments demand a thorough knowledge of the special technic involved.

Gross error is possible in two directions: (1) *Through failure to detect important abnormal elements.* (2) *Through misinterpretation of the elements found.*

Every physician must not only own a microscope, but be able to use it, and no time can be more profitably employed than that which is spent in acquiring a correct technic. He who graduates from the schools of today is, or at least should be trained to a degree that will enable him to do reasonably accurate work in this line. On the other hand, many excellent men of the older generation through lack of early training and opportunity are still sadly deficient in this important branch of clinical medicine. *In many medical schools, moreover, even at the present time, the training is wholly inadequate.*

Illumination.—*In examining a urinary sediment microscopically, the first essentials are correct focusing and a dim light.*

Every microscope should be provided with an iris diaphragm and a nosepiece holding three objectives, one low-power, one medium, and for the third, a high-power oil-immersion lens. In urinary work only the first two are required, save in those cases in which the tubercle bacillus is the object sought.

Light and lenses.

The physician should commence operations with a *dim light* and his low-power lens (1 inch or ½ inch objective), this being by far the best for finding casts as it distinctly shows their outline and gives a larger field. He should then slightly increase his light and bring his medium-power objective (¼ to ⅙ inch) into focus. This brings out the structure of casts and renders distinct any cellular elements or crystals that may be present in the field. Having once found any object of interest, the lenses and the illumination may be changed at will.

Avoid over-illumination.

Substances Found in Urinary Sediments.—The following are the inorganic substances most frequently found in urinary sediments:

Misleading objects.

(*a*) **Extraneous material,** such as fibers of cotton, linen, wool or silk, various vegetable forms from the rinsing water, if it be not distilled, starch grains, etc.

(*b*) **Phosphates.**—The macroscopic appearance of the phosphatic deposit is well known. This grayish-white material so often mistaken for pus is

composed of amorphous calcium and magnesium phosphates and their ready solubility in mineral acids at once identifies them. They are found only in alkaline urine, and, if ammoniacal fermentation has occurred, will be asso-

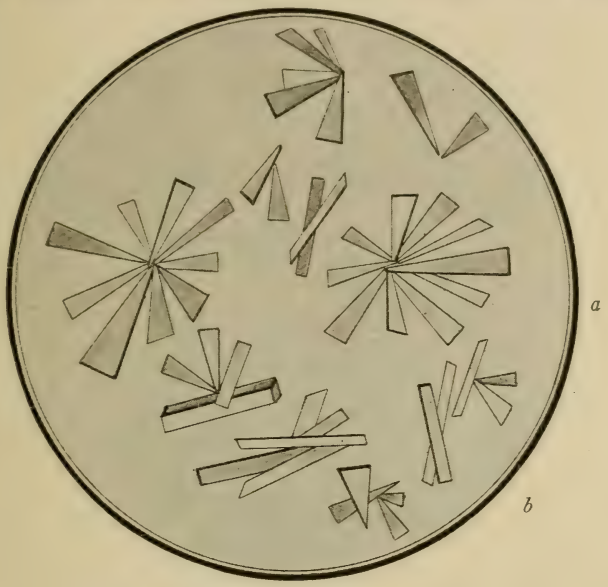


FIG. 68.—*a*. Calcium phosphate. *b*. Calcium sulphate. (*After Jakob.*)

FIG. 69.—Calcium oxalate crystals.

ciated with beautiful crystals of the triple phosphate. Calcium phosphate may also appear in crystalline form.

(*c*) **Urates.**—The ordinary deposit of urates occurs in moderately acid,

"Brick-dust" deposit.

concentrated urine during the process of cooling or when exposed to an unusual degree of cold. The color varies from yellow to rose-red, the latter constituting the so-called brick-dust deposit (*sedimentum lateritium*).

FIG. 71.—Ammonio-magnesium (triple) phosphate.

FIG. 72.—Uric acid crystals.

Upon application of heat they promptly disappear. When nitric acid is added to a urine rich in urates, a deposit of nitrate of urea is formed. The deposited urates are amorphous, excepting only the ammonium urate, which

occurs in ammoniacal urine as the so-called thorn-apple crystals (see Fig. 74).

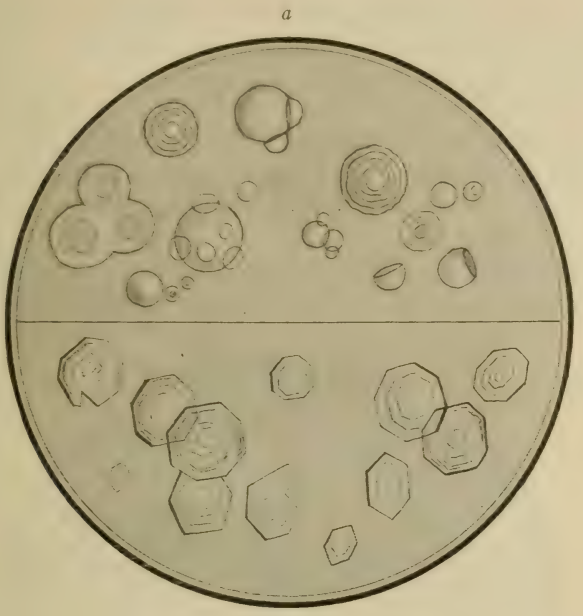


FIG. 73.—*a*. Cystin. *b*. Leucin.

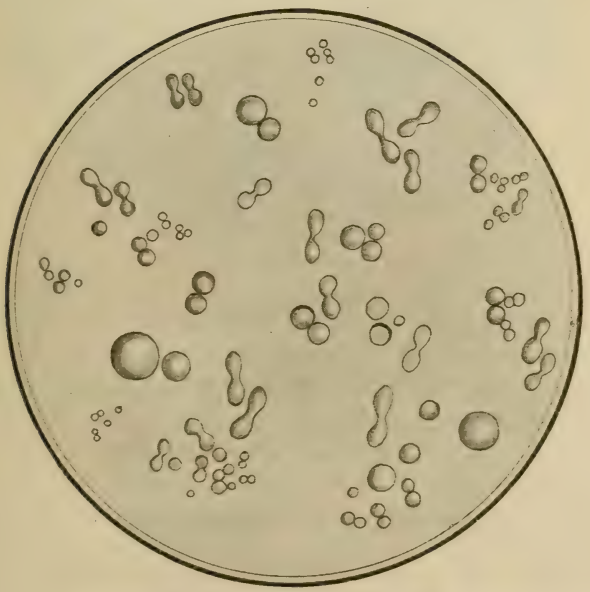


FIG. 74.—Calcium carbonate. (*After Jakob.*)

(*d*) **Uric Acid.**—Uric acid may be precipitated from any urine, if concentrated, during the so-called acid fermentation or in hot weather when the

Cayenne pepper deposit.

high temperature prevents precipitation of the urates. Excessive acidity and concentration or a pathologic excess may occur in certain conditions, but, *when passed*, urine should never contain the crystals as a precipitate. The macroscopic deposit resembles cayenne pepper. The microscopic appearance is best shown by the plate (see Fig. 71).

(*e*) **Calcium Oxalate.**—This rarely forms a visible sediment and is abnormal if found in urine freshly passed. The crystals are characteristic and unmistakable (see Fig. 69). Leucin, tyrosin, and cystin are rarely seen, but may be readily recognized by comparison with the illustrations (see Figs. 72, 75).

(*f*) **Calcium Carbonate.**—This is occasionally precipitated with the earthy phosphates. Usually it is amorphous but occasionally it forms crystals

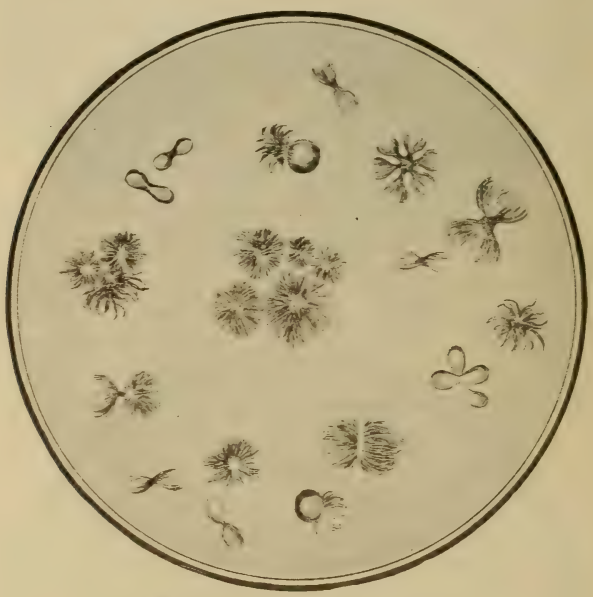


FIG. 75.—Ammonium urate.

Effervescence.

shaped like a dumb-bell. It is easily recognized by the effervescence produced when a mineral acid is added (Fig. 73).

Normal vs. Abnormal.

(*g*) **Blood.**—*Erythrocytes.*—The blood as it appears in the urine may be quite normal in appearance or, on the other hand, be so changed as to make its recognition difficult. If hemorrhage has taken place in the urethra, prostate, bladder, ureters or in the pelvis of the kidney, and the urine is acid and fresh, the red corpuscles appear as yellow biconcave discs, with rounded edges and a light central portion.

Easily overlooked.

Phantom Cells.—If, on the other hand, the hemorrhage has taken place into the cortical portion of the kidney, the urine has stood for some hours or is alkaline, the corpuscles may become *pale and swollen*, their diameter lessened and they may appear as *mere shadowy circles* and as such are easily overlooked.

Crenation.

Crenation of the normal cells may also occur in a urine that is

deficient in salt. Such cells have irregular, star-like processes along their border, but retain the yellow tinge of the normal cell.

(*h*) **Pus.**—Pus in quantity is in most cases easily detected by the chemic test. The pus cell as seen under the microscope is identical with the white cell seen in a smear preparation of normal blood but is less easily recognized as such in the urine.

Differentiation.

In the acid urine the pus cells are usually larger than the red cells, are colorless, granular and, as a rule, in fresh specimens, their nuclei may be readily distinguished by careful focusing.

Useful maneuver.

The presence of these nuclei serves to distinguish them positively from the red cells and any doubt upon this point may be readily removed by allowing a drop of very dilute solution of acetic acid to run beneath the cover-glass,

FIG. 76.—Tyrosin.

assisting the process, if necessary, by laying the edge of a piece of filter or blotting paper against the opposite edge. The acetic acid dissolves the granules and brings out clearly the cell nuclei, *but unless very dilute, will destroy any hyaline casts that may be present. In ammoniacal urine, the pus corpuscles are soon destroyed, becoming agglutinated and losing their structural characteristics.*

This maneuver is also useful in differentiating the leucocytes from renal cells.

Diagnostic aids.

Varying Significance of Pus in the Urine.—As to the significance of pus in the urine, it may be remembered that:

(*a*) *Pus that comes with the first jet of urine, the remainder being clear, is from the urethra.*

(*b*) *A moderate amount of pus, appearing in an acid urine, is usually from the renal pelvis, but may be due to tuberculosis of the bladder.*

(*c*) *Large quantities of greenish pus point to rupture of an abscess* in the urinary passages or to a pyelonephrosis.*

(*d*) *Pus in a urine which is ammoniacal when passed is usually, but not always, from the bladder.*

In all cases the diagnosis must depend upon the character of the associated epithelium and the presence of casts or specific microörganisms.

(*i*) **Spermatozoa** are easily recognized by their well-known form.

(*j*) **Bacteria and Yeasts.**—*Bacteriurea.*—Aside from the tubercle bacillus, many forms of bacteria may be found in the undecomposed urine, and their presence may be unattended by symptoms or accompanied by a variable amount of irritation of the bladder. So, also, in fermenting urine one finds

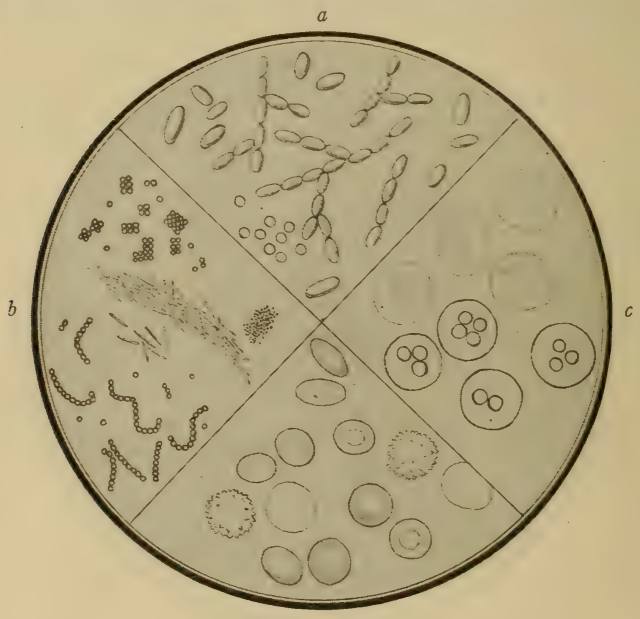


FIG. 77.—*a*, *b*. Various forms of fungi and bacteria. *c*. Pus cells before and after treatment with acetic acid. *d*. Various forms of red blood cells.

both bacteria and spores. The spores are highly refractile and tend to form chains. The most important bacterium is the tubercle bacillus, only to be recognized by such staining methods as are described elsewhere.

Great difficulties.

The ordinary bacterial urine is persistently cloudy, has a musty odor, and presents on shaking a swirling appearance, as if fine grains of sand or dust were put in motion.

(*k*) **EPITHELIUM.—Limits of Differentiation.**—*In spite of the efforts put forth by microscopists, the exact differences existing between the epithelial cells of different portions of the urinary tract have been determined only in part.*

Such variations are shown by the plates, but must be learned from the careful study of specimens obtained from known cases of cystitis, nephritis, etc., a mere description being of slight value.

* Most commonly prostatic.

The renal cell as seen in the urine is usually small, though almost double the size of a red blood cell, round or cubical, and mononuclear.

Always mononuclear.

Cells from the straight tubules are somewhat larger and have a more irregu-

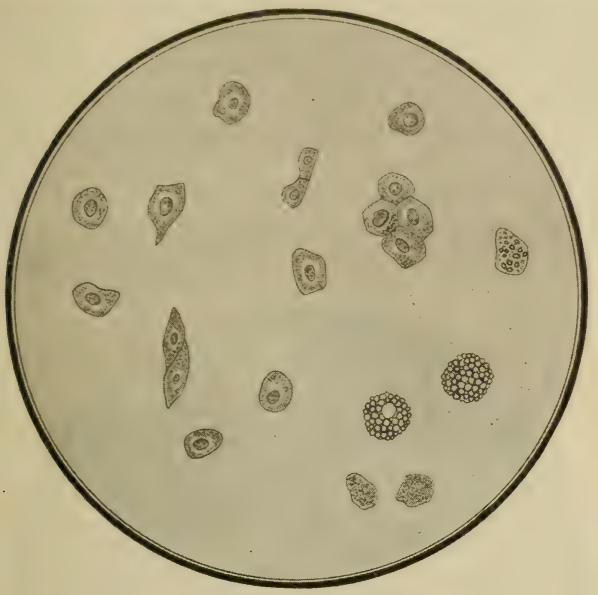


FIG. 78.—Renal cells. Various forms, including compound granule cells.

FIG. 79.—Bladder epithelium. (*Various forms.*)

lar shape, being sometimes polygonal or cubicular, but, *as contrasted with the leucocyte, all renal cells are mononuclear.**

* Degenerated cells may of course show no trace of a nucleus.

In cases of "obstructive" jaundice the *renal cells* are often distinctly bile-stained.

Pelvic cells.

Caudate cells arranged in overlapping layers are often described as the characteristic cells of the renal pelvis, but, as a matter of fact, they represent only the superficial layer, and are seen only in early or mild cases of pyelitis. *The cell from the deeper layer strongly resembles the renal cells.*

Ureteral cells are spindle-shaped.

Pavement epithelium.

Bladder epithelium is large, mono- or polynuclear, and generally of the well-known tessellated or pavement variety. Such cells often occur in aggregations, may be distinguished from *vaginal epithelium* only by the fact that the cells are somewhat smaller, never overlap, and occur in a single layer.

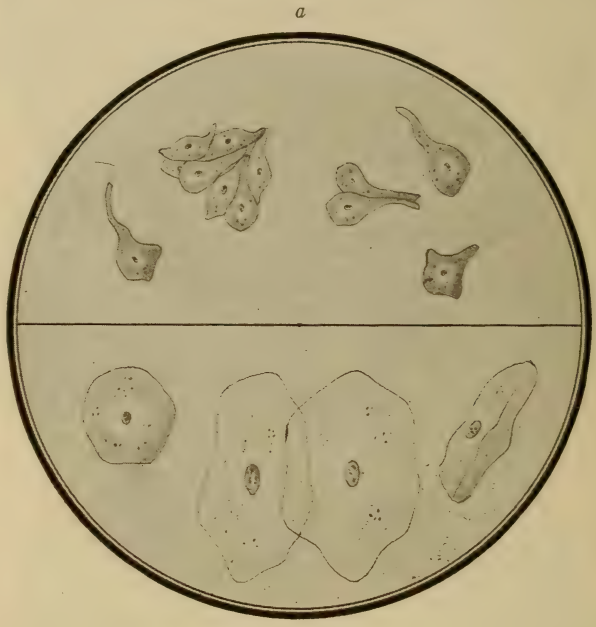


FIG. 80.—*a*. Epithelium from renal pelvis. *b*. Vaginal epithelium.

Bladder cells from the middle layers are smaller, often somewhat drawn out, or perhaps definitely "tailed." Those of the deep layer may be round, cuboid or polygonal. All carry a vesicle-like nucleus.

Vaginal epithelium is rather larger: the cells overlap like the shingles on a roof, and are likely to occur in masses consisting of several layers.

Significance.

The epithelial cells from the neck of the bladder, prostate, and the calices of the kidney pelvis are almost identical.

An absolute differentiation of the epithelial cells in a urinary sediment is oftentimes impossible and will often demand the corroborative testimony of other findings.

Fatty Renal Cells and Compound Granule Cells.—The former are much smaller than the latter which are large, round, bulging with refractile granules

and frequently spiculated by fatty crystals. They have no exclusive disease relation, but may be found in any form of renal lesion characterized by fatty casts and in other chronic inflammatory and ulcerative lesions of the urinary tract.

Renal cells reflect the same changes as are shown by the various types of casts.

CASTS.—Casts are of two kinds: (1) **The true cast,** *which has its origin in an exudate from the tubules of the kidney.* (2) **The pseudo-casts,** or *cast-like bodies, which may or may not originate in the kidney.*

True Casts.—*The exact nature and source of the true casts are not positively known. Whether they consist of disintegrated and modified epithelium, or a morbid secretion of the epithelium itself, or are simply a coagulated albuminoid transudate from the blood, the fact remains that they appear clinically as casts*

Exact nature unknown.

Possible origin.

FIG. 81.—This figure shows the appearance of a group of casts under correct dim illumination.

of the renal tubules, having to a great extent a form and caliber corresponding to that portion from which they come, and appearing hyaline, waxy, fibrinous, granular, fatty, epithelial or bloody, according to the nature and extent of the underlying pathologic changes.

Varieties.

If we assume, for convenience of description, that their basis is that pathologic change known as hyaline degeneration, it becomes very easy to understand most of their modifications.

A convenient hypothesis.

Such a process would lead ordinarily to the formation of a *hyaline* cast. If the granules of the degenerated cell adhered to or became intermixed with the deposit, a *granular cast* would result, and this would be finely or coarsely granular, light or dark, according to the activity of the pathologic process. Fatty degeneration in the cells would be reflected in the *fatty cast.* Adherent

Hyaline.

Granular.

Fatty.

Epithelial.

desquamated cells would form *epithelial casts*; adherent blood cells, *the blood cast*, etc.

A Reasonable Assumption.—It is reasonable to suppose that the *basis* is the same for all varieties, and that in the hyaline, waxy, and fibrinous casts we have but slight variations in fundamental structure, though the conditions attending their presence may be quite different and distinct, and their clinical significance definite and important.

The Hyaline Cast.—*This is transparent, and shows an apparently homogeneous structure; its size is variable, its ends rounded, and its sides nearly parallel.*

Caution.

Its shape is best shown in Figs. 80, 84. Unless careful focusing and proper shading of the light be employed, it will certainly escape detection.

A difficult question.

Representing, as these hyaline casts do, the least degree of pathologic or at least of inflammatory change, it becomes at times a difficult question to determine the significance and importance of an occasional cast.

FIG. 82.—This figure shows the obliteration of same group by high illumination.

Not always present.

Some observers report them as present in nearly every urine if the centrifuge be employed, but such has not been the author's experience. Occurring alone and unassociated with albuminuria or other symptoms pointing to diseased kidneys, it must be admitted that their significance is less grave than would have been assigned them by the older teaching. It is certainly true that they may represent transient and hence often negligible conditions

Persistence.

or localized rather than general renal changes, but as a *persistent* constituent of the urinary sediment they still demand serious consideration and have lost little of their old significance. It is also a much debated question as to whether they are ever found without some degree of albuminuria and, according

Albuminuria minima.

to E. S. Wood, the hyaline cast is always associated with the pale very finely

granular cast.* It rapidly disintegrates in alkaline urine or in that treated with acetic acid.

Hyaline and finely granular forms.

Significance.—*The chief significance of the hyaline and finely granular cast, existing alone, is found in its special relation to acute and chronic infections, acute or chronic intoxications, chronic passive renal congestion, and the early stages of arteriosclerotic kidney, and true interstitial nephritis.*

Isolated findings.

Infection and Toxemia.—We are but just beginning to appreciate the part played by persisting foci of chronic streptococcic infection, and by other forms of persistent or recurring toxemia in the etiology of nephritis.

The readiness with which even a marked albuminuria, associated with hyaline cylinder showers, may be induced by heavy drinking is apparently little appreciated. In the author's practice this has several times led to the detection of the "sneak drinker."

Alcoholic excess.

FIG. 83.—*a*. Waxy casts. *b*. Fatty and fat-bearing casts. Fibrinous casts closely resemble waxy casts but are always yellow or brown.

The Waxy Cast.—*This is dense but highly refractile and is usually relatively short and of large size. It is either without color or slightly gray, never carrying the yellow or brown of its simulator, the fibrinous cast, stands out as clearly upon the field as does the triple phosphate crystal, and, once seen, is never forgotten.*

Unmistakable.

It points to one of three conditions: amyloid kidney, advanced chronic parenchymatous nephritis and the terminal stage of interstitial nephritis.†

Commoner associations.

* This corresponds to the author's own observations as applying to ordinary clinical work and to *true* casts and seems to have a logical pathologic foundation.

† Ultzmann states that it may be present in tuberculosis of the kidney.

Forerunner of death.

In amyloid kidney, it appears earlier than in interstitial nephritis. Indeed, in the latter it marks the beginning of the end, and cases of contracted kidney in which it is found will usually terminate fatally within one year.

The Fibrinous Cast.—*This misnamed cylinder resembles the waxy cast save in color. It is always yellow or even brown, deriving its color from blood pigment, and, as might be expected, points to an active or subsiding inflammation; ordinarily to acute Bright's disease.*

Active or subsiding inflammation.

The Blood Cast, and the Brown Granular Cast.—*This points to acute inflammation as does the brown (dark) granular cast.*

Inflammation.

If, as is usually the case, the cast carries washed-out cells (abnormal blood) it indicates slow effusion of blood or its high origin; if normal blood, more abundant hemorrhage or an origin in the straight tubules or pyramids.

FIG. 84.—Granular casts. Light and dark.

The dark granular cast is actually a blood-pigment cast.

Important variety.

The Finely Granular Cast.—*The special significance of this common form has been discussed together with that of the hyaline cast.*

The Coarse Granular Cast.—*This is usually associated with fibrinous or waxy casts.*

The Fatty Cast.—*This is most frequently seen in convalescent cases of acute nephritis, or in chronic parenchymatous nephritis.*

Good or bad omen.

True Epithelial Casts.—*These occur only in active or subsiding, acute or subacute inflammation, in acute Bright's disease, or the subacute exacerbation of a chronic parenchymatous nephritis.*

Epithelium-bearing.

Many casts carry here and there an epithelial cell and should be so described in case reports, yet these cannot properly be called epithelial casts.

Pus Casts.—*These are found in suppurative disease of the kidney or in*

pyelitis extending to the straight tubules and frequently require treatment with dilute acetic acid to distinguish them from certain epithelial casts.

Indicate suppurative focus.

Significance and Associations of Casts.—*Hyaline and finely granular casts indicate: (a) Interstitial nephritis. (b) Amyloid kidney. (c) Chronic congestion of the kidney.*

They are also frequently found in acute and chronic infections and intoxications and even in asthenic albuminurias of the orthostatic or adolescent types, though in many or most of such cases some chronic obscure infection exists (tonsils, teeth, nasal accessory sinuses, cryptogenetic tuberculosis, etc., etc.).

Waxy casts indicate as stated: *(a) Terminal stage of chronic interstitial nephritis. (b) advanced parenchymatous nephritis. (c) amyloid kidney.*

FIG. 85.—*a*. Blood cast and hyaline cast carrying red blood cells. *b*. Leucocyte or pus cast. *c*. Hyaline cast carrying leucocytes. *d*. Epithelial casts.

Acute and Subacute Nephritis.—*In these ailments one finds: (a) Fibrinous; (b) blood; (c) epithelial; (d) hyaline; and (e) dark granular casts.*

Convalescence from Acute Nephritis.—*Here one finds fatty, dark granular, epithelial and hyaline casts.*

Uncomplicated Chronic Parenchymatous Nephritis.—*In this disease one finds fatty, hyaline, and finely granular casts, and with any decided disturbance, epithelial casts.*

Sources of Error in Searching for Casts.—*Cast-like Forms.* The amorphous urates are frequently grouped in cast-like forms and similar accumulations of bacteria and other sedimentary substances may perplex the tyro. They will seldom or never deceive the trained eye as they lack the definite linear boundaries of the genuine cast. Urates, moreover, are promptly dissolved by heating the preparation. More deceptive still are the *cylindroids.*

Misleading forms.

Pseudocasts.

They are usually longer and more band-like than the hyaline cast, and more likely to be convoluted or twisted; usually they take the form known as mucous cylindroids which occur in many non-albuminous urines and cannot be mistaken for true casts as they appear flattened and ribbon-like, often with frayed ends and faint longitudinal striation.

Mucous cylindroids.

Demand Typical Forms.—*The author believes that fewer casts would be reported in otherwise normal urines if the observer regarded all doubtful forms as disproven until he could find in the same urine a definite cast of the ordinary size and form, and with both ends rounded. If of renal origin, the unusual will certainly be accompanied by the common form.*

Important rule.

Cylindroid Storms.—In certain cases, periods of mental depression are accompanied by *showers* of spurious casts without the slightest change in

Cast showers.

FIG. 86.—Spermatozoa and associated substances in urinary deposit. *a*, *a*, *a*, *a*, Spermatozoa. *c*, *c*. Spermatozoa, tail out of focus. *d*, *d*, *d*. Amyloid corpuscles. *e*, Prostatic cast. *g*. Crystals. *h*. Lecithin-granule cells. *k*, *k*, *k*. Epithelium.

the chemical constitution of the urine and in convalescence from a toxic nephritis of mild grade repeated showers of true casts may be observed.

"Showers" of short granular cysts may precede the onset of a diabetic coma and in the exacerbations of actual nephritis the great and abrupt increase of casts in the urinary sediment sometimes justifies the term "shower," though with less aptness than in the other instances here detailed.

Prostatic Plugs.—*These may closely resemble the large hyaline or fibrinous cast, but usually carry, or are associated with, spermatozoa, and evidence of prostatic irritation or inflammation.*

THE GONOCOCCUS.—As indicating the infectivity of any persistent secretion after urethritis, however remote the original attack, the presence of gonococci is important.

Method.—The usual smear is made, dried in the air and fixed, and, indeed, may be made by the patient if no discharge is present save in the early morning or at irregular times. Ordinarily Jenner's stain or the alkaline methylene blue is sufficient.

The specific cocci appear like pairs of coffee beans with flat surfaces apposed and almost in contact and lie for the most part within the pus cells. If in doubt, one should apply Gram's stain, the organism being Gram-negative. In rare instances it may be necessary to use cultural methods as it is claimed that the *micrococcus catarrhalis* and the *diplococcus intracellularis meningitidis* are also negative to the Gram stain.

Usually intracellular.

Gram-negative.

Shreds.—*The so-called urethral thread, though common after gonorrheal infection, indicates no more or less than chronic congestion of the deep urethra.*

"Tripper Fäden."

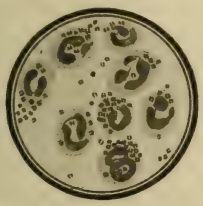


FIG. 87.—Gonococcus.

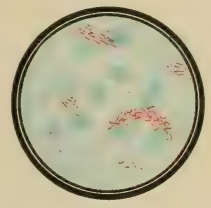


FIG. 88.—Tubercle bacilli in urine. Observe tendency to form groups.

EHRLICH'S TYPHOID DIAZO REACTION.—This test was introduced by Ehrlich in 1882, and, after meeting with much overpraise as well as much opposition and criticism born of misunderstanding and misapplication, has at last been accepted at its true value in many of the important clinics.

True value established.

Solution Required.—*Solution A.*—Hydrochloric acid, 50; distilled water, 1000; sulphanilic acid, q.s. ad. sat. (It should be *thoroughly* saturated.) *Solution B.—Sodium nitrite* (not nitrate), 0.5 *per cent.* (½ of 1 per cent.) solution in distilled water. *Solution C.—(Test solution.)—One hundred parts of A plus 1 part of B.*

Author's modification.

The original test solution of Ehrlich was made by adding to 40 parts of solution *A* 1 part of solution *B*. This the author has slightly modified by using 100 parts of *A* to 1 of *B*, with the effect of eliminating many disturbing factors and doubtful reactions. Dr. C. E. Simon suggested the ring method of testing described below.

Technic.—*Take equal parts of solution C and the urine. Shake thoroughly and add aqua ammoniæ in excess, allowing it to run gently down the tube so as to overlay the mixture below.*

If the reaction be present, a deep red band appears at the line where the ammonia meets the mixture. When shaken, it yields a pink or rose-colored foam, and, after standing several hours, a green precipitate forms.

Terminal reaction.

All-important Rules.—*The following rules must be carefully observed:* (1) *Use fresh urine.* (2) *See that the reaction is acid and the urine filtered.* (3) *Use a freshly mixed test solution.* (4) *Keep the sodium nitrite solution in a black bottle and in a cool place and renew it frequently (the sulphanilic solution*

Strict adherence to detail essential.

keeps indefinitely). (5) *Hold the tube near, but not against, a white background, the source of light being behind the observer. Artificial light should not be used.* (6) **Accept no color but a distinct red, and regard no reaction as a true one in which the solution when shaken does not yield a pink foam.** *False or imperfect reactions occur in which the band is of the proper color, but the foam is yellow or brown.**

Color reactions.

One may oftentimes follow the imperfect but suggestive reaction of early typhoid to its typical manifestation a day or two later.

Absurd errors.

The most absurd errors have arisen from a failure to observe the exact technic and precautions here outlined and adverse reports under high authority have been based upon the use of sodium *nitrate* instead of the necessary *nitrite*, or of weak instead of strong ammonia, and indeed one eminent observer omitted the end reaction altogether and naturally failed to get consistent results. Others have accepted the meaningless yellow and orange reactions with confusingly catholic positive results, or have even used a 5 *per cent.* solution of sodium nitrite (10 times the proper strength).

Sources of confusion.

True Value of the Reaction.—*The test is not pathognomonic, as was originally maintained by Ehrlich, but is constant in all severe forms of typhoid, appearing sometimes as early as the fourth or fifth day, though more generally at the end of the first week or ten days, and persisting until the fever begins to decline.*

Constant in severe typhoid.

The importance of the test is at the present time of course greatly diminished by the availability and specificity of the agglutination test and the even better, but less available, blood-culture method.

If the test is applied according to the author's method the *true and complete reaction* is absent in the ordinary forms of malaria, in appendicitis, pneumonia and in the earlier stages at least of miliary tuberculosis.

Differential value.

According to the author's experience it occurs only in some of the acute exanthemata; in certain cases of advanced malignant disease with fever; clarifyingly *late* in miliary tuberculosis and, unfortunately, in its typical form, in febrile cases associated with *septic absorption.*

Pseudo-reactions are found in a considerable number of diseases and the inclusion of the universally obtainable yellow and orange reactions has tended to discredit a good test.

Value in negation.

It is of great value in negation, for it is the author's firm belief that, within the period previously defined, it will be found present in all cases of severe typhoid, and that its persistent absence in any such case quite certainly negatives the diagnosis of typhoid fever.†

Exact cause unknown.

UREMIA.—Any form of Bright's disease may be associated with symptoms grouped under the head of "Uremia." The exact cause of this condition is still unknown, but its occurrence apparently depends upon the retention of certain products of retrograde metabolism distinctly toxic in their

Retained waste.

* Upon one or the other of these reefs, shipwreck apparently has befallen most of those who have tried this simple test.

† Recently Unverricht, in quoting Michaelis, an enthusiastic advocate, has raised anew the old question of color and demands a more definite reaction. *A deep red band and a pink foam should be demanded by every clinician before the individual reaction is accepted.*

nature, and perhaps, of certain toxic substances (*nephrolysins*) associated with the progressive destruction of renal tissue.*

Deficient Excretion.—*It is a well-known fact that no living organism can resist the poisonous effect of its own retained waste products. In the elimination of these substances the kidney plays the chief part and in almost all cases of uremia the phenomena of relative, or even absolute temporary renal insufficiency, are evident.*

The onset of uremia is attended not only by a diminished excretion of urinary solids in general, but of urea nitrogen itself, and the molecular concentration of the blood is correspondingly increased, as has been shown by the demonstration of a lowering of the freezing-point of the serum.

It must be remembered, however, that during an established seizure, and its subsidence, these conditions may be reversed.

Chiefly nervous.

Symptomatic Expression.—*The symptoms of uremia are made manifest chiefly through the nervous system, although the gastrointestinal and respiratory tracts are almost invariably involved in the severer forms.*

No concrete picture is presented in this curious toxemic state, the symptoms being irregular and inconstant in appearance, grouping and duration. Single symptoms of every grade of intensity may be the only signs, apart from the condition of the urine, which seldom fails to show a diminished excretion of solids.

Nervous Symptoms of Uremia.—*Almost every disease of the nervous system may be simulated by uremia but the cerebral symptoms are the most interesting and important.*

Cerebral.

Headache, drowsiness, stupor or coma may be found alone or in transition stages. There may be slight twitchings or the more terrible convulsive seizures closely simulating epilepsy, while violent outbreaks of acute mania, profound melancholia, or the so-called delusional insanity of Bright's disease such as occur in certain cases may easily lead to serious error in diagnosis or even to the committal of such patients to an asylum for the insane.†

Insanity of Bright's disease.

Varied symptomatology.

Sensory Symptoms.—*Various disturbances of general sensation may be encountered, such as itching, anesthesia, hyperesthesia, formication, and abnormal response to heat and cold.*

Vertigo is often a prominent feature and is usually associated with periods of high arterial tension or its opposite, marked circulatory depression, diminution of total solids or the predominance of the dyspeptic symptoms, often so prominent a feature in chronic renal disease.

* Although all of the older theories have been abandoned, the specific cause of uremia is as great a mystery as ever and the subject is, perhaps, even more involved in conflicting opinions. At least ten new theories of causation are now before the profession, all unproven.

† A remarkable case in the author's practice showed the existence of an uremic delusion, single but fixed and dominant, which lasted about three years, during which period the patient was converted from the warmest friendship to the most bitter enmity, returning abruptly to his first status of exaggerated loyalty at the end of the period. In this case the albuminuria was intermittent with frequent periods of marked deficiency of excretion, as indicated by estimation of the urinary solids. Several physicians had pronounced the man wholly free from disease, because of the absence of albumin from individual specimens of urine.

Transient paralyses.

Blindness and deafness.

Motor and Special-sense Symptoms.—The most remarkable symptoms of uremia are those extraordinary attacks of *transient paraplegia or hemiplegia* simulating true apoplexy, and the *disturbances of the special senses* resulting in tinnitus aurium, deafness or sudden temporary blindness (uremic amaurosis).*

Various more serious eye symptoms may co-exist with uremia and the diagnosis of chronic interstitial nephritis is often first made by the oculist, who discovers evidence of a neuro-retinitis of the albuminuric type. Uremic amaurosis usually lasts but one or two days and ordinarily follows some profound manifestation of uremia, such as coma or convulsions.

Time limit.

Cases presenting marked symptoms of actual albuminuric neuro-retinitis seldom live longer than one year.

Respiratory Symptoms.—*Cases of chronic nephritis are often peculiarly subject to inflammation of the pulmonary structures, but aside from these, we have curious disturbances of respiratory rhythm.*

Uremic dyspnea may be continuous, paroxysmal, alternating, or Cheyne-Stokes.

Uremic asthma.

The paroxysmal type is often mistaken for true asthma, the mode and time of onset being precisely the same.

Continuous dyspnea of uremic origin is not uncommon, and medical literature furnishes many evidences of the *ambulatory Cheyne-Stokes type.*†

Misleading symptoms.

Gastrointestinal Symptoms.—*These so closely simulate various independent diseases as to render diagnosis impossible, except by recourse to the urinary examination and particularly the estimation of blood pressure, the urinary solids, and the use of the phenolsulphonephthalein test whenever possible.*

Minor uremias.

As previously stated, these uremic symptoms may be evident in any case of nephritis. They may also be present in minor degrees, at least, in the absence of demonstrable nephritis, though many if not all of such cases represent probably an early chronic nephritis. In all observed by the author the functional activity or permeability of the kidney has been temporarily reduced. Its most extreme and *bizarre* manifestations are encountered in chronic interstitial nephritis, or, even more commonly, in chronic parenchymatous nephritis‡ during periods of low urea excretion.

Extreme forms.

The Onset.—*The onset of uremia of the severer types is almost invariably associated with lessened quantity of urine, excessively high arterial tension, and a sharp reduction in total solids and urea, or almost complete impermeability*

* Amaurosis, *i.e.*, blindness usually temporary and removable and without apparent lesion may also be encountered in hysteria, migraine, acute severe hemorrhages, tobacco and cocain habituation and diabetes.

† In one case of interstitial nephritis in the author's practice, *typical* Cheyne-Stokes breathing was present at night for over three years before the patient's death and was occasionally observed in the daytime during hours of active business.

‡ A case under the author's observation for five years showed during that period nearly every symptom to which reference has been made in a preceding page, and among these were attacks closely simulating hysteria, but promptly removed by radical therapeutic measures, while no less than three times before death the patient was found in deep uremic coma.

to solids as proven by the phenolsulphonephthalein and other tests. In the rarest instances decided polyuria precedes the attack.

Uremic Coma.—This may closely simulate that of apoplexy in which condition interstitial nephritis plays so large a part, but is rarely so sudden in onset and is more generally preceded by convulsions, a history of which should be carefully sought. Such cases are the *bête noire* of the hospital physician on account of the difficulties attending "emergency" differentiation.

The following points should be carefully noted: (*a*) *Examination of the urine obtained by catheterization is of the first importance, as it may yield evidence of an active or chronic nephritis or show a marked reduction in solids.* (*b*) *A history of antecedent convulsions or convulsive movements may be obtainable.* (*c*) *The pupils yield no certain signs.* (*d*) *Paralyses are rare, though transient hemiplegia is a rare possibility and there may be muscular twitching and rigidity of the extremities if severe convulsions have occurred.* (*e*) *The temperature may be elevated, but usually it is normal or subnormal.* (*f*) *Opthalmoscopic examination may or may not show retinal changes.* (*g*) *The general aspect of the patient may clearly indicate the existence of renal disease.* (*g*) *No absolute dependence can be placed upon the odor of the breath as indicating renal toxemia, yet it is strongly suggestive and helpful.*

High Blood Pressure.—*High arterial pressure may prove a most valuable and suggestive finding.*

Uremia vs. Acetonemia.—The uremic odor is sweet, nauseating, chloroform-like and may pervade a whole apartment, but is found in connection with profound toxemia, resulting from cardiac failure, advanced hepatic disease, renal disease and sometimes in connection with malignant growths. It lacks the peculiar quality of the breath encountered in diabetic coma, in which there is an actual fruity fragrance of the most penetrating quality.

CRYOSCOPY.—The introduction of cryoscopy by Korányi represents an effort to secure more exact information especially concerning renal activity and efficiency by determining the freezing point of the blood and urine. The greater the molecular concentration of a watery fluid the lower is its freezing point, and assuming that the average freezing point of normal blood ranges from $-0.56°$ to $-0.58°$C., and that of urine between $-0.9°$ and $-2°$C., it is evident that the lower freezing point on the part of the blood indicates a greater concentration and that a higher figure for the urine indicates a lessened molecular content.

Clinically useless.

It further appeared that a test of the urine taken by catheterization from each kidney might determine the relative functional activity of those organs and that the procedure would prove a guide to the surgeon in nephrectomy as well as to the physician in connection with the so-called uremic states and renal inefficiency.

As a matter of experience the cryoscope has proven a distinct disappointment, being wholly untrustworthy in ordinary clinical urinary work by reason of its extreme delicacy and the serious errors incident to its use.

Untrustworthy.

No hard and fast lines.

THE CLASSIFICATION OF RENAL DISEASES.—The student would be spared much distress of mind and disillusionment were it more generally taught that a *rigid clinical classification* of renal lesions is an absurdity.

Blending of lesions.

We may divide the nephritides into acute or chronic and distinguish anatomically certain leading types such as the acute and chronic parenchymatous nephritis, chronic primary interstitial nephritis, and amyloid degeneration, but neither clinically nor anatomically can we draw hard and fast lines.

We know little of the "real beginnings" of an interstitial nephritis or of that vague zone where "functional changes" end and "degenerative changes" begin. We do know that there is no "interstitial nephritis" without some degree of parenchymatous change, and vice versa.

Classifications are Artificial.—*In the main, all classifications are clinical conveniences based upon the preponderance of clinical evidence or of suggested anatomic changes, rather than upon clean-cut and convincing data.*

These incontrovertible facts should discourage fine-spun diagnosis, yet emphasize the value of thoroughgoing intelligent examination.

All nephritides "mixed."

Arbitrary grouping is a teaching necessity hallowed by custom, but let us neither forget that no man has defined successfully "physiologic albuminuria" in clinical terms, nor lose sight of the fact that all forms of actual nephritis are in some degree "mixed."

Diagnostic reverses.

Life insurance companies find "physiologic albuminurias" expensive and clinicians are sometimes appalled by the scant autopsy findings in apparently clean-cut nephritides or, rarely, astounded by the demonstration of an acute nephritis in a case yielding no clinical urinary symptoms.

RENAL INADEQUACY.—Aside from cryoscopy and the estimation of urinary solids, many efforts have been made to determine directly and by simple means the functional efficiency or inefficiency of the kidneys, especially with relation to actual or suspected medical or surgical diseases of those organs, but until recently no method had been evolved which was free from serious objection and, at times, gross error.

McLean's method, already described, is not adapted to the use of the practitioner (see p. 203).

Total nitrogen.

The accurate estimation of total nitrogen can hardly be adapted to the examination of specimens obtained by ureteral catheterization which represents small separate specimens, and furthermore involves an absolute knowledge of the ingesta, of excretion through channels other than the kidney, a complicated chemical estimation, and repeated tests covering a considerable period; all of which requirements *place it entirely outside the possibilities of the everyday clinical work of the general practitioner.*

Rough estimations are of course extremely helpful and at times of great value but all medical men have felt keenly the need of some method at once simple and accurate.

Methylene Blue.—Achard and Castaigne some years ago recommended and used methylene blue for the determination of the functional activity of the kidneys, using intramuscular injections and noting the time elapsing

between its administration and the subsequent discoloration of the urine as well as the approximate amount of the coloring matter recovered. It has been found, however, that the test is of little value as a measure of renal insufficiency, the elimination being unmodified in parenchymatous and amyloid nephritis and too widely variable in different cases of interstitial nephritis.

The Relative Toxicity of Urines.—Bouchard's method of determining the relative toxicity by injecting urine into guinea-pigs is entitled to little respect, nor is it in any way adapted to ordinary clinical work.

The Phloridzin Test.—It is probable that the sugar excretion following the hypodermic use of the glucoside phloridzin furnishes a reasonably accurate index of renal *secretory* activity.

Test.—1 c.c. of a sterile 1:200 watery solution of the glucoside, gently heated to dissolve any persisting crystals, is introduced into the tissues. Sugar should appear in the urine within from thirty to sixty minutes and sugar excretion should cease within four hours. The total amount excreted varies from 0.5 to 2.5 grams.

If the functional capacity of each kidney is to be determined, the ureteral catheters should be introduced and left in position during the whole test period.

No sugar, or but minimal amounts, will be secreted in acute or subacute nephritis and advanced interstitial nephritis.

As the clinical evidences of the first two forms is almost invariably frank, the test can only be of great value in certain rare cases of interstitial nephritis.

Determination of Electric Conductivity.—This is little more than a complicated, and clinically unimportant, method of estimating the mineral constituents of the urine.

THE PHENOLSULPHONEPHTHALEIN TEST.—*The Best Procedure.*— Simple and accurate.
Rowntree and Geraghty have introduced a test of renal functional activity* which has maintained its place for several years and represents the highest degree of accuracy so far obtained by simple and practical methods. Renal.

The procedure proved of distinct usefulness and value in the author's clinic.

Drug.—The drug is a non-toxic, bright red powder moderately soluble in alcohol and water and readily soluble in the presence of alkalies.

Normal Excretion.—*It is excreted with almost incredible rapidity, appearing in the urine a few moments (five to eleven) after hypodermic injection, and being eliminated in normal cases almost wholly within two hours. From* 50 *to* 60 *per cent. of the drug should be excreted during the first hour, and from* 70–90 *per cent. by the end of the second.* Time limits.

Test Solution.—0.6 gram of the phenolsulphonephthalein and 0.84 c.c. of a 2/N NaOH solution (8 per cent.) are added to normal salt solution sufficient to make 100 c.c. The addition of a few drops more of the 2/N NaOH

* It should be thoroughly understood that normal excretion does not prove that the kidneys are free from disease. It does show that, at the time, the renal function is not seriously affected. We do not know what degree of change is required to produce recognizable impermeability.

solution to the injection fluid at the time it is used changes the color of the test fluid to a wine red and renders it non-irritating to the tissues.

Technic.—(*a*) *The patient is given* 300 to 400 *c.c. of water to insure free secretion.* (*b*) *A catheter is passed into and retained in the bladder after this viscus is completely emptied.* (*c*) *At a carefully noted time, twenty to thirty minutes after the ingestion of the water,* 1 *c.c. of the test solution* (6 *mg. of the drug) to which* 2 *or* 3 *additional drops of the* 2 */NNaOH solution have been added to prevent irritation, is injected into the upper arm.* (*d*) *From this time all of the urine is led into a test-tube containing a drop of* 25 *per cent. sodium hydrate solution and the first appearance of a pink color carefully noted.*

This represents the initial excretion period which normally varies between five and eleven minutes.

Obstructive vs. Unobstructive Cases.—If no obstruction to micturition exists the catheter now may be removed, the patient voiding at the end of both the first and second hours.

In obstructive cases the catheter is clamped and maintained *in situ* until two hours have elapsed, the bladder being drained at the end of each hourly period.

Colorimeter.—The amount excreted is measured by a colorimeter.* Each hour's voiding, being measured and its specific gravity determined, is placed in a 1-liter volumetric flask and *treated with sufficient* 25 *per cent. NaOH solution to alkalinize it and thus develop the maximum color, a brilliant purple red.*

End color reaction.

Sufficient distilled water to bring the solution to the 1-liter mark is then added, the mixture thoroughly shaken up and a filtered portion taken for comparison with a standard solution consisting of 1 c.c. of the test solution in 1 liter of water, plus 1 or 2 drops of a 25 per cent. NaOH solution.

Standard color.

The standard solution is accurately adjusted to the 10-mm. mark of the scale and compared with the unknown solution, the percentage of drug excreted being calculated by the differences in scale required to secure uniformity of color in the two solutions.†

Clinical Application.—In renal disease, both parenchymatous and interstitial, but especially the latter, the excretion is retarded and greatly diminished if the kidneys are insufficient.

Advantages Claimed for the Test.—Its originators claim for it the following advantages:

(*a*) *It represents a non-toxic agent promptly secreted by normal kidneys, or by diseased kidneys, while functionally efficient, but retained for suggestively longer periods in actual renal insufficiency.*

* Geraghty and Rowntree's recent modification of the Autenrieth-Königsberger colorimeter permits a direct percentage reading by an indicator on the scale of the instrument. (See also "Kuttner's colorimeter," described in "Blood" section.)

† As the standard is 10 it is evident that a reading of 20 for the unknown solution indicates the presence of but 50 per cent. as much dye as is contained in the standard solution (3 mg.) which represents but one-half the quantity of dye injected for the test (6 mg.). Hence the reading of 20 on the scale indicates an excretion of but 25 per cent. of the dye and the same proportion will be true for any other readings obtained.

(*b*) *The brilliant red of the solution lends itself readily to colorimetric determinations.*

(*c*) *In cardiorenal cases the drug may show clearly the extent of actual renal insufficiency inasmuch as simple chronic congestion, save in the most extreme degrees of incompensation, affects excretion but slightly as compared with actual nephritis.* Differential value.

(*d*) *It permits better prognosis, forecasts or determines the presence of uremia and is of great importance with relation to proposed surgical procedure, and in warning the obstetrician of impending eclampsia.* Prognosis and prediction.

(*e*) *Within reasonable limits and in conjunction with ureteral catheterization it permits an estimate of the relative functionating values of the two kidneys.* Relative excretion.

Uremic Seizures.—*During an actual uremic attack the elimination during two hours is almost nil.*

Impending Attacks of Uremia.—*In any nephritic patient a showing of* 10 *per cent. or less indicates an impending uremic seizure.*

CHRONIC PASSIVE CONGESTION OF THE KIDNEY

The Kidney of Stasis.—This is always obstructive in the clinical sense, whether the primary disease be of the heart or pericardium, lungs or liver, pressure on the renal veins by abdominal tumors (including the pregnant uterus and ascitic fluid) or the cardiac weakness of prolonged exhausting disease. Venous obstruction.

Minor degrees of passive venous congestion are much more common than is generally taught or believed.

Morbid Anatomy.—The kidneys ordinarily show a simple venous hyperemia though in cardiac incompensations of long standing or such as result from or are associated with an actual nephritis, both parenchymatous and interstitial changes may be manifest. Indeed simple cases of long standing may show the well-known "cyanotic induration" kidney in which marked atrophy of the secreting structures and some interstitial changes may be manifest. Often "mixed."

Symptoms.—There are no symptoms of importance save the urinary findings and those referable to the primary lesions.

Urine in Chronic Passive Congestion.—*The total amount* is diminished, the total solids approximately or actually normal.

Reaction.—Acid.

Color.—Distinctly high save in diseases characterized by marked impairment of nutrition.

Albumin is present only as a trace, except in pregnancy when large amounts may be found, as is also the case when decided parenchymatous changes co-exist.

The amount of albumin in simple passive congestion usually varies inversely with changes in the heart strength, and such variations often assist one in the diagnosis of the minor cardiac decompensations. Important fact.

Microscopic Findings.—An occasional hyaline or finely granular cast, together with a few renal cells constitute the usual findings.

THE KIDNEY OF PREGNANCY.—*There is no single and distinct anatomic type which fits this clinical condition.*

Even though the condition may be one of simple pressure hyperemia, the amount of albumin is likely to be larger than in other instances of chronic passive congestion, and it must not be forgotten that active hyperemia or acute or chronic inflammatory diseases of the kidneys may accompany this state, a toxic tubular nephritis is the commoner type.

Caution.

In this connection it is of the utmost importance that specimens of urine be submitted frequently to the attending physician and that these represent the twenty-four hours' urine, and not the individual haphazard specimen.

Total solids.

Furthermore, the essential feature of the examination, as indicating danger to the patient, lies in the estimation of urea, *the blood pressure* and in the use of the Rowntree and Geraghty test when deemed necessary. This point is often overlooked by the practitioner, who may mistakenly regard the amount of albumin as the important feature.

ACUTE SEVERE CONGESTION AND ACUTE NEPHRITIS

Faulty classification.

Clinically Inseparable.—*These conditions should be considered together, inasmuch as the causes operating to produce them are essentially identical and, further, because no line can be drawn clinically between actual acute nephritis and* **severe** *congestion, save that based upon the duration of symptoms.*

Mild congestion of the arterial type occurs under many conditions of slight potency, and is the antithesis of the severer types, the amount of urine being increased, but a small trace of albumin being present, and the urinary solids being actually or approximately normal.

Etiologic Factors.—*Irritating Drugs.*—Among these are copaiba, cubebs, sandalwood oil, turpentine, cantharides, carbolic acid, phenol compounds generally, arsenic, lead and mercury. These cause trouble only when ingested in excessive doses or for too long a period.

Insoluble Urinary Constituents.—Uric acid, calcium oxalate, cystin, acid urates and phosphates, *as constituents of "gravel" or "calculi,"* are the chief factors.

Toxins.—These may be divided into three groups: (1) Those associated with acute febrile infections and especially with the exanthemata and certain virulent tropical diseases, of which yellow fever is the chief.

Chief cause.

(2) *Suppuration with septic absorption which should include the acute and chronic disease of the tonsils, peridental abscesses, chronic prostatitis, sinus diseases and like obscure sources of infection.* (3) Chronic diseases, such as syphilis, gout, malaria, pulmonary tuberculosis, diabetes mellitus and certain of the anemias. *Streptococcus infections are especially potent.*

The toxic albuminurias may be extremely mild and transitory, as acute clinical conditions, and are best exemplified by the so-called **"febrile"** albuminuria. Whether such conditions leave permanent lesions behind them or not, depends upon the virulence of the toxemia and the condition of the

patient prior and subsequent to the attack. Clinically, they represent no more than a mild renal congestion.

Nervous Influences.—Formerly it has been customary to place under this head the nephritis attending such conditions as acute mania, delirium tremens and the so-called ascending or reflex congestions connected with diseases of the bladder, seminal vesicles, urethra and the prostate. This is incorrect, for the ingestion of large amounts of alcohol is in itself sufficient to produce marked albuminuria and reduce resistance to infection from within and without the body. Negligible.

Add to this the exposure to the elements and the excessive exhaustion attending delirium tremens, and acute renal congestion or an actual nephritis is readily explained. In many cases of acute mania, nephritis is the antecedent factor of which mania may be but a symptom. In delirium tremens the habits of the individual, the likelihood of preëxisting disease, the exposure and exhaustion attending a debauch, leave little room for the older classification. In the case of the so-called reflex or ascending congestion, save in the rarest instances, we are dealing with an extension of infection. "Reflex" a Misnomer.

Pregnancy.—The acute renal disturbances of pregnancy are associated with a marked diminution in the amount of urine, a profusion of casts and albumin, and an associated edema of varying degree, often decided or even excessive. Toxic symptoms are decided and the condition tends to the production of convulsive seizures of an uremic type. Blood cells are present in the urine in many or most instances but not to the degree usually observed in connection with the actual acute nephritis of the ordinary type. Emptying the uterus, if timely, is followed by a rapid recession of the symptoms and, in most instances observed by the writer, an uneventful and complete recovery on the part of the patient.

General Causes.—Sexual excesses, the excretion of large quantities of bile, and extreme concentration of the urine seem adequate to produce mild congestion.

Fatigue, mental or physical, particularly when combined with exposure to cold or wet, would seem to be a predisposing cause, but the most potent active factors are the chronic infective foci already mentioned.

Morbid Anatomy.—The complex description and varying terminology in use embarrass the student when he approaches the subject of renal pathology.

Essential Factors.—*All renal lesions, acute or chronic, actually fall under four heads:*

1. *Those in which glomerular changes predominate.*
2. *Those in which tubular changes dominate.*
3. *Those in which the interstitial changes are most prominent.*
4. *Those in which all structures are affected without determinative dominance of any one group.*

The finer classification and subdivisions are of pathologic interest only and in any *severe* nephritis, acute or chronic, one finds all structures involved in some degree, so that it becomes simply a question of *relative predominance.*

Clinical Aspect.—*In an acute nephritis or severe acute congestion of the*

kidney, any one of the three pathologic types, glomerular, tubular, or interstitial, may be primary or predominant.

Aside from the rare and clinically unrecognizable *acute non-suppurative interstitial nephritis*, the three fundamental structures are in some degree involved and, in any event, no consistently accurate clinical distinction can be made.

Microscopic Pathologic Findings.—We ordinarily find cloudy swelling, desquamation, and hyaline, dropsical and fatty degeneration or complete necrosis of the epithelium of the tubules, which are crowded with inflammatory detritus. We are likely to find acute intracapillary glomerulitis, the capillaries being filled with cells and thrombi and the epithelium of the tuft and capsule being involved. The latter is crowded with blood cells and leucocytes, and interference with the renal circulation and nutrition, no less than with the excretion of the urine and contents, is inevitable. Furthermore, the loss of integrity on the part of the filtration apparatus must necessarily lead to the escape of albumin. In rare instances a portion only of the kidney may be involved, or the process may be unilateral.

Macroscopic Appearance.—The gross appearance of the kidney varies somewhat; it is ordinarily swollen and red, dripping blood on section, having a succulent feel and an easily stripped capsule. Rarely it may be pale and mottled, exuding on section a milky fluid (acute non-suppurative interstitial nephritis). The glomeruli are usually red and prominent, but may be pale and indistinct.

Variable onset.

GENERAL SYMPTOMS.—The onset may be sudden and frank, with chill and sharp temperature rise, but is usually gradual, afebrile, and insidious.

There is usually some pain or heaviness in the loins, a dry skin and increased frequency of micturition, the pulse tension increases, and nausea, vomiting, headache and thirst may be present.

The severity of the initial symptoms bears no definite relation to the type of the disease, save that those of simple acute congestion are ordinarily

Early edema.

milder and more evanescent. In many instances the first thing noted is puffiness under the eyes, the development of general edema, or even uremic convulsions or coma. As a rule, however, these two last symptoms occur only after several days of illness.

Hypertension.

Arterial Tension.—One of the best guides when nephritis complicates other disease is found in the arterial tension, which is usually but not invariably increased in the presence of either severe acute renal congestion or inflammation (180 to 200 mm.).

Fever.—Though ordinarily present, it is seldom high and often is masked by that of some primary disease of which the nephritis is a complication.

Peculiar facies.

Edema.—*The edema of acute nephritis is oftentimes extreme* and is likely to appear first in the eyelids and tissues of the face, the skin being pallid* and "pasty."

* A secondary anemia of considerable or excessive degree may be present but the "*pallor*" does not prove its presence nor conform to the degree of anemia when that does coexist.

In the extremities the swelling is comparatively firm, though pitting deeply on pressure, and, because of the blue veining on the dead-white background which is especially well marked on the child's skin is often termed "marble edema." Marble edema.

There is a special tendency to secondary complicating symptoms especially affecting the serous membranes and the myocardium and to attacks of edema of the glottis which, if not immediately relieved, may cause sudden death by suffocation. Serious complications.

Special Precautions.—At least once daily the attending physician should by careful examination exclude endocarditis and myocarditis, no less than pericarditis and pleurisy, with or without effusion, the condition last named often coming on rapidly and insidiously.

Edema of the glottis with urgent stenotic dyspnea usually demands immediate recourse to the most radical measures; the free scarification of the engorged tissues, prompt application of astringent preparations and, very often, an *emergency tracheotomy*. A *few moments* of indecision and delay has more than once caused death. Glottic edema.

URINARY FINDINGS.—The urinary findings in acute congestion vary with its severity, but tend to assume, *pari passu*, characteristics identical with those of acute nephritis.

It is evident that severe acute congestion can be differentiated from acute nephritis only by its shorter duration, but the mildness of the slighter grades makes their recognition easy. Acute congestion.

The student should refresh his memory regarding the subject of albuminuria and the finding and significance of casts before taking up the following section. The findings fall naturally under three heads:

First Stage.—*Quantity for twenty-four hours*, 100 *to* 400 *c.c.* *Color*, dark smoky or black. *Specific gravity*. This may be high or low for albumin may raise it even to 1030. *It is invariably low if albumin is removed. Reaction*, acid or, from large quantities of blood, slightly alkaline. *Solids*, the solids are absolutely diminished, urea being greatly lessened and the chlorides absent if dropsy is extreme. Acute stage. Lasts 5 to 10 days.

Sediment.—An abundant dark sediment is present. *Casts* are abundant, the urine containing dark granular, epithelial, fibrinous and blood casts with a few hyaline and fine granular forms. Casts carrying leucocytes, caudate pelvic cells, and round cells from calices are present in severe cases.

Albumin.—This varies from ¼ to 1 per cent. according to the degree of tubal involvement. Red blood cells are abundant, as are leucocytes and brown granular epithelial cells.

Second Stage.—*Quantity for twenty-four hours*, 600 to 1500 c.c. *Color*, dark and smoky. *Specific gravity*, 1015 to 1020, *Reaction*, acid. *Solids*, still diminished. 5 to 10 days

Albumin.—This will vary from ⅛ to ¼ per cent. diminishing *pari passu* with urine increase. Improvement.

Sediment.—Profuse and dark; *fatty changes now become evident through fatty casts, fatty renal cells and compound granule cells.* The amount of fat Characteristic changes.

in some degree measures both the improvement and the severity of the primary attack.

Convalescence.

Third Stage.—*Edema if originally present has now disappeared. Urine,* the quantity is increased (1500 to 4000 c.c.) and the tendency to polyuria may last for weeks. *Color,* pale or slightly smoky. *Reaction,* acid. *Specific gravity,* 1006 to 1020. *Solids,* normal or slightly increased by absorption of exudate.

Albumin.—This will vary from a mere trace to ⅛ of 1 per cent.

Sediment.—This is scant and of lighter color and contains abnormal blood cells (rings) (ghosts), and occasionally hyaline and granular casts both light and dark. Possibly an epithelial, fatty, or fibrinous cast may be found and a few renal cells. The casts and renal cells may carry fat droplets or abnormal blood cells, but as improvement continues the evidence of active degenerative and regenerative changes disappears and the urine at last becomes normal.

A common error.

Any chronic renal disease may undergo acute or subacute exacerbations and a most common diagnostic error lies in the failure to appreciate the fact that in adults most of the apparently acute cases are but exacerbations of an unrecognized chronic nephritis.

Prognosis.—The disease is less common and less fatal in dry than in damp climates. Children give a large mortality (30 per cent.), especially scarlatinal cases. Low tension, extremely high tension, major uremic symptoms, and serous effusions are ominous, and lack of any decided improvement after ten days means a *probability* of chronic disease. Persistent profound anemia is also a threatening sign *but recovery may take place even after one or two years.* Relapse and multiple relapses are frequent.

Portentous signs.

ACUTE FOCAL GLOMERULONEPHRITIS.—These cases are clinically undemonstrable as such, and represent focal embolic nephritis, involving especially the glomerular capillaries in minute widely scattered areas.

Albuminuria, a few red blood cells in the sediment, and, sometimes, fugitive edemas, constitute the only bases for diagnosis.

In the presence of "*endocarditis lenta,*" or other conditions of a similar sort, this condition may be assumed with some justification if the symptoms named above are present.

ACUTE INTERSTITIAL NON-SUPPURATIVE NEPHRITIS.—This interesting pathologic picture is revealed only at autopsy, and, chiefly, in fatal cases of scarlatina or, less frequently, acute rheumatic arthritis. As stated previously, the change is *almost* exclusively confined to the interstitial tissue, in which round cell infiltration is demonstrable. Clinical signs of renal involvement may be wholly absent during life.

CHRONIC PARENCHYMATOUS NEPHRITIS

Etiology.—*An antecedent acute nephritis, prolonged exposure to wet and cold under conditions of fatigue, chronic malarial infection, syphilis, alcoholism, chronic suppuration and chronic obscure infections are the common factors.*

The many sources of streptococcic infection must be considered, for this organism plays the chief part in all probability.

Morbid Anatomy.—*Glomerular, tubular, and interstitial changes are present, the two first predominating.*

The kidney is large, its capsule may be adherent, but usually strips easily from the pale and mottled kidney surface; section shows increased resistance and a swollen white cortex with curious areas of opacity.

Microscopic Findings.—These include obliterative hyaline degenerative changes in the vessels of the glomeruli, involving both the cells and the vessel walls, and swelling and nuclear proliferation of the tufts is evident. There is a tendency to connective-tissue ingrowth and to proliferation and desquamation of the capsular epithelium.

The tubules show degeneration, desquamation, and necrosis of their epithelium and edema and connective-tissue infiltration of the intertubal tissues.

Symptoms.—*The onset is insidious but the general symptoms usually become frank, anemia and edema alike being prominent.*

The various uremic phenomena are common, early, and recurrent, there being a special tendency to headache, dyspepsia, intractable neuralgias, nausea, vomiting and diarrhea. Pleurisy and pericarditis with effusion are frequently observed. Ulceration of the colon occasionally occurs. There is constant tendency to general anasarca, and quiet, insidious transudations into the pleura, pericardium, peritoneal cavity, and *tunica vaginalis testis* may complicate the case.

Although edema of the face and pallor are ordinarily the first noticeable objective signs, the edema usually appears promptly in the ankles and may at this time act much like the cardiac form though far more shifting and inconstant with respect both to degree and location. About the face, head, and neck it is particularly pronounced in the early morning.

The circulatory changes are less marked and constant than in interstitial cases, though the pulse tension is usually increased and may become very high as the disease progresses. The second aortic and apical first sound are in such cases markedly accentuated, the left ventricle enlarged and changes in the retina are common. In many cases the heart remains throughout a weak dilated or readily dilatable organ, the myocardium being persistently affected by the toxemia.

URINARY FINDINGS.—It is customary to separate the urinary symptoms into those of the active, as compared with the inactive stage. *It will be noted that the essential symptoms are the same under both conditions, i.e., a decided albuminuria, the presence of fatty casts, and marked diminution of the urinary solids.* The disease is especially liable to subacute exacerbations, giving to the urinary findings a resemblance to an acute nephritis of the early stage of convalescence.

Misleading exacerbations.

Active Stage.—*Amount*, 200 to 800 c.c. *Color*, high or pale, yellow or greenish, often smoky from exacerbation. *Reaction*, acid. *Specific gravity*, high from albumin, 1026 to 1035; but with this removed, low.

Solids, greatly diminished, especially chlorides and urea as in all dropsical cases of renal disease.

Albumin.—This is profuse and the urine may boil solid. One-half to two per cent. is the common variation (5 per cent. has been reported, but the average is 1 per cent.).

Sediment.—This is always considerable and one finds hyaline, granular, fatty and epithelial casts, fatty epithelium and compound granule cells.

Epithelial casts indicate in any form of nephritis a superadded or recent acute or subacute trouble. Free fat, fat crystals, cholesterin and waxy casts occur in the late stages. Red blood cells are present and may of course be very plentiful and associated with blood casts. The number varies with the intensity and stage of the active process.

Inactive Stage.—*Quantity*, 800 to 1200. *Color*, pale or greenish. *Reaction*, acid or neutral. *Specific gravity*, 1010 to 1015. *Solids*, greatly diminished. *Albumin*, ¼ to ½ per cent. *Sediment*, as in active stage.

Prognosis.—If the disease is strongly intrenched and not merely a protracted case of acute Bright's disease, its victims rarely recover, but die in a few years of pulmonary edema, general anasarca or uremia, the average duration of the disease being a little over two years.

Cases of Mixed Type.—*Cases are frequently observed which present evidences of marked tubular and glomerular involvement for long periods without excessive general edema, and ultimately become clinically almost typical cases of interstitial nephritis.*

In these mixed cases the urinary findings much more closely resemble cirrhotic kidney than do those assigned to the type described below.

A misnomer.

THE SMALL WHITE KIDNEY (*Secondary Contracted Kidney*).—This misnamed condition is supposed to represent an advanced stage of an unusually prolonged chronic parenchymatous nephritis with ultimate predominance of the interstitial elements. It represents actually a "mixed" nephritis.

Morbid Anatomy.—The kidney is *sometimes* small and yellowish but more often approximates the size of the large white kidney. Connective-tissue hyperplasia is marked, the capsule being thick and adherent, and the kidney surface under it rough and granular and on section there is increased resistance. The cortex is thin, pale, yellow, and covered with yellowish-white spots.

Microscopic Findings.—*The interstitial changes are marked*, there is arterial thickening, and the glomerular and tubular structures are degenerated and largely destroyed.

General Symptoms.—These are essentially those of a combination of parenchymatous and interstitial nephritis, changes in the heart and blood vessels being more pronounced than in the parenchymatous form.

Essential features, like interstitial form.

Urinary Findings.—*Quantity*, normal or slightly increased. *Color*, pale. *Reaction*, acid or neutral. *Specific gravity*, 1004 to 1010. *Total solids*, greatly diminished. *Albumin*, ¼ of 1 per cent. or less.

Sediment.—This is like that of chronic parenchymatous nephritis of the

"inactive" stage, save that casts are less abundant, and that, in some cases, late in the course of the disease, waxy casts are found in unusual numbers.

FOCAL ARTERIOSCLEROTIC FORM.—This represents the senile type of diseased kidney and is characterized pathologically by its tendency to maintain an approximately normal size, associated with a certain amount of induration.

The involvement of the vessels is patchy, irregular, and not confined to the arterioles. This condition is reflected in the macroscopic "patchy" depressions scattered over the surface of the kidney. In nearly every instance, the condition is associated with decided peripheral arteriosclerotic changes throughout the body. Urinary signs are scant, and may be wholly absent over long periods. *Arterial pressure* may be high, low, or inconstant and widely variable. Signs of renal insufficiency may be lacking up to the time of death.

A masked process.

CHRONIC INTERSTITIAL NEPHRITIS

("Gouty" kidney, "contracted" kidney, "cirrhotic," "granular," "sclerotic" or "small red" kidney, "chronic diffuse" nephritis, etc.)

Morbid Anatomy.—The kidneys are usually small, an extreme instance having been reported in which their combined weight was but 1½ ounces. The capsule is thickened and adheres to the dark red, nodular, granular surface, and section shows an increase in resistance. The arteries are prominent, the cortex very thin, the pyramids wasted and the pelvic fat increased.

Microscopic Changes.—These are essentially those of connective-tissue overgrowth, with atrophy and degeneration of both glomerular and tubular structures. Many of the glomeruli and tubules are entirely destroyed.

Etiology.—Here, as in arteriosclerosis and aneurysm, the worship of Venus, Bacchus and Vulcan have long been regarded as the primary factors in causation, though to this group one might well add Minerva and also Mammon, the non-Olympian.

In other words, overwork (mental and physical), syphilis, sexual excess, exposure and privation, heavy eating and drinking, all play a prominent part.

The author believes, nevertheless, that with the exception of lues, these are merely predisposing factors.

Chronic foci of infection and the obscure and subtle changes wrought by past illnesses are probably the most potent elements.

Arteriosclerosis is a prominent feature in cases of interstitial nephritis and one is not surprised to find what seems like a direct hereditary predisposition to the development of the latter disease. Lead poisoning, lues, alcoholic excess, chronic malaria, and the various forms of gout are common and prominent in the case histories. As in the case of arteriosclerosis proper, we here find the "young old man," old by virtue of inherited vascular weakness or gout, or through sexual excesses, syphilis, or excessive mental strain. It

is wholly probable, however, that acute infections or obscure chronic infections of the *streptococcus* type play a large part or the leading rôle.

The chronic or remittent toxemia of interstitial nephritis itself doubtless promotes and advances arteriosclerotic changes in the general arterial system.

General Symptomatology.—*The pallor, edema and the frankly albuminous and cast-filled urine of chronic parenchymatous nephritis makes mistaken diagnosis in the case of that form of nephritis unpardonable. The reverse is true of chronic interstitial nephritis.*

Incredibly slow.

In the earlier clinically recognizable stages of the disease the patient may appear to enjoy unusually good health, this being particularly true of the sthenic type. Pallor, edema, and pigmentation are late symptoms, and the urinary findings are sometimes both variable and obscure.

Often unrecognized.

Many cases die under other diagnoses and in the case of many others, recognition is deferred until autopsy.

It must be borne in mind also that most of these cases exist for years before clinical recognition is possible. We know but little of such ailments in their earlier pathologic stages.

Urine and circulatory organs.

Much dependence must be placed upon the secondary signs in the heart and blood vessels, and the urinary examination demands care and the intelligent application of a full knowledge of the vagaries of this extraordinary disease.

Silent hypertension.

Most patients with well-established lesions seem to become habituated to, and carry well an arterial systolic pressure of 170 to 180 or 185 mm. of mercury, but usually show symptoms if the higher figures are much exceeded.

Certain hypertension symptoms.

Sensory Disturbances.—These symptoms are often obscure and consist chiefly of numbness, tingling or a sense of weakness in the extremities.

The general symptoms are essentially those of arterial hypertension or uremia, either or both of which may be present at any time in any of their various forms. The severer manifestations of uremia may be postponed to the very end of the case, or never occur. (The section dealing with uremia should be carefully reviewed.)

Circulatory Signs.—*Advanced interstitial (diffuse arteriolar) nephritis is invariably attended by increased arterial tension, at least until the extreme terminal cardiac incompensation supervenes.*

This hypertension is indicated by the sphygmomanometric readings and reflected in the overacting or hypertrophied left ventricle, a hard radial pulse, and, usually, marked accentuation of the aortic second sound and mitral first sounds, with or without reduplication of the latter.

Significance of extreme hypertension.

In advanced cases with uremic manifestations the systolic pressure, ordinarily 165 or 170 to 215, may reach 250 or even 300 mm., though the last figure has been seen by the author only just before or a few days preceding an apoplectic stroke, uremic convulsions or coma.

Most authorities state that abnormal tension follows rather than precedes definite urinary findings, but the author believes this to be an exact reversal of the true sequence.

It should be understood that in many cases of decided arterial hypertension,

the aortic sound is not heard as a sharply accentuated tone. Many errors result from the usual teaching with reference to this matter.

A misconception.

In many cases, early or late, the classic accentuation of heart sounds is lost or modified by reason of valvular changes or a weakened myocardium.

The sphygmomanometer will ultimately give us, in all probability, our first knowledge of the true "early stages" of interstitial nephritis and the phenolsulphonephthalein test may also aid us. Neither, however, can take us back more than a part of the way to the beginnings of interstitial nephritis of the common type.

Arterial tension is often maintained at a high figure even when death is impending and cardiac insufficiency extreme, and indeed excessive tension in any case is often found to be due in part to stasis and decided amelioration may follow brisk cardiac stimulation.*

Stasis hypertension.

Edema.—*When marked edema appears in this disease, it is usually due chiefly to cardiac failure, and shows the characteristics of a passive congestion edema, the most dependent portion being first involved, as is the case in primary valvular or myocardial diseases of the heart.*

Usually cardiac.

In many cases of established interstitial nephritis, however, one may see for years a curious fullness of the eyelids, especially the lower. In the morning the skin appears thinned, pearly, and almost translucent, whereas later in the day the fluid may disappear and the delicate integument subsides into fine wrinkles.

A suggestive sign.

Some of the cases come under the head of "*mixed nephritis*" and in these the renal facies is likely to be more marked and unmistakable, owing to the presence of parenchymatous degeneration and glomerular changes.

Mixed cases.

Traube's Heart.—In the case of the heart of long established renal disease and arterial hypertension the whole organ may be affected, indicating quite clearly an underlying general toxemia.

This is not always the case, however, and in many instances observed by the author the left ventricle only has shown change during many years.

The Fundus Oculi.—In these as in other forms of renal disease, the eye changes may be pronounced and important; indeed, many cases are referred by the oculist to the internist under a correct diagnosis without urinary examination. The usual changes consist of flame-shaped hemorrhages, papillitis, retinal edema, or peculiar fawn-colored patches, radiating from the macula lutea. Glaucoma is not uncommon, and uremic amaurosis may occur. As a rule, these eye changes indicate a fatal termination in a short period, yet the author has observed one case in which the original diagnosis was made by the oculist, and a subsequent glaucoma led to enucleation of the affected eye; yet the patient is still living after fifteen years of apparent good health. The rule admits few exceptions nevertheless.

Warning.

Readily recognized.

Prognostic value.

Respiratory Tract.—*It is important to remember the special liability of renal cases to attacks of bronchitis, pleurisy, asthma, edema of the glottis, and dyspnea in its various forms.*

Pleurisy, asthma, and glottic edema.

* Probably because of the varying degrees of asphyxial irritation of the vasomotor center.

A massive pleural or pericardial transudate may come on so quietly as to attract no attention save through the embarrassment in respiration. In a case recently observed the acute edema of the laryngeal tissues, that followed a sharp attack of tonsillitis, proved to be due to the acute toxic congestion superimposed upon an old nephritis associated with passive congestion due to a weakened myocardium.

The inflammation of serous membranes which may complicate Bright's disease are for the most part, doubtless, mere streptococcic infections which attack by preference a weakened body.

Scant findings.

URINARY FINDINGS.—*The essential symptoms in the* **typical case** *consist in the increased amount of night urine, attended by increased frequency of micturition; a total increase for the twenty-four hours; a more or less persistent and extreme tendency to diminution in the total amount of solids; traces of albumin, and a few hyaline and granular casts.*

Nightly increase.

Increased night frequency may be due to causes other than nephritis, such as enlarged prostate, chronic irritability of the neck of the bladder, or cystitis, but when the symptom is associated with a marked disturbance of the normal ratio between night and day, the symptom becomes one of primary importance.

Often important.

Variable.

Diminution of Solids.—*Urea Variations.*—A man with interstitial nephritis may during years pass a normal or nearly normal amount of urinary solids the greater part of that time, though unrecognized periods of retention undoubtedly occur.

Valuable warnings.

In the later stages of the disease the excretion of solids will almost invariably fall below normal, and this is especially true of urea. Valuable information is often given by sudden drops in urea excretion, associated with heightened arterial tension and in many such cases, *under close observation*, one may avert an impending attack of uremia or apoplexy through these warnings.

Watch urea excretion.

Deceptive findings.

Albumin.—Albumin may be present continuously or intermittently, at one time of the day and not at another, but in uncomplicated cases will be found only in small traces. The specimens least likely to show it are those passed in the early morning, those most likely being the ones voided several hours after a full meal taken in the middle of the day or after a heavy dinner at night. Exposure to cold, slight infection, physical exertion and the process of digestion seem to increase the albumin output.

A negative examination of the single specimen, even of the twenty-four hours' urine, proves no man free from interstitial nephritis.

Briefly we may summarize as follows: *Color*, usually pale. *Specific gravity*, low—1002 to 1014. *Total amount*, 2000 to 4000 c.c.; may reach 7000 or 8000 c.c. *Reaction*, faintly acid or neutral. *Urinary solids*, diminished, coloring matter diminished, except indoxyl, which is usually increased. *Albumin*, usually a trace, rarely reaches ¼ of 1 per cent. in the later stages.

Sediment.—This is usually slight or absent macroscopically in undecomposed urine.

Casts.—The casts are chiefly the hyaline and faintly granular varieties.

Cells.—A few renal cells may be found and some cases are associated with *cylindroid storms*. Even with the use of the centrifuge casts may be very few in number.

Note.—*All urinary findings may be modified by a superadded passive congestion due to a failing heart or by a subacute exacerbation, though the latter is unusual in the straight interstitial type.*

Watch the heart.

Defective elimination, *as shown by the phenolsulphonephthalein test, is especially marked in this form of nephritis.*

Blood-pressure.—It is wholly probable that an abnormal arterial tension is the earliest sign available for the practitioner.

Symptoms of the Terminal Stage.—*Cardiac and Renal Symptoms.*—There may be passive congestion due to a failing heart, resulting in an increased amount of albumin, and, as a rule, more numerous casts showing coarser granules and an occasional waxy cast. The amount of urine is diminished, the total solids are low, though the specific gravity may be *relatively* high. The general appearance of the patient is that of a cardiac edema and such cases offer great therapeutic difficulties especially to those who undervalue the need of supporting the weakened heart.

Support the heart.

Uremic symptoms usually become prominent and such cases may terminate in pulmonary edema or uremic coma.

Prognosis.—*Duration Indefinite.*—Inasmuch as we know little or nothing of its beginnings, we cannot say for how long a period the individual may endure changes of interstitial nephritis nor can we recognize its earliest stages. It is safe to assume that cases have endured in some instances for forty years, and that the average duration is a long one.

Extreme chronicity.

Through inheritance of cases, the author has been able to follow certain patients through a known period of at least twenty-five years, and is prepared to believe that some of them will add several years more before the end comes.*

Such represent, however, the few surviving out of a large group observed, and in most instances of interstitial nephritis, as in chronic myocardial disease, a patient is far advanced before the disease is recognized or even encountered.

Belated recognition.

Subacute Attacks.—Some cases, and especially those of the "mixed" type, are greatly jeopardized by the occasional occurrence of subacute attacks which may be sufficiently severe to produce symptoms of acute congestion or be so mild as to show little more than a few blood cells or epithelial casts.

Intercurrent exacerbations.

Prognostic Factors.—*The occurrence of certain characteristic retinal changes, symptoms of marked and persistent or decidedly resistant cardiac incompensation and the appearance of waxy casts usually mean that the terminal*

Heralds of death.

* One patient whose case seemed very definitely to represent the consecutive small white kidney, died a short time ago in uremic coma after being under observation for twenty-four years. Strangely enough he had lived the life of a *bon vivant* for the greater part of that entire period upon the old theory of "a short life and a merry one." The old physician who attended him during his original attack twenty-six years before gave him two years to live and he proposed and proceeded to make the most of them according to his lights.

stage has been reached though death may be postponed for a year or two in favorable cases.

Apoplexy.—As might be expected an apoplexy is a very common event, terminal or otherwise.

TRENCH NEPHRITIS.—The attention of military authorities was directed to nephritis during the Great War of 1914–18 because of the extent to which this disease played a part in the morbidity of attending Army service especially at the front, although there seems to have been nothing in the types of the disease occurring which would permit any separate classification or demand detailed consideration here.

The term "trench nephritis" is not an accurate one, inasmuch as the disease occurred amongst men in all branches of the service though naturally to a greater extent amongst those whose duties were especially arduous and who were exposed most to cold, wet, and profound fatigue.

The conditions of service at the front throughout the war were peculiarly trying, as all men know; minor infections were frequent and the relighting of hidden foci extremely common. Hence every condition suited to the redevelopment of an old nephritis or the excitation of an acute congestion or an actual acute nephritis was present.

By far the greater number of cases were of such a nature as to suggest apparently the presence of a relatively severe degree of acute congestion or an actual acute nephritis.

It would appear that for the most part the duration of these attacks was relatively short and the mortality low.

AMYLOID KIDNEY

Urinary Findings Indeterminate.—*From the urine alone the diagnosis cannot be made in this disease, but the presence of conditions with which it is known to be associated, viz., chronic suppuration, chronic ulcerative tuberculosis, or inveterate syphilis, is suggestive, particularly if hepatic and splenic enlargement co-exist.*

Diagnosis by association.

Symptoms.—*Polyuria with a large amount of albumin, hyaline casts* in variable numbers, associated not infrequently with granular and occasionally fatty and waxy casts, constitute the urinary findings.

Significant syndrome.

The most significant diagnostic feature is the combination of polyuria, low specific gravity and a large albumin content in a patient showing the amyloid changes in the spleen and liver which usually occur in this form of nephritis.

Spleen and liver.

Edema and effusions occur as in other forms and cachectic *facies* is common.

Valuable guide.

Other Differential Factors.—*The urinary findings vary greatly and the total solids are usually little affected. The consequent absence of marked toxic hypertension and cardiac changes is of some diagnostic significance.*

MOVABLE AND FLOATING KIDNEY.—The normal kidney may or may not be palpable, but usually, in the female, its lower border may be detected under proper conditions of muscular relaxation and correct technic.

Bimanual palpation.

Technic of Palpation.—To palpate the kidney, one hand should be placed over or just below the floating ribs behind, the other below the costal margin on the outer side of the rectus abdominis border. By steady firm pressure

the two hands should be approximated, during a forced full inspiration and allowed to separate slightly, when the movable kidney may be felt to pass between them and may be directly engaged and palpated during the maneuver.

Caution.

No attempt should be made to grasp the kidney primarily, and in any event it should be engaged between the fingers of the two hands by simple approximation rather than by any grasping or clutching movement such as is sometimes recommended.

Useful postures.

Patient's Attitude.—Though ordinarily such an examination can be carried out when the patient is in a dorsal position, it is often useful to examine them in a position between the dorsal and the lateral, the arm on the side under observation being allowed to hang loosely forward and the patient receiving some support to relieve the abdominal tension. So also one may often find that the organ is most readily reached when the patient is standing and bent forward or even when in the knee-chest position.

Movable vs. floating kidney.

Degrees of Renal Displacement.—The palpable kidney is one whose lower edge can just be felt by the examiner. Such a kidney is not abnormal; the *movable kidney* is that which slips back and forth like a "pea in a pod," or one which can be fixed by passing the examining fingers above its superior border during full inspiration.

The term floating kidney is applied to those having more than a vertical displacement, or such as are vertically displaceable to a lower level than the umbilicus. The range of mobility is often extraordinary and in some instances the kidney may be found in the pelvis.

Manipulation-albuminuria.

Important.—Not infrequently albumin may be found in the urine after the kidney has undergone such manipulations and may lead to a faulty conclusion. It is trivial, transient and unimportant.

Usually congenital.

Etiology.—*The disease is ordinarily congenital and but a part of what Stiller has justly called "asthenia universalis congenita."*

Important associated conditions.

Almost without exception a floating kidney is associated with gastroptosis, splanchnoptosis, a narrow, mobile, readily dilatable, low-lying, modified or typical, "drop heart," a more or less "phthisical" bony conformation of the chest and a more or less decided tendency to nutritional instability which may or may not be present at the time of examination.

Functional and nutritional instability.

There is in such cases a congenital delicacy of structure and unstable functional and nutritional equilibrium of the utmost importance to both diagnostician and therapist.

It is far more common in women than in men and most marked in multiparæ and in poorly nourished individuals. About three-fourths of the cases occur on the right side and in about one-seventh the condition is double.

Increase of mobility.

Congenital relaxation of the ligaments is primary, tight lacing and repeated pregnancies, the wasting of the peritoneal fat of the capsule, traumatism or muscular strain increase the mobility and *failure or marked depression of nutrition invites symptoms.*

In most cases a combination of these causes may be operative. A curious relationship, possibly associated with the enteroptosis, sometimes seems to exist between floating kidney and the occurrence of appendicitis.

Production of symptoms.

Symptoms.—In well-nourished phlegmatic individuals extreme ptosis may exist without symptoms, while slight cases may contribute to the general discomfort in the undernourished individual.

Symptomless cases.

In view of the exaggerated importance attached to this condition by the laity it is often wise to withhold information from the patient where no symptoms seem to be present or where the condition is incidentally or casually encountered.

When symptoms are present they vary from those of "nervousness," dyspepsia, troublesome psychasthenia, or hysteria, chiefly attributable to the general state of the patient, to the *remarkable pain crises, first described by Dietl.*

Dietl's Crises.—*These are attacks, sudden in onset, characterized by severe abdominal pain, nausea, vomiting, and in extreme cases, by chill, fever, and even symptoms of collapse.*

The utmost care should be observed in diagnosis, as errors are astonishingly frequent.

The author ventures again to express the opinion, founded upon somewhat extensive observations, that true Dietl's crises are extremely rare; and that the appendix, gall-bladder, gastric ulcer, renal calculus, and especially gastroptosis with pyloric spasm are vastly more likely to prove the essential factors.

PYELITIS AND PYELONEPHRITIS

Definition.—By pyelitis is meant an inflammation completely or chiefly confined to the pelvis of the kidney; by pyelonephritis, an inflammation involving both the kidney substance and the pelvis. The former can hardly exist without a slight invasion of the kidney texture.

Etiology.—Various microörganisms are capable of causing these conditions. Among these are: the pyogenic streptococci, staphylococci, typhoid and colon bacilli, gonococci and tubercle bacilli.

Ascending vs. blood-borne infections.

Infection in some cases is ascending, the bladder or ureter being the primary source. They arise also in connection with certain virulent acute infections or in those of the chronic sort, and under these conditions the infective agent would appear to be brought directly by the circulation. Renal calculus is a potent cause, *yet stones may exist for years in a kidney without causing any marked disturbance.*

Fever of infection.

Symptoms of Pyelitis.—*An acute pyelitis is ushered in by fever, pain in the back, or tenderness in the region of the twelfth rib, and is usually marked by frequent micturition. The urine in acute, severe cases is diminished in*

Acid urine.

amount, contains pus in quantity and is usually acid in reaction in primary pyelitis.

The reaction is largely dependent upon the microörganism present. The colon bacillus, gonococcus, and tubercle bacillus are, fortunately, associated with acidity. The staphylococcus, streptococcus and proteus cause an alkaline fermentation.

Rarely a colic

The pain which is usually more or less severe at the onset even in simple suppurative pyelitis may be so extreme when, as sometimes happens, complete or even partial temporary blocking of the ureter occurs, as to simulate

renal colic and may radiate in the same way to the groin, inner side of the thigh or testicle.

Chill and fever may occur at any time during the course of an acute attack and certain cases have been mistaken carelessly for malarial fever on this account. Occasionally in unilateral cases, the ureter or an affected kidney becomes blocked, and retention on the part of the unsound kidney permits a misleading secretion of normal urine from the sound side. In such cases a pyonephrotic tumor is evident on the affected side. So also masses of stringy pus and débris may produce obstruction at the neck of the bladder and pain simulating that of stone in the bladder.

Misleading findings.

Sediment.—The urinary sediment shows large quantities of pus, a variable amount of blood, usually of the slightest quantity, and the more or less characteristic cells of the renal pelvis. Ordinarily one will find also a few renal cells and an occasional cast. Under prompt and efficient treatment these symptoms rapidly subside so that in a period varying from a few days to two or three weeks, an uncomplicated simple case may recover.

Symptoms of Pyelonephritis.—These are essentially the same as those of pyelitis, save that the amount of pus is usually larger and the sediment shows a decidedly greater number of renal elements.

The characteristic features of the typical case are: (*a*) *An acid urine,* (*b*) *containing pus,* (*c*) *yielding a sediment showing characteristic elements, i.e., casts and renal and pelvic epithelium.*

The picture may be much obscured by the presence of a complicating or primary cystitis, in which case the urine may be ammoniacal, and the sediment be so profuse and with such predominance of the cystic elements as to greatly obscure the diagnosis unless modern methods of direct examination are employed.

Cystitis vs. Pyelitis.

RENAL TUBERCULOSIS

Its Two Forms.—This occurs in two forms—the *acute miliary,* which is merely a part of a general tuberculosis, and invariably obscured by the other manifestations of the disease, and, the so-called "tuberculous infiltration" (*caseous form*), which is of great clinical interest. The latter commences usually as a miliary or larger nodule which undergoes much the same changes as would occur in other portions of the body. Either one or both kidneys may be affected and the process frequently involves the whole urinary tract, including the urogenital apparatus.

Miliary.

Caseous.

Etiology.—Predisposing influences such as trauma, chronic cystitis, pyelitis, urethritis, and the congestion of pregnancy may play a part and it is unquestionably most frequent in persons of the congenital asthenic type who afford a favorable soil for the tubercle bacillus.

Tuberculosis of the kidney is now regarded as primary rather than ascending and we know that it tends to confine itself for long periods to a single kidney and spare the ureter and bladder. It is, therefore, a blood-borne infection and from the standpoint of actual origin is secondary to some primary focus elsewhere in the body.

Primary and blood-borne.

Frank vs. latent case.

Diagnosis.—The diagnosis may be extremely easy or surprisingly difficult, the development being in some instances that of a *frank tuberculous pyelitis or pyelonephritis*. In others it pursues a latent course with no symptoms save those of irritation.

Examine for t. b. elsewhere.

In every case attention should be given to the organs so often secondarily affected, such as the spermatic cord, testis, prostate, and in the female the ovaries and tubes.

Dysuria.

So also persistent dysuria without signs of bladder disturbances sufficient to account for the condition will oftentimes prove to be of tuberculous origin. In most cases there is a slight elevation of temperature and, in a goodly number, tuberculosis present or past or a suggestive family history is made evident. Symptoms of lithiasis may co-exist and complicate the picture. The affected kidney may or may not be movable primarily and in some instances becomes greatly enlarged and readily palpable.

Not always progressive.

According to the author's experience the caseous chronic tuberculosis of the kidney acts much as does tuberculosis elsewhere and may go rapidly to the bad, recover temporarily or even become permanently arrested, under the same general conditions as apply to tuberculosis of the lungs. Spontaneous healing under rest, improvement of nutrition and favorable environment is certainly more common than is generally believed.

He cannot, therefore, coincide with the opinion expressed by some of our best surgeons that the tuberculous change is of necessity and invariably a progressive and destructive one, however great the comfort and increased sense of safety conferred by nephrectomy in unilateral cases.

In a considerable number of cases of slight involvement occurring in patients who declined operation and in a number who presented a double tuberculosis so advanced as to forbid operation, the process has shown the same or indeed a greater tendency to arrest than is observed in the lungs when proper conditions were obtainable.

Scant findings.

*Finally, the diagnosis must depend upon the findings of the tubercle bacilli in the urine and their differential staining by proper methods and the localization of lesions by means of modern procedure. It must be remembered that they may be found in urines showing but the slightest traces of pus.**

In some instances the diagnostic use of tuberculin is justifiable and may be followed by the appearance of the bacilli in a urine from which they were absent previously.

May or may not be symptomless.

RENAL INFARCT.—This common condition may produce no symptoms sufficient to attract attention though in rare instances there may be localized pain and tenderness, chills, fever and vomiting associated with albuminuria and perhaps hematuria.†

* In several cases recently observed the urine was almost clear and the only subjective symptom was a slight dragging pain over the kidney.

† In case the urine is being closely watched, the author believes that even though subjective symptoms are lacking albuminuria will usually be present and hematuria more frequently than is taught at the present time.

Such emboli may be suppurative, in which event renal abscess promptly follows, but they are usually non-infective and *the irregularly contracted kidney* of repeated infarction is a common finding in the autopsy room in connection with valvular disease and myocardial degeneration of the left chambers of the heart. A positive clinical diagnosis of renal infarction can rarely be made but their frequency in diseases involving the left heart should be held in mind.

RENAL TUMORS.—Their general characteristics have already been described and the subject is one for surgical rather than extended medical discussion.

A surgical topic.

The malignant hypernephroma is of congenital origin and springs from the suprarenal tissue. It and primary renal sarcoma are peculiarly frequent in children under ten years of age. Otherwise the tumors of the kidney are of the usual types.

Hypernephroma.

Age incidence.

Malignant disease of the kidney occurs chiefly in children under ten and in adults over fifty years of age. An apparently causeless *renal* hemorrhage should always suggest it as a possibility for it is present as an early symptom in from 80 to 90 per cent. of renal tumors and as the first sign in three-fourths of such cases.

Renal hemorrhage.

If tiny clots are present in urine which is but lightly blood-tinted and they are pale red, yellow or white, and of the size of maggots or *tripper* shreds, the probability of their origin in a malignant growth is somewhat strengthened.

Clots.

Such early hemorrhages as have been described in the preceding paragraphs may be wholly unassociated with pain.

If the growth primarily affects the renal pelvis, large hemorrhages are the rule.

Preoperative differential diagnosis of the different varieties of renal tumors is usually quite impossible.

RENAL SYPHILIS.—Nephritis associated with the secondary stage of syphilis is bilateral and differs in no respect from other forms of Bright's disease save in its response to prompt antiluetic treatment.

Secondary bilateral.

The rare cases of renal gumma are more likely to be unilateral and may exactly simulate a malignant new growth.

Tertiary unilateral.

In such cases even though the process seems far advanced, vigorous medication will often result in prompt amelioration.

RENAL CYSTS.—Classification.—We may divide renal cysts into four forms, viz.: (1) *Simple solitary cysts.* (2) *Paranephric cysts.* (3) *Echinococcus cysts.* (4) *Cystic degeneration.*

Simple Solitary Cysts.—These are simple hemispherical retention cysts, occurring chiefly in the aged, sometimes reaching a large size, and are caused probably by the blocking of certain renal canals, the formation of multiple small cysts and their ultimate fusion to form a single cavity.

Differential diagnosis as between such a cyst and a hydronephrosis is often impossible before exploration.

The use of the ureteral catheter is, of course, helpful and often decisive

as to hydronephrosis. Aspiration yields indefinite results, should be always extraperitoneal and is no safer than an exploratory incision.

The fluid from all renal cysts is practically the same, inasmuch as the urinary characteristics pertain to all.

Paranephric Cysts.—These are clinical curiosities and their diagnosis from a differential standpoint is likely to be made only by the pathologist.

Echinococcus Cysts.—These are relatively rare even in established echinococcus infection.

Usually escape diagnosis.

Small cysts must escape diagnosis unless they discharge their contents into the renal pelvis and large cysts seldom yield the pathognomonic hydatid thrill.

If extraperitoneal aspiration or formal incision reveals the hydatid hooks or peculiar membrane, the diagnosis is positive.

The content of the cyst is rich in chlorides, poor in albumin, alkaline in reaction, clear as water and of medium weight (specific gravity, 1007 to 1016).

CYSTIC DEGENERATION OF THE KIDNEY.—This interesting condition is a polycystic degeneration honeycombing the parenchyma of the kidneys which are present as bilateral tumors and are found to consist of a multitude of small cysts varying from the size of a pea to that of a cherry.

Diagnostic Signs.—If certain clinical facts are kept in mind the diagnosis of some of the cases of this interesting and relatively rare condition may be rendered reasonably definite.

Characteristic syndrome.

If one encounters bilateral tumors in the flanks associated with high arterial pressure together with uremic manifestations and cardiovascular signs of chronic nephritis, the diagnosis of congenital polycystic kidney is practically the only one fitting the symptoms.

Variable urinary findings.

The urinary signs are not dependable but in many instances the clinical picture is that of an interstitial nephritis *complete*, with urinary casts and albumin, *plus bilateral renal tumor*. Hemorrhage is not uncommon and may be profuse. Leucin-like colloid bodies (Beckmann's rosettes) may appear in the sediment. These show from two to five concentric rings and radiate striæ.

The tumors representing the degenerated kidneys need not be equal in size and unilateral cases are reported.

Summary.—In the presence of such cases as show (*a*) *bilateral tumors of renal origin*, (*b*) *high arterial pressure*, (*c*) *accentuation of the aortic second and mitral first sounds*, (*d*) *hypertrophy of the left ventricle*, (*e*) *uremic manifestations*, (*f*) *polyuria*, (*g*) *albuminuria and* (*h*) *the presence of hyaline casts, the diagnosis of polycystic congenital kidney may be made with some certainty.*

Even in the absence of distinct urinary findings, the cardiovascular signs and the *bilateral tumors* with or without distinct uremic manifestations make such a diagnosis wholly justifiable, the high arterial tension being especially suggestive.

May produce cyst-like tumor.

HYDRONEPHROSIS.—Definition.—By hydronephrosis is meant an overaccumulation of urine within the kidney due to an obstruction in some portion

of its ureter, or, to a less degree, to persistent bladder retention. A persistence of this condition may result in a conversion of a kidney into a large cyst. If persistent the condition is characterized by the large cyst-like tumor which occupies the renal site and may fill and empty in intermittent obstruction.

Persistent or intermittent.

Etiology.—The condition may be temporary or permanent, persistent or intermittent, and it may be either congenital or due to disease. Among the causes are stricture of the ureter or urethra, calculi, clots, a twisted or compressed ureter, and in short any blocking of the urinary channel. The condition is commonly temporary and intermittent, but due in most instances to temporary torsion, pressure or removable obstruction.

ACUTE CYSTITIS.—Etiology.—An acute inflammation of the mucous membrane of the bladder may be due to direct or hematogenous infection, to injury or irritation from calculi, foreign bodies, wounds and the introduction of sounds, or may be associated with urethritis, tuberculosis, prostatitis or mere exposure to cold and wet, sexual excess or the toxemia of infectious diseases.

Symptoms.—The onset may be sharp with a decided febrile reaction. Dysuria with increased frequency and marked cutting, burning tenesmus are the chief symptoms, though pain may be severe and radiate to the perineum, the glans, hypogastrium or thighs; fever is usually slight and may be absent. *The urine* is scant, strongly acid or variable, usually of high specific gravity and contains an approximately normal amount of solids, albumin, blood and pus in variable quantity. The sediment consists of pus and blood cells in quantity with much bladder epithelium and numerous round cells.

Dysuria and tenesmus.

CHRONIC CYSTITIS.—Etiology.—This may result from an acute attack or may insidiously develop as the result of an enlarged prostate, a stricture, the frequent use of the catheter, infection from the genitals (female), or from calculi, growths or tuberculosis affecting the viscus. Like every other hollow, muscular organ, the changes may be either atonic or hypertrophic and in many cases of long standing the bladder is greatly contracted and tends to become incrusted with urinary salts.

Contracted bladder.

Symptoms.—These are those of the acute form, usually in a milder degree both as to subjective symptoms and urinary findings, though in the latter blood is less often present in quantity, and the reaction is more likely to be ammoniacal. The solids are little affected and the sediment will show triple phosphate and often ammonium urate crystals if the urine is ammoniacal.

Comment.—*It should be noted that these cases, if primary, lack all evidence of renal involvement and that the albuminuria present is usually in direct proportion to the blood or pus from which it is derived.*

Source of albumin.

TUBERCULOSIS OF THE BLADDER.—A chronic cystitis presenting no symptoms of stone, stricture, or enlarged prostate, should always suggest tuberculosis. It usually involves primarily the trigone and the urethral orifices. The seminal vesicles and prostate should always be examined for nodular infiltrations and weight should be given to recent hemorrhages.

Acid chronic cystitis.

Symptoms.—The appearance is that of a severe or mild chronic cystitis, with or without hemorrhage, but associated usually with acid urine. The diagnosis depends upon the finding of the tubercle bacilli, the use of the cystoscope and in some instances the tuberculin test.

A cause of hematuria.

TUMORS OF THE BLADDER.—These fall properly under surgery, and it need only be said that papillomata are the most frequent, aside from malignant growths of advanced age, and that they produce chiefly symptoms of chronic cystitis associated with marked intermittent hematuria. Occasionally bits of the growths may appear in the sediment, together with shreds of tissue and caudate cells from the villi of the growth.

ACUTE PROSTATITIS.—This may result from the same causes that lead to an acute cystitis and is recognized by the swelling, heat and tenderness of the gland, associated with throbbing, pain in the back and legs, dysuria and constant urgency, both rectal and vesical, with marked cutting, burning pain and tenesmus and increase of pain attending the end of urination. Casts of the prostatic ducts may be present in the sediment and occasionally spermatozoa as well, otherwise the urinary picture resembles occasionally acute cystitis.

Complications.—Prostatic abscess may develop and is usually associated with known symptoms of sepsis and may cause mechanical retention of urine.

CHRONIC PROSTATITIS.—This is most frequent at advanced ages in connection with chronically enlarged prostate, but may also follow acute attacks or be associated with chronic posterior urethritis. The association of modified symptoms of the acute form with palpable hypertrophy or swelling of the prostate associated with tenderness and the symptoms of chronic cystitis make the diagnosis.

Chronic urethritis need not be considered in this volume.

URINARY CALCULUS.—This most commonly occurs in the bladder or renal pelvis, but stones may also occupy the ureters. They vary in number from one to several hundred, and in size from a mere grain to that of a large orange. They also differ greatly in form, usually assuming the shape of the cavity in which they lie and occasionally being polished by attrition when several lie in contact. Uric acid, cystin, and phosphatic stones present usually a relatively smooth surface and the calcium oxalate stone is lobulate and rough (mulberry calculus). Uric acid and urates vary in color from pale yellow to deep brown, phosphatic stones are grayish or white, those of calcium oxalate deep brown, of cystin, yellow. Section should always be made to determine the constituents, as several concentric layers may be found. As to frequency, uric acid and urate stones predominate. In all forms the urinary sediment is likely to furnish suggestive findings during the stage of stone formation, containing the characteristic crystals of uric acid, ammonium or sodium urate, calcium oxalate, or triple phosphate crystals, etc., according to the nature of the process.

Tests for Urinary Calculi.—The concentric layers should be sawed through and tests made from the scrapings of the different layers and of the powdered

nucleus. The following table is taken from Dr. J. B. Ogden's admirable book.*

CHEMIC EXAMINATION OF URINARY CALCULI

1. **Preliminary Examination.**—Heat on platinum foil:

Albumin = a flame with odor of burnt horn.

Urostealith = a flame with odor of shellac and benzoin.

Cystin = a blue flame with odor of SO_2.

Xanthin and uric acid = char without a flame.

Alkaline urates = alkaline residue soluble in H_2O.

Earthy phosphates = a residue soluble in acetic acid *without* effervescence.

Calcium oxalate and calcium carbonate = a residue soluble in acetic acid *with* effervescence.

Calcium carbonate = original powder soluble in acetic acid *with* effervescence.

Calcium oxalate = original powder insoluble in acetic acid.

Silica = residue insoluble in HCl.

Murexid Test for Uric Acid.—Original powder + HNO_3 and evaporate = pink residue + NH_4OH = purple color = uric acids and urates.

Original powder + HNO_3 and evaporate + KOH = violet color, which disappears on heating = uric acid. Violet increases on heating = xanthin.

2. **Systematic Examination.**—Presence of uric acid shown by (1). Boil in H_2O and filter.

A. Filtrate +HCl. Let stand 24 hours = crystals of uric acid. Bases in solution. Concentrate.

Calcium urate = 1 drop of solution + solution ammonium oxalate = crystals calcium oxalate.

Magnesium urate = 1 drop of solution + NH_4OH + Na_2HPO_4 = crystals ammonio-magnesium phosphate.

Sodium urate = 1 drop of solution + $Pt.Cl_4$ = after concentrating, prisms of sodioplatinic chloride.

Potassium urate and *ammonium urate* = 1 drop of solution + $Pt.Cl_4$ = duodecahedra of potassioplatinic chloride and ammonioplatinic chloride.

Potassium Urate.—Evaporate solution and ignite on mica. Residue + HCl + $Pt.Cl_4$ = potassioplatinic chloride.

Ammonium Urate.—Evaporate solution and ignite on mica. Residue = no crystals with $Pt.Cl_4$.

B. Portion insoluble in H_2O. Add HCl.

Uric acid = insoluble.

Calcium carbonate = soluble with effervescence. Filter + NH_4OH = precipitate of calcium oxalate, calcium phosphate, and ammonio-magnesium phosphate. Wash. *Calcium oxalate* = insoluble in acetic acid. Filter + ammonium oxalate to filtrate. *Calcium phosphate* gives precipitate of calcium oxalate. Filter + NH_4OH to filtrate = precipitate of ammonio-magnesium phosphate.

* "Clinical Examinations of the Urine and Urinary Diagnosis."

METHODS AND MEANS EMPLOYED IN THE DIAGNOSIS OF THE DISEASES OF THE THORACIC VISCERA

Methods of Chief Importance.—*The elicitation and interpretation of the physical signs of disease chiefly depend upon: (a) Inspection. (b) Palpation. (c) Percussion. (d) Auscultation. (e) Auscultatory percussion. (f) Fluoroscopy and X-ray photography.*

To a less degree in practice, one may employ certain other auxiliary methods depending upon special procedures or specially devised instruments of precision such as the *polygraph* and *electrocardiograph.**

Seek the normal.

Proper Mental Attitude.—*To establish the presence of normal conditions should be the primary aim of the examining student or physician.*

Incentive to accuracy and thoroughness.

This makes both for accuracy of observation and thoroughness of examination and the study of the physical signs of health, of normal breath sounds and heart tones, together with the character and extent of normal variation, is of the utmost importance to every student of physical diagnosis.

Serious mistakes.

Many damaging and even fatal errors result from the opposite attitude which leads one to seek **primarily** *for râles or heart murmurs and, in their absence, fail entirely to note less obtrusive departures from the norm though these may be matters of even greater importance.*

The greater achievement.

To detect the weakened or modified heart sounds of acute or chronic myocardial weakness, a failing pulmonary second tone in pneumonia, the significantly widened area of a murmurless but rotten heart, are far more creditable achievements than the discovery of some frank murmur.

Economy of time.

Thoroughness.—*Superficial examinations are fatal to reputation and prestige. True economy of time depends upon a correct and systematic technic, quick perception, and intense concentration.*

"Harking back."

Avoidance of Multiple Repetitions.—As one avoids asking the same question twice, so should he try to fix firmly in his memory the physical signs as they are elicited, and the well-trained student will make a complete and thorough examination and draw his conclusions, while the poorly trained one is still running in circles.

Fundamental knowledge and technic.

Essentials.—*A knowledge of topographic anatomy and of the physiology and pathology of the structure under examination, a good technic and a practised hand, good eye sight and hearing, must be combined with an accurate knowledge of the physical signs of health and disease.*

Genius dispensable.

Anyone who possesses his quota of members, normally acute special senses, and a combination of diligence and ordinary intelligence, can become a skilled diagnostician.

* The polygraph, and electrocardiograph are very valuable instruments, adding much to the exactitude of cardiac diagnosis in the hands of a specialist, but their use by the *general practitioner* is limited because of the time required for special training and the considerable expense involved in their purchase and maintenance (see "Electrocardiograph" and "Polygraph"). Mensuration is also used but is of comparatively little value and *spirometry* and *pneumometry* are of little use.

THE PREPARATION OF THE PATIENT.—Proper Light.—The chest surface should be adequately exposed and flooded with light having its source behind the physician who is thus aided in his search for physical signs, his study of physiognomy and the play of the patient's emotions. Being in the shadow he can, in some measure, avoid betraying surprise, disappointment or dismay.* Important details.

Interest, cheerfulness, encouragement and calm are the only emotions which may be reflected in the face of the physician whether in the office, wards, or sick room.

The Arrangement of the Clothing.—*This must depend somewhat upon the sex of the patient.* In dealing with women or young girls it is usually possible to conduct a satisfactory examination while some thin, soft garment loosely enfolds the patient save for that portion directly under examination. This should be so arranged as to enable one to shift it at will and expose any required area of the chest or abdomen while still preserving a comforting illusion of protective covering. *In the male the chest should always be uncovered, and so also in the female if any real necessity exists or if by greater exposure some doubt may be resolved.*† Women. Men.

A Common Source of Error.—*No proper examination can be made through heavy or starched clothing.*

Crepitations in lung apices or heart murmurs may be simulated by the crackling, rustling, or rubbing of superimposed material and their conduction obscured or lost from the same cause.

Attitude of Patient and Physician.—Whether in or out of bed, a patient should, whenever possible, be in an easy, unconstrained position, the tissues relaxed and the shoulders square. In the examination of the lung apices especially, both face and chest should be squarely to the front and the head quiet; otherwise deceptive differences in percussion may result from muscular contraction. The patient.

The physician, himself, should adopt the easiest and most unconstrained position possible for both percussion and auscultation. The physician.

Changes of Position during Examination.—*Whenever practicable and safe, patients should be examined both when recumbent and when sitting or erect, because of the peculiar postural variations of cardiac murmurs referred to further on. the modification of physical signs occasionally encountered in some of the pulmonary lesions and the effect of attitude upon pulse rate and rhythm.*

THE TOPOGRAPHIC ANATOMY OF THE CHEST

Regional Divisions.—The "sternal angle," or "angle of Louis," is a ridge marking the junction of the manubrium with the gladiolus of the sternum. *"Angulus ludovici."*

It indicates the lower border of the aortic arch, the bifurcation of the trachea, the junction of the borders of the right and left lung, and the second costo-sternal junction. Important landmark.

* A matter of some moment in relation to sudden and unexpected revelations affecting intimate friends or relatives especially, as every physician of experience can testify.

† Such is the confidence placed in professional honor and clean mindedness that the readiness to comply with such requests is usually a measure of the woman's refinement and real modesty.

A useless landmark.

The Nipple.—*The nipple indicates, ordinarily, the fourth interspace, but is subject to marked variation, both vertical and lateral, particularly in the female, and should not be used as a fixed landmark.*

The Great Pectoral.—*The lower border of the pectoralis major should correspond to the upper border of the sixth rib.*

2nd rib above, 7th below.

The Scapula.—*With the arm at the side, the upper border of the scapula corresponds to the second rib; its inferior angle to the seventh or its interspace; the root of its spine to the third rib and to the starting point of the right interlobar fissure.*

FIG. 89.

FIG. 90.

FIGS. 89 AND 8.—Illustrating the absurdity of the mammillary (nipple) line as a landmark. The discrepancies of location encountered in the male are almost as marked.

Interlobar fissure.

This fissure follows the vertebral border of the scapula when the corresponding hand is carried forward and across to the opposite shoulder and represents a region most important in connection with the primary line of invasion by the tubercle bacillus *as first detectable by physical signs.*

Regional divisions.

The modern regional divisions and orientation lines necessary to verbal or written description are shown clearly by the plates.

THE THORACIC VISCERA

With Especial Reference to the Lungs, Pleuræ, and Bronchi

"Contained" vs. "Sheltered" Viscera.—The heart with its great vessels, the lungs and their primary bronchi, are *contained* viscera. The liver, spleen, and kidneys, and even a part of the stomach are *sheltered* viscera, though actually subdiaphragmatic and abdominal.

Topography.

The Lungs.—The lungs occupy nearly all of the upper chest, their *apices* extending usually to the level of the seventh cervical spine behind and 1 to 1½ inches above the clavicle in front. The right apex is slightly higher than the left, and their *resonance* may be elicited over the whole of the supraclavicular and suprascapular regions. The *anterior borders* of the two lungs meet at the sternal angle and, in contact, pass vertically downward to the level of the fourth cartilage. At that point that of the *left lung* passes outward, leaving a portion of the heart uncovered and forming the left border of the superficial cardiac area. That of the right continues downward to the sixth cartilage.

Apex resonance.

Lung borders.

Superficial cardiac area.

Mid-clavicular line.

Lower Borders of Lungs.—*In the mid-clavicular line both lower borders are represented by the sixth rib and, from the axillary line outward, the lower borders of both lungs are practically horizontal.*

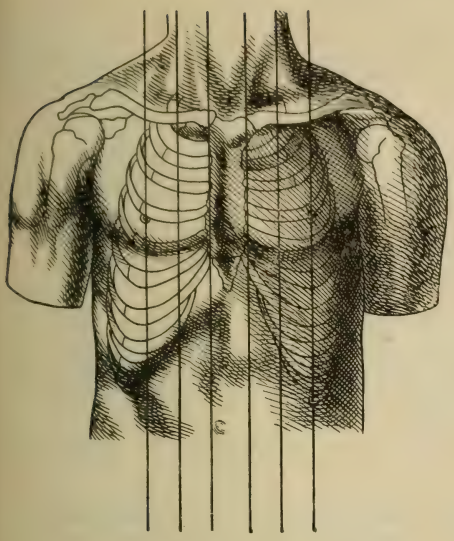


FIG. 91.—Regional divisions of the chest (anterior surface-verticals). The various divisions are plainly indicated. *a, a.* Sternal lines. *b, b.* Parasternal lines. *c, c.* Midclavicular lines.

Axillary and scapular lines.

In mid-axilla they cut the eighth ribs; in the scapular line, the ninth; and near the spine they reach the level of the tenth spinous process.

Inferior lung border.

Hence a line drawn from the sixth chondro-sternal articulation on the right side, or from the sixth rib in the parasternal line on the left side, to the spine of the tenth dorsal vertebra, indicates the inferior lung border in each instance.

Sheltered Viscera.—Below the midriff, as before stated, is an important area including the liver, spleen, stomach and kidneys.

Absolute liver dulness.

The Liver.—*The liver*, lying beneath and adapting itself to the dome-like surface of the diaphragm, rises into the thorax to a much greater degree than is shown readily by percussion, *but absolute thoracic liver dulness* begins at the lower border of the lung and extends downward to the costal margin. Its lower border crosses the epigastrium from the tip of the tenth right cartilage to the tip of the eighth left and meets the left extremity of the organ in the fifth left intercostal space at a point closely approximating the location of the normal heart apex.

Tracing the border.

Traube's Semilunar Space.—This is included between the lower border of the left lung, the spleen, the inferior costal margin, and the left lobe of the liver, and is normally hyperresonant because of the underlying stomach.

Normally tympanitic.

Loss of resonance means pleural effusion or adhesions, enlargement or tumor of the liver or spleen, or massive growths of the stomach, kidney or bowel.

Clinical value.

The Pleuræ.—*The inferior folds of the pleuræ extend practically to the costal margin and,*

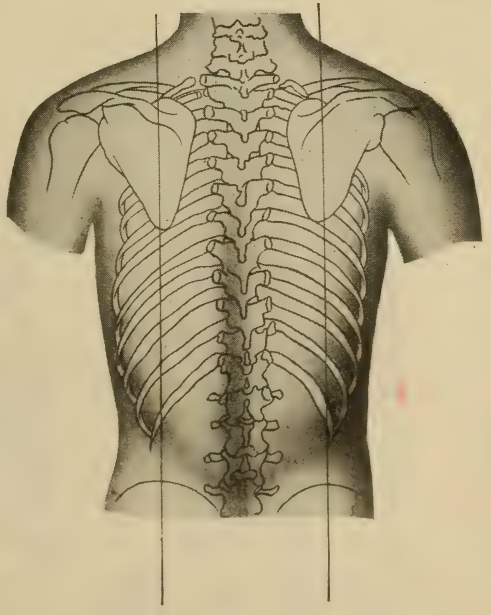


FIG. 92.—Regional divisions of the chest (posterior surface-verticals). *a, a.* Scapular lines.

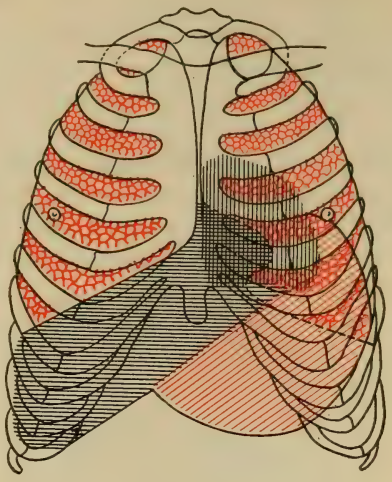


FIG. 93.—Percussion areas, normal chest (anterior surface). Lungs—red. Liver—horizontal black lines. Relative cardiac dulness—vertical black lines. Absolute cardiac dulness—cross-hatching. Stomach tympany—oblique red lines. This represents the incomplete cardiac area obtainable by flat-finger percussion in the normal heart. The more modern methods closely approximate the x-ray outline and should be used exclusively.

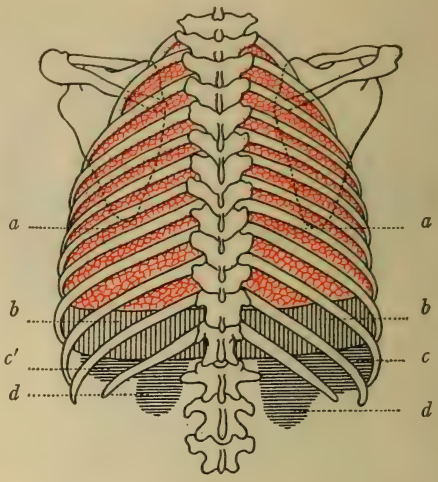


FIG. 94.—Percussion areas (normal chest, posterior surface). *a*, *a*. Lungs. *b*, *b*. Pleural space. *c*. Spleen. *c'*. Liver. *d*, *d*. Kidneys. Area *b*, *b* yields percussion dulness from spleen, kidneys, and liver, unless lungs are distended.

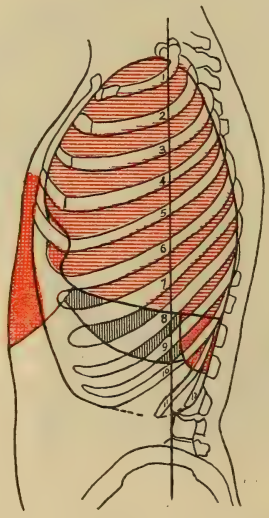


FIG. 95.—Traube's space. Bounded by the lung, spleen and liver and the costal margin. Shows region of pleural sinus in which movable dulness may appear in left-sided pleural effusion.

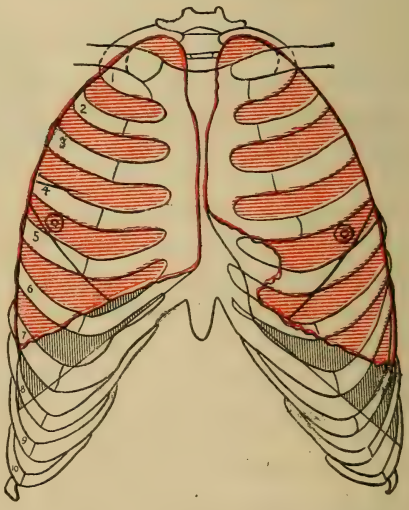


FIG. 96.—Lung boundaries (anterior surface). (*Modified Panch-Fowler.*)

Important boundaries.

Pleural sinuses.

therefore, lie much lower than the lung margin, being 2 inches inferior in the midclavicular line, reaching a maximum of 4 inches in midaxilla, and of 1½ inches in the scapular line.

It will thus be readily seen that any small effusion of fluid into the left pleural sac will, if free, produce an area of movable or shifting dulness in Traube's space, and that any increase in the size of the left lobe of the liver or of the spleen may reduce its lateral dimensions.

Furthermore, the flat note of free effusion will extend lower than any form of lung dulness.

Upper and middle lobes.

The Lobes of the Lung.—*Delineation.*—A line drawn about the chest from the second dorsal spine through the armpit to the middle of the sixth costal cartilage, indicates the division of the lung into its upper and lower lobes.

Right mid-lobe.

On the right side a second line drawn from the middle of the first line to the fourth chondro-sternal joint marks the upper boundary of the middle lobe.

Anterior vs. posterior.

Relation to Surface.—*The front of the chest, largely represents the upper—the back, the lower—lobe of the lung, the apex being accessible both anteriorly and posteriorly.*

Right median lobe.

The right middle lobe occupies a portion of the upper axilla and of the anterior surface of the chest.

EXAMINATION OF THE CHEST, WITH ESPECIAL REFERENCE TO THE LUNGS AND PLEURA

The first glance.

INSPECTION.—*A glance reveals the general contour, nutrition and muscular development of the chest, the symmetry of the two sides, equality of expansion, the presence of scars, pigmentations, skin eruptions or abnormal growths, local bulging or retraction and the absence of normal or the presence of abnormal pulsations.*

Essential Points.—Inspection is a procedure of great value and its importance is too generally underestimated. In order that it should be properly done it is absolutely necessary that the source of light should directly face and illuminate the surface under examination. Furthermore, both anterior and posterior surfaces should be inspected not only in the usual manner but also from above downward and in profile, the patient sitting, the physician standing.

In a bedfast patient these rules cannot always be fulfilled but should be observed whenever possible.

The utmost care must be observed in the case of very sick individuals to avoid exhausting them by prolonged examinations or throwing an undue strain upon a weak heart by forcing the assumption of a sitting posture.

Certain negligible factors.

General Form of the Chest.—Absolute symmetry is unusual because of slight lateral curvature of the spine, right- or left-handedness, or, the occupation of the individual, and many variations in chest outline are trivial and negligible.

Wasting and Deformity.—*A slight wasting of muscle in the region of the lung apex is often more immediately important than some gross deformity.*

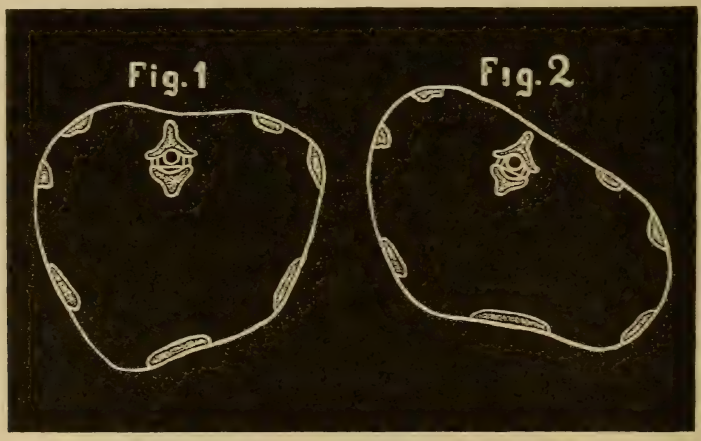


FIG. 97.—1. Unilateral retraction. 2. Spinal curvature. Outline of horizontal section. (*Gee; modified.*)

Visceral crowding and displacements.

The kyphotic, scoliotic or scoliokyphotic deformities need no extended discussion, but the student who has seen the bodies of markedly deformed persons (hunchbacks) upon the autopsy table will realize the importance, to

diagnosis and prognosis, of the extraordinary displacement and crowding of their thoracic viscera.

Unilateral Enlargement or Shrinkage.—*Unilateral enlargement* may be due to vicarious emphysema, tumors, effusions, or congenital or juvenile heart disease.

Unilateral shrinking indicates pleuritic adhesions, cirrhosis, pulmonary collapse or cancer.

Harrison's Grooves.—These are zones of retraction frequently encountered in delicate asthenic children who, by reason of congenital asthenia combined with the obstructive effect of adenoid disease or hypertrophied tonsils, have persistently imperfect lung expansion leading to partial atelectasis (*airlessness*) of the lung margins and chronic retraction of the chest along the line of the diaphragmatic attachment.

A common stigma.

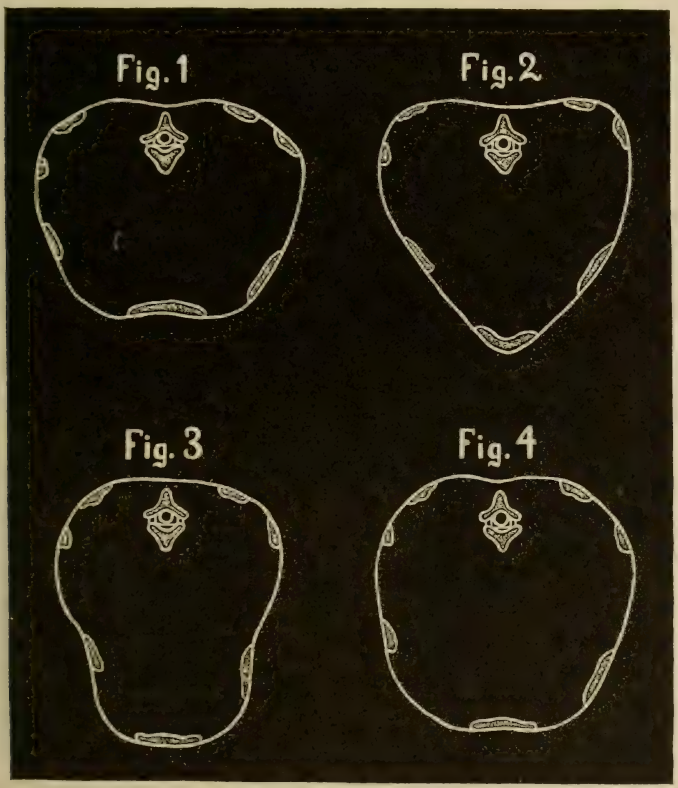


FIG. 98—1. Normal chest. 2. Pigeon breast. 3. Rickets. 4. Emphysema. (*Gee; modified.*)

General Deformities.—(*Chiefly congenital.*)—Among these are the rachitic chest, the transversely constricted chest, the flattened chest, pigeon breast and the "trichterbrust" (funnel breast).

Emphysema.—The true "barrel-shaped" chest of advanced emphysema is ordinarily manifest only when the compensatory curve of the spinal column

The barrel chest.

Obscurant spinal curve

is straightened out by placing the patient in the dorsal recumbent position on a table or firm mattress. Such a chest often appears flattened anteriorly when the patient is erect, the shoulders being rounded and slightly stooped.

Chest of forced inspiration.

The chest outline in emphysema is that of permanent forced inspiration, the epigastric angle being broad, the neck short, the sterno-mastoid muscles prominent, the ribs unusually rigid and the movement more lifting than expansile.

Overdistended inelastic lungs.

In such cases the lung borders are low; the range of their excursion lessened; the superficial cardiac area partially or more often wholly obliterated; the expiratory element of the breath sounds is prolonged and the lungs unusually bright in the X-ray picture.

The winged chest.

The "Alar" Chest.—This, called also the "*pterygoid*," "*paralytic*" or "*phthisical chest*," has a small anteroposterior diameter, long vertical measurement, broad interspaces and narrow epigastric angle. If the patient is thin the neck is long and slender and the projecting scapulæ give it its somewhat

Opposite of the barrel chest.

fanciful name. Of itself the possession of an "alar" chest or one of its modifications *does not prove the existence of a tuberculosis but is rather one of the expressions of "congenital asthenia" which offers to the tubercle bacillus the most favorable soil for its development.*

The most striking examples are seen in cases of advanced tuberculosis with extreme emaciation, yet the same skeletal *type* of chest may be observed in plump individuals.

Common forms.

Various Deformities.—*The rachitic chest* is best exemplified by the "*pigeon breast*," and the "*transversely constricted thorax*" indicates the coincidence of deficient nutrition and some chronic obstruction to breathing in childhood, usually adenoids. The line of retraction is that of the diaphragmatic attachment indicating its close relation to imperfect chest expansion.

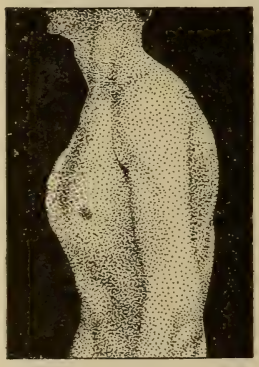


Fig. 99.—Pigeon breast (rickets).

Fig. 100.—Funnel breast (trichterbrust). In this case congenital.

Rachitic rosary

A "beading" of the ribs at the chondro-costal articulations is well known as the "rickety rosary."

"Funnel" breast.

"Trichterbrust" is of little importance in diagnosis, though an interesting and striking phenomenon. It is represented by a groove involving chiefly the median inferior portion of the chest *deepening from above downward* and corresponding to the second portion of the sternum, the ensiform often point-

ing sharply forward. Usually congenital, it may be occupational in those who in early life have performed work necessitating continuous pressure over this region. Congenital usually.

The *"thorax en bateau"* observed in certain cases of syringomyelia shows a deep anterior median groove of less limited vertical extent.

Localized Changes in Outline.—*These are of much more significance than general deformities, and marked retraction of one side or localized retraction in any area nearly always means an old pleurisy, fibroid phthisis, tuberculosis or injury.* Usually important.

Apex Retraction.—*Symmetrical retraction of the apices due to chronic nasal or tonsillar obstruction is common, is of slight importance in the absence of physical signs of disease and, even in adults, may promptly disappear after its cause is removed by operation.* Symmetrical.

Unilateral supraclavicular, infraclavicular, or suprascapular hollowing suggests existing or past disease of the lung or pleura. Unilateral.

CHEST MEASUREMENTS.—The life insurance requirements in regard to dimensions and freedom of expansion are extremely simple. They demand that the circumference of the chest at the level of the armpits shall equal one-half the height of the individual and that the difference between full inspiration and forced expiration shall not be less than 2 inches. Insurance requirements.

*As a test of lung capacity this last requirement is an absurdity, for one who under proper instruction cannot expand one-tenth his chest circumference in inches can hardly be considered normal.** An absurdity.

In measuring chest expansion, one should draw the tape *very closely* at the level of the nipple, especially in fat individuals. Correct method.

Abdominal measurement should show the maximum girth with *lightly* drawn tape and for full life insurance eligibility should never materially exceed that of the chest in full inspiration, inasmuch as statistics show that such *"bow-windowed"* persons furnish a heavy early mortality. Ascertain *maximum* girth. Bow-windowed risks.

Marked unilateral variations *in expansion, shown by measurements or visible to the eye, suggest retraction due to fibroid phthisis; pleural adhesions or spinal deformity; or the bulging caused by pleural effusions, aneurysm, congenital or youthful cardiac enlargement, or new growths.* Unilateral variations.

CHEST MOVEMENTS.—Normal breathing is of two types: (1) *costal;* (2) *abdominal.* The first predominates in corset-wearing women, the second in men. *That of women is largely superior-costal, that of men combined inferior-costal and abdominal.* Costal vs. abdominal.

In normal breathing, the ratio of respiration to pulse rate is about as 1 to 4, its rate in the new-born being 40 to 45; at the age of five, 22 to 26. It should be symmetrical, easy and quiet, and the two sides of the chest should move *equally and synchronously.* Normal respiration. ratios.

Counting Respiration.—*The respiration is best counted* by watching the rise and fall of the epigastrium coincident with the action of the diaphragm or, A useful expedient.

* Oddly enough, *athletes,* and *consumptives* not too far advanced, usually show the highest figures in chest expansion, because both have been especially instructed and the latter practice deep breathing as a therapeutic measure.

in women, the rhythmic lift of the upper chest. If, at the bedside, one ostensibly counts the pulse, keeping the eye on the proper area, neither clothing, nor bed covering prevents the use of this method nor does the patient's self-consciousness interfere.

Phantom shadow.

Litten's Diaphragm Phenomenon.—*This is a phantom shadow of inspiratory rhythm passing downward from the antero-lateral aspect of the sixth rib and vanishing just above the costal margin.*

Cause.

It corresponds to the slight drag exerted upon the intercostal spaces by the rhythmically recurring separation of the costal and pulmonary layers of the pleural fold coincident with the descent of the diaphragm and the inferior border of the lung.

Normal range.

In the normal chest its movements in forced inspiration and expiration should range from 2 to 4 inches.

Technic.

To elicit the sign the patient should be placed upon the back facing the light, the shoulders being somewhat elevated. The observer should stand with his back to the light 5 or 6 feet away, opposite the patient's knees. The illumination should come from one window only, and be not too intense.

Clinical significance.

This is readily seen in all normal chests not heavily overlaid with fat, but is lost or interrupted in pleural adhesion. It is absent in effusion, pneumonia of the lower lobe or tumors occupying the lower chest, and absent or of lessened range in incipient or advanced tuberculosis and emphysema. All lesions involving the lung or pleura which check expansion diminish the range of the shadow and it is of special value in relation to the diagnosis of adhesions.

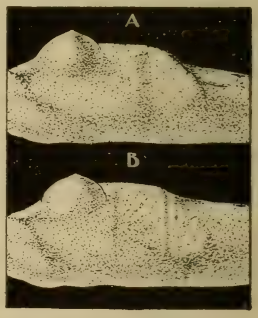


Fig. 101.—Litten's sign. (*a*) Reduced excursion in pulmonary tuberculosis. (*b*) Normal excursion.

Subdiaphragmatic abscess.

On the other hand, tumors or fluid below the diaphragm may not entirely obliterate it, and as a differential factor in the diagnosis of subphrenic abscess it has not borne out its early promise.

A valuable adjunct.

Fluoroscopic Method.—*As the descending diaphragmatic shadow accurately determines the relative range of excursion of the lower border of the lung, this constitutes one of the most definite methods of detecting deficiencies of lung excursion due to the presence of adhesions, emphysema and incipient tuberculosis, even a small area of tuberculous infiltration often checking to a marked degree the movement of the affected lung.* (See Roentgenography.)

PALPATION

Wide range.

Scope of Procedure.—*Palpation as applied to pulmonary disease has chiefly to do with the detection of fremitus, abnormal pulsation and lung expansion; but also determines the form, consistence, extent, mobility and sensitiveness of morbid growths; the nature of swellings, the presence of abnormal heat, the location of painful areas, the presence or absence of moisture and the quality, fit and elasticity of the skin.*

Friction sounds, coarse râles, the hydatid thrill or the crackling of a subcutaneous emphysema may also reveal themselves to the fingers.

Respiratory Movements.—In this connection, palpation confirms, corrects and amplifies the result of inspection. In deep breathing, expansion of the two sides should be uniform, coincident and equal. One notes:

1. *Generally increased or diminished movement.*
2. *Unilaterally retarded inspiration (inspiratory lagging).*
3. *Unilaterally retarded expiration (expiratory lagging).*

Technic.—The hands are placed fairly upon the chest, palm down, and the patient sits facing the light while the physician, standing behind him, views the chest obliquely from above. Two pencils, toothpicks, or matches, placed vertically between the fingers better show the extent of the movement. For the upper lobe it is well to place the thumbs in the supraclavicular space and the fingers in the infraclavicular region. In examining the back the patient should be turned about.

Proper light.

Justifiable and Necessary Departures from Formal Methods.—*The examination of very sick patients must be conducted with whatever of adherence to strict form and rule the circumstances permit and no more.*

In some instances the physician's examination may be of so thoughtless a nature as to actually injure the patient or jeopardize his life.

Calamitous results.

This statement is particularly applicable to the common custom of raising very sick persons to a sitting posture in an attempt to examine the lung bases, when the same knowledge might be gained by having an attendant turn the patient on the side either wholly or partially *as conditions may permit.*

In the routine examinations of known pneumonia patients sufficient access is usually obtainable with very little disturbance of one to whom sitting up in bed is a positive danger. The same may be said of victims of massive pleural effusions or the dilated heart of diphtheria.

The author has witnessed two sudden deaths directly attributable to a failure to recognize the fact that if a patient is too ill to "*sit up*" he also may be too ill to be "*set up.*"

When the lungs of any person in the lateral decubitus are examined a slight allowance must be made for the compression of the thorax on the side in contact with the bed. This tends to *slightly* dull the percussion note, increase vocal fremitus and intensify the breath sounds.

Deficient Expansion.—*General bilateral lack of expansion may be due to pain, deficient lung capacity, emphysema, a rigid chest wall, or a lack of skill on the part of the patient.*

Often trivial.

Many persons cannot breathe or cough to order and some do not know how to expand their lungs.

Unilateral defects of expansion *indicate a crippled lung or diseased pleura, be the cause what it may, the lesion ancient or recent.*

Important.

Pleurisy, pleuritic effusions, pneumonia, tuberculosis, pleuritic adhesions, a permanently retracted wall, painful or obstructive lesions of the thoracically sheltered abdominal viscera, myalgia of the intercostals, and new growths,

Important associations.

constitute the chief and most frequent of the causes of unilateral deficiency of expansion.

Significance of reversal of type.

Deficient upper-chest expansion in women, *even if bilateral, suggests apex lesions or naso-pharyngeal obstruction, for costal breathing is the normal feminine type.*

Conversely, superior costal breathing in men should lead one to examine the lung bases and abdomen, and any lack of the normal inspiratory fullness in the epigastrium, unilateral or bilateral, such as is seen in diaphragmatic paralysis and painful abdominal affections should be carefully noted.

Epigastric movement.

One cause of shallow breathing.

The breathing of one who has a painful growth or inflammation involving a movable subdiaphragmatic organ is likely to be shallow and of the feminine type.

Tender Areas.—*In the detection of tender thoracic areas, the expression of the face is the safest guide.*

Invaluable in diagnosis.

Vocal Fremitus.*—*Vocal fremitus, that vibration of the chest wall and pulmonary structures caused by the vibrations attending the production of sounds at the glottis, is best detected by simultaneously placing the palmar surfaces of both hands firmly upon the chest at corresponding opposite points.*

Counting.

The patient is then asked to enunciate slowly, clearly, evenly, and repeatedly the word "ninety-nine," the resultant vibration of the chest under the hands being noted and that of one side carefully compared with that of the other.

It is a useful procedure alternately to raise and lower the palpating hands, thus rendering the contrast in fremitus more distinct, or to apply the same hand, first to one side and then to the other.

Palpation often involves the use of one hand only, in unilateral localized lesions.

Normal predominance.

There is normally a perceptible difference in fremitus as between the right and left side, especially in the region of the upper lobes, that of the right being the stronger.

This difference is due in part to the larger size and more direct course of the right bronchus but also to the closer and more direct relationship of the right apex to the trachea.

Reversal of normal variation.

A marked difference is a suspicious sign, especially if the fremitus of the left side exceeds that of the right, and even an equality of fremitus should suggest an especially critical examination of both lungs.

Laws of Fremitus.—Vocal fremitus conforms to the laws of sound conduction. In general it follows therefore:

Voice.

1. *That, if free bronchial communication exists, the louder and deeper the voice, the greater is the fremitus.*

Density of structure.

2. *The denser the conducting material, the greater the fremitus, e.g., consolidated lung yields increased fremitus provided that it is in proper relation to a patent bronchus. Conversely the fremitus is relatively diminished over emphysematous lung or areas of pulmonary relaxation.*

Reinforcement.

3. *The transmission of the sound through a tube and into an air chamber causes*

* The palpable vibrations attending cough ("tussive fremitus") and those due to stenosis of the air passages or their obstruction by exudate ("rhonchal") or "stenotic" fremitus) are of slight importance.

conservation and reinforcement of the sound waves and thus increases fremitus, e.g., a cavity communicating with a bronchus causes increased fremitus.

4. *The more homogeneous the conducting medium, the greater is the intensity of transmitted vibration* (fremitus). — Structural homogeneity.

5. *The interposition of substances of a different molecular structure between the source of vibration and the conducting body or between the latter and the palpating hand acts as a damper and interrupts the conduction of vibrations*—e.g., absence of fremitus in extensive pleural effusion, liquid or gaseous, or diminished fremitus in pleural adhesions, save in the case of bands or cords carrying vibrations to the chest wall from consolidated or compressed lung or patent bronchi. — Sound dampers.

In the rare "massive" pneumonias the larger bronchi tributary to the area involved are filled with exudate which acts as an interposed barrier or damper to vibrations.* — Massive pneumonias.

Deductions.—*The following points are to be borne in mind:* (*a*) *Markedly increased fremitus points to consolidation of lung tissue or cavity formation.* — Increase.

(*b*) *Markedly diminished fremitus suggests emphysema, pleural adhesions, pleural new growths, pleural effusions, pulmonary edema or an obstructed bronchus.* — Decrease.

It is evident that the strength and pitch of the voice and the presence of fat and muscle modify fremitus. — Variable factors.

Any bilateral increase or decrease in corresponding chest areas is of comparatively slight significance in the absence of otherwise demonstrable bilateral lesions, such as tuberculosis, emphysema, or hydrothorax. — Bilateral variations.

Pressure Palpation.—By a mere thrusting pressure of the finger-tips, or a thrusting stroke, decided resistance areas may be quite easily defined by the specially trained finger, and the resistance felt by the pleximeter finger is used, more or less unconsciously, as an auxiliary to percussion in outlining organs, detecting exudates and large areas of infiltration. — Unconscious use of method.

PERCUSSION

The Finger as a Pleximeter.—The body may be directly struck with the tips of the fingers (*immediate*) or a pleximeter, such as the finger, an oblong piece of *hard* rubber, or a pencil, may be struck by the finger or by a percussion hammer (*mediate*). — "Immediate" and "mediate" percussion.

It matters little what the physician uses as a pleximeter provided he adheres to one method, but the finger is something which cannot readily be lost or left behind.

The percussion stroke is best made with the middle finger, and should be delivered with a loose wrist as if striking a single note on the piano. — The stroke.

Whether it be "staccato" (quickly rebounding), *a mere dropping of the hand, or, sustained, depends upon the structure percussed and the direct purpose of the individual stroke.*

* The interposition of a lead joint constitutes a common device for cutting off sound conduction in an iron pipe circuit.

Over hollow air-containing organs or pulmonary cavities or under any conditions where hyperresonance or tympany is present or suggested the *"staccato" stroke* is preferred. *A sustained stroke* best brings out resistance and if exaggerated becomes a form of palpatory percussion.

"Staccato" vs. sustained.

It is useless to get a special plexor and pleximeter, for two pencils, a coin and a pencil, etc., etc., are sufficient even for the rod pleximeter percussion.

Avoidance of technical error.

Vital Points in Technic.—(*a*) *The strokes should be of equal strength over corresponding bilateral areas.*

(*b*) *The pleximeter finger should be placed accurately, firmly and exactly but not too forcibly upon the chest and the pressure exerted should be equal over all areas under comparison.**

FIG. 102.—Percussion of apex. One of several faulty methods. The pleximeter finger is not flatly applied to the chest.

Accurate comparison

(*c*) *Exactly the same areas on each side should be alternately percussed.*

(*d*) *No change in position that involves sustained muscular action upon one side only should be permitted.*

Concentration upon tone.

(*e*) *The attention should be so concentrated upon the tone elicited as to render unnecessary a prolonged tapping of the same region, with its resultant loss of time and dulled perception.*

Direction of stroke.

(*f*) *The direction of the stroke should be perpendicular to the surface percussed, except in defining the right or left heart border, in which instance one should maintain the same vertical plane throughout, regardless of the lateral curvature of the chest* (**"orthopercussion"**). See percussion of the heart.

(*g*) *The force of the stroke should be determined by the nature of the underlying structures.*

Defining boundaries.

(*h*) *In outlining an organ, the pleximeter finger should be kept parallel to the edge of the object percussed.*

(*i*) *The more forcible the stroke, the firmer should be the pressure of the pleximeter finger.*

Respiratory stages.

(*j*) *In critically comparing pulmonary tones, the same respiratory stage should be observed.*

(*k*) *Percussion should be practised both after full expiration and deep held inspiration.* (The lung note is normally more resonant and of higher pitch in the latter stage, the lung borders low and the superficial cardiac area diminished.)

Important.

The nail of the plexor finger should be cut short so that only the pulp of the tip meeets the pleximeter finger, and as in golf, billiards or driving a nail, the eye should be fixed upon, and the stroke fall at, the exact center of the pleximeter.

The finger is by far the best pleximeter for ordinary purposes and may be perfectly adapted to every useful type of percussion.

Seldom needed.

Strong Percussion.—This may sometimes be useful when the chest wall is very fat or muscular or when one wishes to detect some non-resonant body

* This "flat-finger" percussion should never be used to determine the heart outline.

lying beneath one that is resonant, but should be avoided when possible as too greatly extending the area of vibration and confusing the results.

A stroke loud enough to be heard throughout a large classroom is always faulty and the term "strong" is relative and does not mean a pounding stroke. Don't pound.

Light or Moderate Percussion.—This is the more generally useful form and especially so when one has to deal with children, a thin wall, or with a non-resonant body overlying a resonant one. Most used.

Auscultatory Percussion.—The combination of auscultation and percussion is a valuable procedure in competent hands and especially so in determining the boundaries of thoracic or abdominal organs, the detection of A valuable subsidiary method.

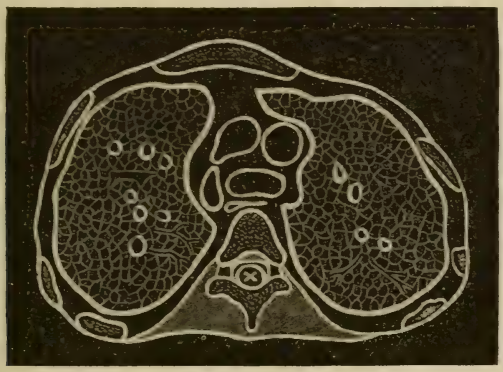


FIG. 103.—Normal chest. This figure shows the conditions affecting percussion of the normal chest. *a*. Variation in shape and volume of the two lungs. *b*. Modified resonance due to ribs and sternum overlying pulmonary tissue. *c*. The uselessness of percussion near the spinous processes of the vertebral column.

cavities, or the elicitation of the "coin sound" of pneumothorax. It may be practised by the "rod pleximeter" method with a small coin as plexor and a lead pencil as pleximeter, or, in pneumothorax, *two coins*.

The stethoscope is placed over the part to be tested or bounded and an assistant percusses lightly, gradually receding until a point is reached at which the specific note is lost or *abruptly and decidedly changed in pitch and quality*. Abrupt transition important.

*Mere gradual decrease of intensity is not definitive. It must occur abruptly.**

The heart, liver, spleen, lung apices, stomach and intestines are more or less readily and accurately differentiated, and, save the two latter structures, delineated, by this method.

Position of the Patient.—For percussion of the *anterior surface* of the lungs the hands should hang loosely at the side; for *axillary percussion* they should be placed upon the head; for percussion of the *posterior surface* they should be lightly folded in front, *not* placed upon opposite shoulders, inasmuch as the latter position necessarily involves muscular tension that interferes with the elicitation of the true note. In auscultation, however, this position is useful in determining the condition of the interlobar region. Selective postures.

* Much the same results may be obtained by stroking the surface, or by using a vibrating tuning fork.

Descriptive terms.

PERCUSSION SOUNDS.—*It is customary to describe the percussion notes as "resonant," "hyperresonant," "tympanitic," "dull" or "flat." Nothing but practice will serve to differentiate these sounds but any one of them may be elicited and studied through percussion of some portion of the normal body.*

Practice on the normal body.

Normal Areas Available for Comparative Study.—*The typical normal pulmonary resonance is that of the upper axilla, and its pitch, intensity and duration should be carefully noted and compared with that normally elicited over the apices, interscapular spaces and lung bases.*

Pulmonary resonance.

Modified dulness.

Dulness.—*A dull note is yielded by the liver below the lung margin and modified dulness just above this point, where only the thin wedge-shaped lung border intervenes.*

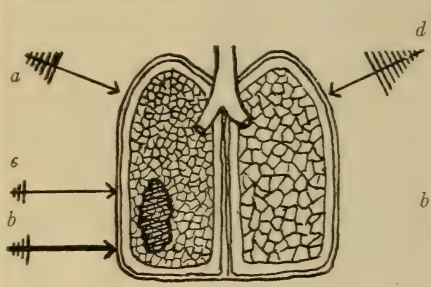


FIG. 104.—*a*. Normal. *d*. Hyperresonance (emphysema). *b*. Heavy and *c*, light percussion over consolidation.

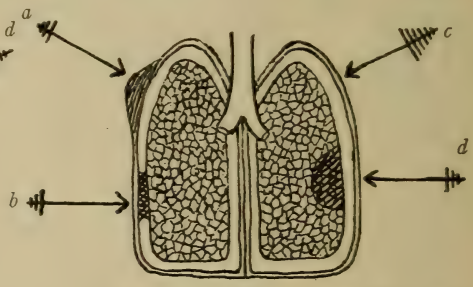
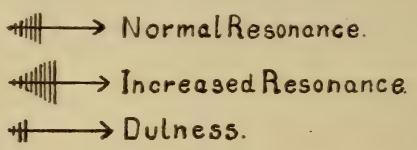


FIG. 105.—*a*. Dulness from thick wall. *b*. Pleural adhesion. *c*. Normal. *d*. New growth.



Both are extremely valuable for training the ear and acquiring an appreciation of "resistance" to the plexímeter finger.

Flatness.—*The flat note may be elicited by percussing the thigh or the deltoid muscle.*

Tympany.—*The tympanitic note is yielded by the stomach or intestines as in Traube's semilunar space, and an extremely important modification, viz.,* **dull tympany** *is heard over the main bronchi close to the sternum or over the upper sternum itself.*

Characteristics of the Percussion Sounds.—All percussion notes possess certain well-recognized characteristics and ordinary percussion also determines resistance as felt by the plexímeter finger.

Cardinal Factors.—*Each sound has a certain quality, intensity, pitch and duration.*

Intensity *depends upon the energy and amplitude of vibrations.*

Pitch *rises with increase of the rapidity of the vibrations, varying with the tension.*

Quality *depends upon the material.*

Duration *varies with the strength and amplitude of vibration and the density and tension of the structure.*

Increased resistance *goes hand in hand with dulness, and guided by this alone a stone-deaf man might make a very fair percussor.*

In general, the more air the organ contains and the greater its deep diameter the more marked is its resonance. Air content.

THE NORMAL PERCUSSION SOUNDS.—*It is important that the normal variations peculiar to different areas of the chest should be held clearly in mind, and these standard notes can be learned only by painstaking practice upon a sound chest.* Study the normal.

The Apices.—*The apices* yield normally a resonant note, clear but not intense and tending to rise in pitch and shorten in duration (dulness or "impaired resonance") as the pleximeter finger approaches the vertebral line posteriorly, or the trachea, anteriorly.

The Infraclavicular Region.—*The mid-infraclavicular space* is typically resonant, the pitch of the percussion note being slightly higher upon the right than upon the left side. Typical resonance.

Primary Bronchi.—As stated, any tendency to approach the region of the trachea or a *primary bronchus* results in a note of heightened pitch, increased resistance, and shortened duration (impaired resonance or modified dulness). Modifiers of tone.

The Hepatic Area.—*Below the right second rib anteriorly* there is increased resonance until the fifth rib is reached, when the pitch rises because of the underlying solid tissue of the liver. Modified hepatic dulness.

At the sixth rib resonance normally ceases and a line of absolute dulness marks the lower limit of the lung and the upper border of the uncovered surface of the liver. Absolute hepatic dulness.

The Axillary Region.—In the *axillary region* typical pulmonary resonance persists until the seventh interspace or eighth rib is reached (lung border).

The cardiac area markedly modifies the percussion note of the aspect of the left chest anteriorly from the lower border of the third rib downward within the mid-clavicular line. Anteriorly along the whole internal boundary of the lung the note rises as one approaches the sternum. Heart dulness.

Clavicular Percussion.—*The clavicle* is usually utilized as a pleximeter and directly percussed and its center yields a markedly resonant note. Internally and externally, pitch and resistance rise rapidly, but as a pleximeter the author believes it to be extremely fallible and often misleading. Unreliable.

Posterior Surface.—Here the height and mobility of the apices are best determined by carrying percussion upward during a forced and held *inspiration*, marking the *limit* of resonance, and repeating the procedure during forced and held *expiration*. Mere increase of intensity in the pulmonary tone in inspiration over that of expiration proves nothing. *It is the extension and diminution of the area of resonance that is important.* The heavy muscles covering the back make necessary the use of greater force and the note is less clear and satisfactory than in front. This is particularly true of percus- Apex percussion. Resonant area.

sion over the scapula itself and a glance at a transverse section of a chest shows the general futility of percussion near the spine, save for the determination of Grocco's triangle. Passing downward, the superficial *liver dulness marking the lower border of the right lung is* encountered at the *ninth rib in the scapular line.*

Liver dulness

Auscultatory Percussion of the Apices.—By placing the stethoscope bell over the suprascapular space and making direct percussion upward over the

Valuable but fallible.

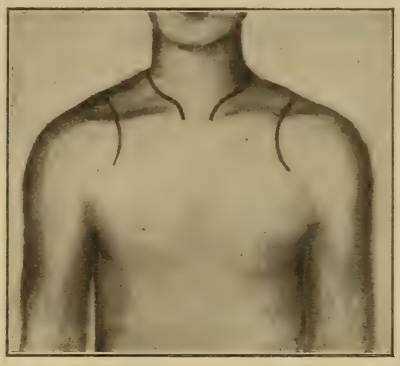


Fig. 106.—Normal apex resonance. Krönig's method.

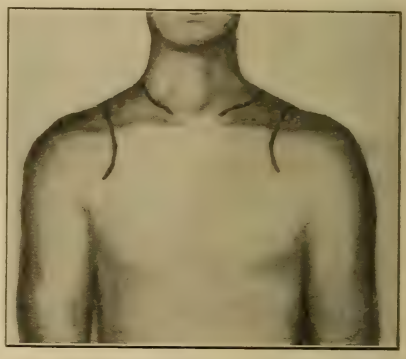


Fig. 107.—Retracted left apex. Krönig's method.

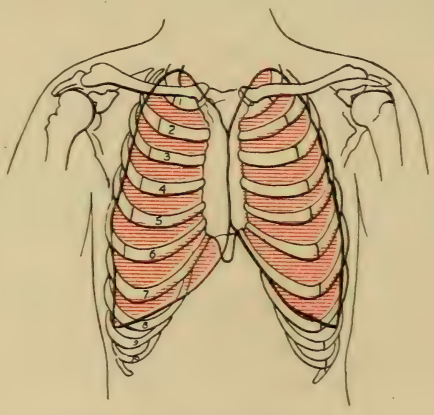


Fig. 108.—Anterior surface. Lung borders. Forced inspiration.

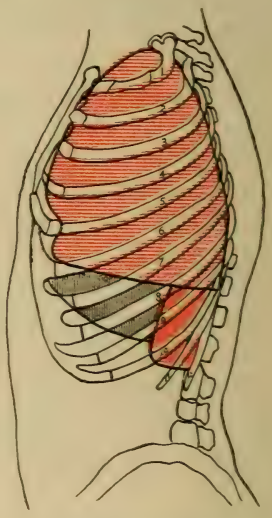


Fig. 109.—Lateral surface. Forced expiration.

apex one may mark the upper limit of resonance in full inspiration and expiration. Then without moving the stethoscope the summit along the suprascapular space should be directly percussed from the center outward to right and left to determine the lateral boundaries of transmitted resonance. The use of this method with the stethoscope well below the clavicle will determine approximately the anterior upper level of the apex and

the breadth of the resonance zone in the upper subclavicular region. This method, though fallible, is of great value in detecting obscure apex lesions.

Krönig's Method.—Krönig uses a light, finger to finger, percussion making firm pressure with the pleximeter finger and outlining the whole field of apex resonance. He lays much stress upon, what might seem to many, minor details.*

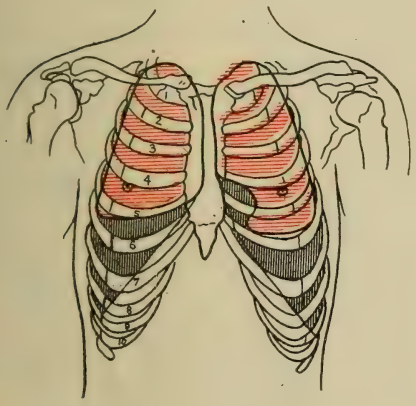


FIG. 110.—Anterior surface. Lung borders. Forced expiration.

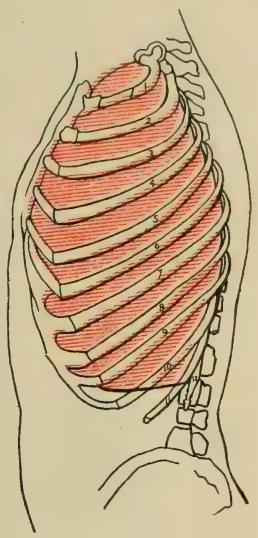


FIG. 111.—Lateral surface. Lung borders. Forced inspiration.

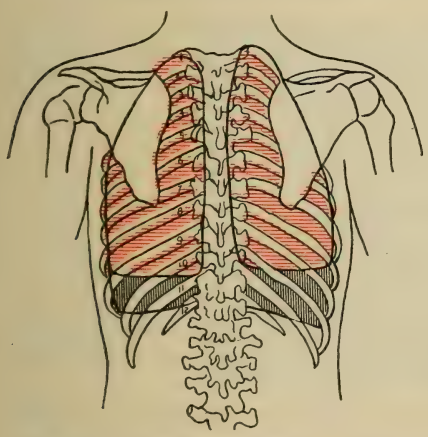


FIG. 112.—Posterior surface. Lung borders. Quiet breathing.

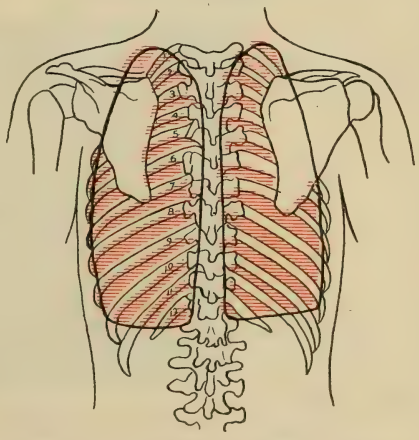


FIG. 113.—Posterior surface. Lung borders. Forced inspiration.

The Lung Borders.—*The position and mobility of the lung borders are affected in every serious chronic disease of the lung.* Important sign.

In tuberculosis, they show a decided lack of mobility, both at apex and base.

* See Deutsche Klinik., Band ii, 1907.

Important limitations.

In true emphysema, and to a less degree in vicarious emphysema, ***in pulmonary engorgement and obstructive dyspnea*** of any type, they are lower than normal at the base, and markedly lacking in range of movement.*

In fibroid phthisis, chronic pneumonia and pleural adhesions the change in position and movement is a striking symptom. In Figs. 108 to 113 the respiratory changes of the lung borders are clearly shown.

It must be borne in mind that all sound lungs cannot be held to the same limits, that equality of movement as between the two sides is the real test and that a considerable displacement accompanies mere change of posture.

Compressed lung bases.

High Inferior Lung Borders.—The lung may be crowded upward or aside by pericardial exudates, a dilated heart, malignant growths, pleural exudates, meteorism, ascites, or abdominal tumors, and thus an atalectasis may be produced in cases of long standing sufficiently complete to yield a misleading dulness at the bases, though the commoner finding is the hyperresonance of relaxed lung tissue.

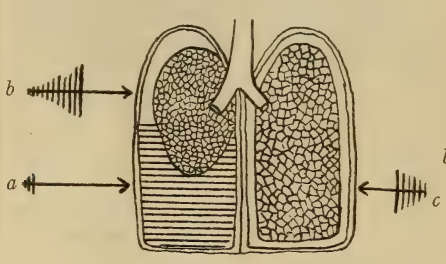


FIG. 114.—*a*. Pleural effusion. *b*. Hyperresonance above fluid exudate. *c*. Normal lung. (Some vicarious emphysema.)

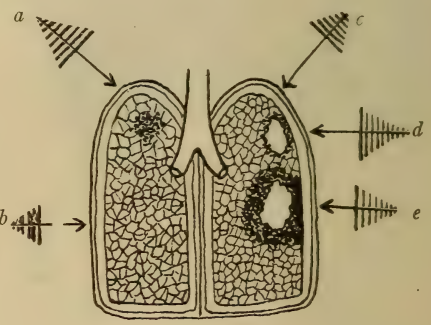
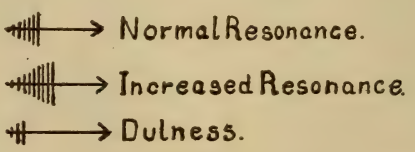


FIG. 115.—*a*. Hyperresonance over relaxed lung surrounding tubercular focus. *b*. Normal. *d*. Superficial cavity. *e*. Thick-walled cavity.



Spinal Percussion Zones.—*The spine yields a peculiar note susceptible of division into dulness, osteal resonance, modified resonance and flat tympany* (Fig. 115).

The osteal tones are so affected by the adjacent structures as to yield some information of value in certain thoracic and abdominal lesions. Thus, "A" would be lengthened in mediastinal tumor and shortened in hypertrophic emphysema. "B" would be affected by adjacent pulmonary consolidation or pleural effusion (see page 291). "C" may be affected by hepatic renal, pancreatic or gastric growths.

Of limited utility.

Most painstaking practice is necessary if one would obtain much

* In vicarious emphysema the loss is less marked.

information from these areas and it seldom offers diagnostic information which is not elsewhere and otherwise obtainable by more reliable signs.

Hyperresonance.—*This, a note of lower pitch, relatively longer duration and greater intensity than that yielded by normal pulmonary tissue, is heard over distended, relaxed, or emphysematous lung tissue.*

Low pitch and long duration.

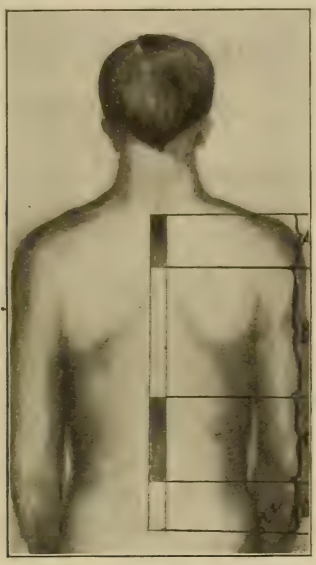


FIG. 116.—Spinal percussion zones. (*Korányi—DaCosta.*)

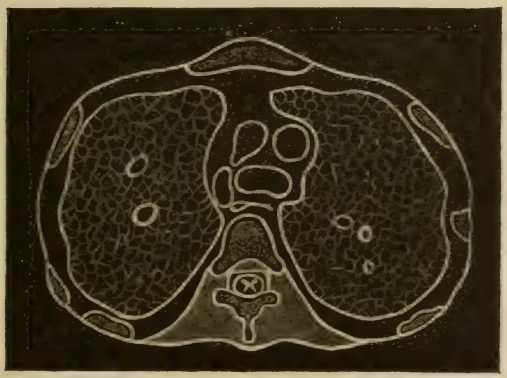


FIG. 117.—Emphysema. The distended air-cells and voluminous lung are clearly shown.

In some cases of senile (atrophic) emphysema it is replaced by a note that is distinctly high-pitched and somewhat lacking in resonance. Well-defined hyperresonance may be encountered at the lung bases in cases where extreme intra-abdominal pressure exists with upward displacement of the diaphragm and the sheltered abdominal viscera or, as previously stated, the inferior lung margins may be dull and silent under such conditions, if of long standing, because of complete marginal atalectasis.

Intra-abdominal pressure.

Drum-like note.

Tympany.—Tympany is characterized by its clear, hollow, drum-like quality which is most typically shown in percussion over large, smooth, elastic, thin-walled, empty cavities. The intensity and pitch of a pulmonary tympanitic note varies greatly with the size of the cavity in which it is produced, the thickness of its walls, its proximity to the surface, and the size of the communicating bronchus.

Cavity or hollow viscus.

Pitch of note.

The larger the cavity the lower the pitch; the greater the size of the communicating opening, the higher the pitch; and, lastly, the pitch of the percussion note varies directly with the tension of the walls.

Excessive tension.

The tympanitic quality of any note may be lost if tension reaches a certain point.

Tympanitic Note in Pneumothorax.—In open pneumothorax the percussion note is extremely drum-like over the air-filled pleural cavity, but it should not be forgotten that in certain of the cases of valvular or closed pneumothorax the percussion sound is quite distinctly dull because of extreme high tension.

Misleading dulness.

It may be metallic, but is never flat, as in the case of liquid effusion, save at the base where a small associated free exudate is usually present.

Free exudate.

Amphoric Percussion Note.—This relatively high-pitched, hollow, *metallic* note indicates a large, superficial, thin-walled, smooth, tense cavity, either wholly closed or not freely communicating with a bronchus and so formed as to produce selective reinforcement of vibrations. This tone can be closely imitated by snapping the cheek with middle finger and thumb when it is strongly distended and the mouth closed.

The "jug sound."

How imitated.

Effect of tension.

It will be noticed during such a test that certain degrees of increase of tension tend to increase the metallic quality of the tone.

SPECIAL MODIFICATIONS OF THE PERCUSSION NOTE.—The Bruit de pot fêlé.—This is precisely like the "chinking" produced when the borders of the palms of the hands are placed transversely together so as to leave a central air space free and struck sharply against the knee.

How imitated.

When normal.

It may be present normally in the thin, elastic chests of children percussed while they are crying. In the adult chest it can be obtained most readily in the infraclavicular region if there be a superficial cavity with thin walls and either a stenotic or "slit-like" opening. The mouth should be open and the heavy percussion applied during expiration.

Cavity sign.

Necessary maneuver.

This sign *may be,* but rarely is, present in open pneumothorax; in the vicinity of pneumonic areas; or above a pleural effusion.

Other associations.

Friedreich's Phenomenon.—The percussion note over a cavity may be higher during deep inspiration than in expiration.

Wintrich's Phenomenon.—*The pitch of the tympanitic percussion note over an open cavity is higher and clearer when the mouth is open.*

Sign of "open" cavity.

The changes are best appreciated when the examiner's ear is kept close to the wide-open mouth of the patient inasmuch as the amplification and reënforcement of the percussion vibrations transmitted by the bronchi and trachea are amplified by the pharyngeal and buccal walls.

Effect of posture.

If the cavity contains fluid, change of posture may block the opening of

bronchial communications by closing the cavity and abolish the sign until another position is assumed.

High-pitched tracheal tympany of the same type as regards its tonal changes sometimes occurs over infiltrated or compressed lung tissue of the ordinary type (William's tracheal tone); or over the manubrium in certain cases of mediastinal new-growth, aneurysm or massive pericardial effusion (Hoover). William's tracheal tone.

Biermer's Sign.—In hydropneumothorax the percussion note is higher pitched when the patient is in the erect or sitting posture than in recumbency because of the distribution of the fluid in the latter position which is such as to increase the long diameter of the pleural cavity and lengthen the vibration of the overlying chest wall. Postural change of pitch.

Gerhardt's Sign.—This term is applied to the well-known postural variation in the percussion note over a cavity, and is, of course, dependent upon the presence of movable fluid in a vomica freely communicating with a bronchus and possessing unequal axes. *It is a Biermer's sign in miniature.* Miniature of Biermer's sign.

Coin Sound.—This is described under "Auscultation."

Skodaic Resonance.—This hyperresonant or tympanitic note is heard over relaxed lung tissue, as, for example, in pleuritic effusion above the level of the fluid; in the neighborhood of an advancing pneumonic consolidation; in early edema of the lungs; to a lesser degree, in certain stages of incipient apical tuberculosis, and in pressure upon lung tissue by massive pericardial effusions, new growths or aneurysmal tumors. Relaxed tissue. Associated conditions.

In tuberculosis it may prove puzzling and lead the physician into a futile search for a lesion in the less resonant sound lung. A misleading sign.

Diminished Resonance.—As resonance varies directly with the amount of air in the underlying structures accessible to a percussion stroke, deep-seated areas of consolidation covered by air-containing lung tissue yield a mixture of the vesicular and dull note, such as is well illustrated by the relative dulness of the liver an inch or two above the inferior lung border. Large areas of consolidation at the surface of the lung yield a dulness exactly like that of the liver *just* below the lung border. Deep vs. superficial lesions.

Forms of Pulmonary Dulness.—It is obvious that subtle or decided differences must exist in the so-called "dull" percussion note and that these will depend upon the density and degree of airlessness of the tissue yielding it, as well as its depth from the surface, its size, its relationship to other more resonant tissues or organs and the condition of the neighboring or possibly enveloping lung tissue. Modified vs. true dulness. Variants.

Lobar Pneumonia.—If central, one may find primarily a hyperresonance which undergoes a gradual transformation to frank dulness as the process becomes superficial. So also in the stage of congestion our first percussion may elicit hyperresonance due to the disturbed tension of the tissues (relaxation) and, for the same reason, as the process of consolidation advances, a band of hyperresonance may precede it. Misleading hyperresonance.

In frank established pneumonia the note is of a dulness comparable to that of the liver just below the lung margin and it is usually definitely limited by Important points.

known lobar boundaries, most commonly being found posteriorly and, in the larger number of instances, on the right side.

Apex pneumonia.

The upper lobes are often involved secondarily or even primarily, the latter *locus* being not uncommon in influenza and in the misleading tuberculous lobar pneumonia. *If the bronchi leading to a pneumonic area are blocked (massive form) flatness results and the entire involved area becomes silent.*

Hyperresonance the rule.

Broncho-pneumonia.—Dulness does not characterize this form and if present is usually due to the fusion of lesser areas which may previously have been hyperresonant and are usually represented by strips of interscapular dulness or anterior zones along the lateral lung margins. These are usually best defined by light percussion *but in exceptional cases a frank lobar consolidation is closely simulated.*

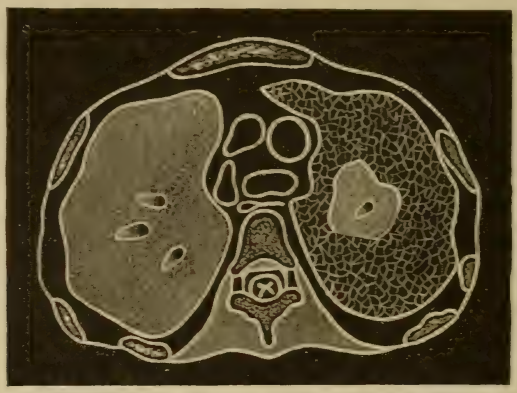


FIG. 118.—Lobar pneumonia (left); Central pneumonia (right). The lobar consolidation on the right side would present the classical signs of complete solidification with patent bronchi. The central area of consolidation might yield no percussion signs, or, hyperresonance, and be chiefly denoted by distant tubular breathing obscured by the vesicular murmur of over-lying lung-cells.

Effect of emptying.

Lung Cavities.—All pulmonary cavities may yield a dull, or if very large, a flat note when filled with secretion and one often finds a sudden transition from dulness to tympany after free and copious expectoration.

Atelectasis.—The percussion note of a beginning compression atelectasis is hyperresonant because of tissue relaxation but when carnification occurs the airless tissue yields a dull note. (See pages 383 and 384.)

Tuberculosis.—This ailment offers every variant of dulness calculated to perplex the physician. In early cases the patches are likely to be small and relaxation of surrounding tissue and vicarious emphysema add to his difficulties. Miliary cases of the pulmonary type offer as a rule only an extreme and significantly generalized hyperresonance.* Cavities may show central tympany or its modifications together with the marginal dulness of infiltration.

* The author has never seen this sign given the prominence it deserves. The hyperresonance is usually decided throughout on the lung but may be intensified over certain areas. Cyanosis is usually marked and the combination is of decided clinical value in the presence of fever.

Pulmonary Edema.—The primary percussion note of lung edema is hyperresonant but in prolonged cases the areas may become airless and the bronchi filled with liquid, in which event dulness or even flatness may be present as in the case of massive pneumonia.

Flatness.—This characteristically dead or toneless note indicates dense adhesions, liquid pleural effusions or solid growths in close contact with the chest wall. The sound is of great assistance in differentiating the puzzling cases of pleural effusion in which the breath and voice sounds have almost precisely the character of those heard in pulmonary consolidation. It is by no means a difficult matter to distinguish between dulness and flatness.

Growths and liquid exudates.

The cardiohepatic angle is represented by the resonant area in the right fifth interspace bounded by the cardiac right border and the hepatic

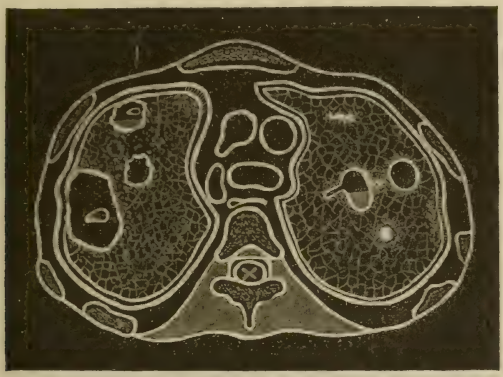


FIG. 119.—Various forms of pulmonary cavities; incipient tubercular deposits; area of softening.

dulness below. Its angular outline is lost *to percussion* early in pericardial effusion* ("Rotch's sign") right-sided pleural effusions and adhesion, and right-sided basal pneumonia.

Rotch's sign.

AUSCULTATION

Unilateral vs. Bilateral Variations.—*The first law of auscultation demands that the test of symmetric breathing shall precede inference. In other words, before concluding that a slight departure from the type of breathing on one side represents a pathologic change, the corresponding opposite area should be investigated. Slight symmetric departures from the normal are often transient and negligible.*

Fundamental rule.

Basis of Auscultatory Phenomena.—*The art of auscultation rests upon the same laws of sound as underlie palpation and percussion. The sound heard when the ear is applied to the chest is chiefly produced in the glottic chink, but transmitted and modified by the bronchial tubes, lung tissue and chest wall.*

Rationale.

The nearer the ear approaches the glottis, the greater is the predominance of a tubular element in the sounds heard and if the normal pulmonary structure

Glottic proximity

* This, despite the fact that the profile X-ray picture may show preservation of the angle with outward displacement.

Variants.

is replaced by consolidation, the glottic sounds are transmitted almost unmodified. All degrees of shading may occur according to the situation of the areas of induration and their relation to the bronchi.

Broncho-vesicular type.

If the patch of thickened lung be remote from the surface, a vesicular sound due to overlying pulmonary tissue will be superadded to, and modify the tubular sound.

Bronchus caliber.

Again, the larger the bronchus that is in direct communication with the indurated area and the more superficial the patch, the more intense will be the sound.

Cavities.—*Lung cavities in communication with a bronchus yield modifications of the same glottic sound, and follow the same general law.*

Reinforced vibrations.

In them we have a definite air chamber surrounded by more or less rigid walls of consolidated lung tissue.

FIG. 120.—Pleural effusion. Especial attention should be directed to the compressed lung of the larger effusion.

Cavernous or amphoric.

If cavities communicate with an unobstructed bronchus, are wholly or partly empty, and if the communicating tube itself be unobstructed, the glottic sounds take on a hollow, metallic or even muscial quality from the walls of the cavity and thus modified will be transmitted to the surface of the chest.

The voice.

The intensity of conducted sound varies directly as the depth and intensity of the voice and inversely as the thickness of the chest wall.

Diminished conduction.

Interposition of Air or Fluid Between Lung and Chest Wall.—*Any form of pleural effusion, liquid or gaseous, acts ordinarily as a damper to transmitted vibrations from the glottis.*

Hence, in any form of pleurisy with effusion, breath sounds are likely to be diminished or lost below the level of the fluid. Certain exceptions to this rule will be considered later (see "Pleurisy").

A huge cavity.

Open Pneumothorax.—It will be readily understood that in some cases of pneumothorax a free opening between the pleural cavity and lung may be present and that in such a case the physical signs would be those of a very large cavity.

Mediate and Immediate Auscultation.—Auscultation may be either mediate or immediate at the pleasure of the auscultator.

The Stethoscope.—As to stethoscopes, it matters little what one is used if the examiner is competent to interpret what he hears.

Cerebration the chief factor.

Essentials.—*The chief essentials in any stethoscope are:* (*a*) *That it shall clearly conduct sound in proper volume from the chest wall to the ear.*

(*b*) *That its chest piece or bell shall be of a size adequate to the purpose of the instrument, yet not too large or of such a form as to make it impossible to examine thin chests or those of children.*

(*c*) *That the ear-tips are of a form and size that will properly fit and completely close the external auditory canal without exerting undue pressure.*

Many of the modern stethoscopes have a diaphragm and are especially useful in auscultatory percussion. The diaphragm intensifies breath sounds, heart sounds and murmurs but often obscures certain faint vibratory murmurs, such as may be present in certain cases of mitral stenosis.

Advantages and disadvantages of diaphragm.

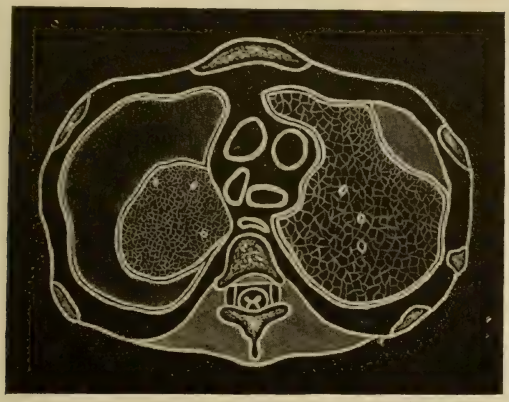


FIG. 121.—Pneumothorax (left); encysted pleurisy (right).

The author carries an instrument having a reversible chest piece, one side of which, the larger, carries a diaphragm while the other, of lesser area, is freely open, the original diaphragm having been removed. The chest piece revolves upon the branched, metal conduction tube in such a manner as to leave but one opening patent when either chest piece is employed.

He uses also, on all stethoscopes, the frame devised by the late Dr. Arthur Sansom which is perfectly adjustable, being made of malleable metal that has a trifling but adequate resiliency and can be bent to any degree desired. These arms are so jointed as to close quite compactly for the pocket. The more complicated stethoscopes have not proven useful in the author's hands, nor does he believe that it is wise to use those which excessively magnify sound.

Excessive magnification useless.

After all, there is no stethoscope that equals the unaided ear, if that has been properly trained.

High-pitched feeble sounds are often lost if the stethoscope alone be used and furthermore, one's ears are always "brought along."

Familiarity with both mediate and direct methods is an absolute necessity.

Study them.

Commoner Sources of Avoidable Error.—Every student should study carefully the sounds produced by muscle contraction, joint motion, stethoscopic rubbing, the presence of hair or remnants of the almost inevitable porous plaster on the chest and also the curious crackles which may be heard over the female breast when this is fat and is subjected to firm stethoscopic pressure.

Muscle sounds, *if due to shivering, fibrillary tremor or stethoscopic pressure, continue after breathing is stopped.*

*If due to other muscular activity or joint motion associated with respiratory effort, it will usually be evident that the patient is breathing overstrenuously.**

The presence of hair may demand the use of water or soap, and the peculiar superficiality of the breast crackles, their direct response to stethoscopic pressure in the absence of respiratory movement, and foreknowledge of their frequent occurrence over that region will prevent error.

Avoid overpressure.

Stethoscopic Pressure.—*The careless or inexpert frequently inflict much unnecessary pain by applying forcible stethoscopic pressure to tender areas, forgetting that only such force need be applied as will accurately and comfortably adapt the whole circumference of the bell to the chest wall.*

Varying pressure.

In the auscultation of heart murmurs, however, it is necessary to vary the contact, as systolic murmurs are intensified and presystolic murmurs diminished by pressure, *but painful, excessive pressure is seldom or never required.*

Quiet Necessary.—Whatever the form of stethoscope or the physician's natural or acquired power of concentration, a quiet place is essential to good work.

"Losing" extraneous sounds.

Absolute quiet is usually unattainable however and the faculty of unconsciously "losing" extraneous sounds while detecting internal abnormalities may be developed to a remarkable degree and must be acquired.

Precautionary measure.

Instruction of Patient.—It may be necessary to show the person under examination just how he should breathe, and ordinarily the mouth should be open and the respiration be thoracic, deep, uniform, regular and free both from blowing and puffing on the one hand and extreme deliberation and

The "Sandow effect."

Sandow-like "pouter pigeon protrusion" on the other. If these precautions are not observed, accidental sounds will be abundant, disturbing and misleading.

Extreme deliberation kills the pulmonary sounds, which should be distinctly heard, whereas excessive straining produces the misleading murmurs of muscular contraction.

In any event it is well to observe and listen to a few of the patient's inspirations before applying the ear or stethoscope to the chest.

Effect of Cough or Crying.—In broncho-pneumonia, or in early apical-tuberculosis, cough, or, in the child, crying, may develop well-defined, pathologic breathing or suggestive modifications in areas previously silent.

* The true source of such sounds oftentimes may be determined by having the patient go through the act of inspiration and expiration with the nostrils stopped and the lips tightly closed.

Attitude of Patient during Auscultation.—The position of the patient during auscultation should vary with the different areas under investigation, precisely as in percussion, save that the most effective examination of the region of the posterior interlobar space requires that the hand should be carried across the chest to the opposite shoulder. Posterior interlobar region.

The scapula is thus carried forward and outward, and by its posterior border very nearly defines the posterior line of division between the upper and lower lobes.

Pulmonary Areas Demanding Special Attention.—Fowler, of London, has emphasized the necessity for a special examination of certain auscultation areas. He shows that *the physical signs of* tuberculosis nearly always first appear at the apex; not the extreme tip, but a point nearer to the posterior than the anterior surface and somewhat external, and that they follow a definite "line of march," *with respect to audibility*, passing downward *and into* the upper portion of the lower lobe along the interlobular fissure. Tuberculous invasion. "Line of march."

FIG. 122.—An important area. (*After Fowler.*) Position for auscultation of interlobar region.

Regions Most Important.—With reference to tuberculosis the most important areas are:

(*a*) *Posteriorly, at a point opposite the second dorsal spine but well toward the scapula.*

(*b*) *Anteriorly, at, or just below the middle of the clavicle.*

(*c*) *The supra- and infrascapular spaces.*

(*d*) *Along the inner border of the scapula, when the hand of that side rests upon the opposite shoulder.*

(*e*) *The upper part of the axillary space.*

VESICULAR BREATHING.—*Its characteristics are a peculiar, soft, breezy, rustling quality, low pitch, moderate intensity, and no definite break or silence interval between inspiration and expiration.* Normal breathing.

The latter has no more than one-third the duration of the former, has less of the typical quality, and may be inaudible in ordinary auscultation.

The sound is closely imitated in the prolonged after-sound of the soft, whispered enunciation of "F." How imitated.

PUERILE OR HARSH RESPIRATION.—Normal.—In the chest of a normal child, or when listening over a lung that is performing vicarious duties, one hears the so-called puerile breathing, which is practically *intensified vesicular respiration.* Children.

The prolonged after-sound of a loudly whispered "F" fairly represents it.

Pathologic.—It constitutes in adults an important sign of bronchopneumonia or vicarious overactivity and may be heard over the unaffected Bronchopneumonia. Vicarious activity.

side in phthisis, lobar pneumonia, or pleurisy with effusion and over the healthy portion of a diseased lung.

Early tuberculosis.

It is, moreover, an early, important and suggestive sign of incipient tuberculosis when properly supported by other evidence and may of course be heard in certain pulmonic and extrapulmonic dyspneic states.

Of obstruction.

Prolonged Expiration.—Aside from the stridor of obstructive or spasmodic dyspnea, a respiratory sound possessing this as its most marked characteristic is frequently heard on auscultation, and suggests at once *a delayed expulsion of air from that portion of the lung yielding the sign.*

Impaired expulsive power.

Suggested ailments.

If the expiratory note be high-pitched, a small area of infiltration is indicated; if the pitch be low, one thinks of distention or relaxation of the lung tissue, such as occurs in emphysema, chronic bronchitis, or in areas immediately surrounding an incipient tuberculous deposit.

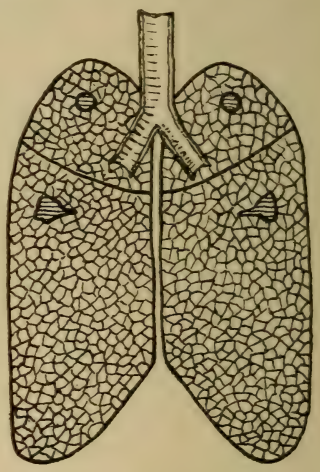


Fig. 123.—Selective points for manifestation of physical signs in pulmonary tuberculosis.

Absent or Suppressed Breathing.—*Absence of the breath sounds over any part of the lungs indicates feeble breathing, the interposition of some substance between the lung and the chest wall, an obstructed chief bronchus or certain types of atelectasis.*

Various causes.

Senility, profound exhaustion, shock or collapse represent the first; pleurisy with effusion, closed pneumothorax, superficial filled cavities, and pleural growths or thickening, the second; and the rare cases of massive pneumonia prolonged pulmonary edema, pressure of growths or aneurysm, or occlusion by foreign bodies, the third.

Incipient phthisis.

Suppressed or feeble breathing is common at the apex in incipient tuberculosis, when small multiple foci are enclosed by relaxed lung tissue. Such scattered foci are oftentimes too small to yield signs of infiltration.

Overrated.

Cog-wheel Breathing.—Much stress has been laid upon this as a symptom of early phthisis. It is overrated and one should remember that mere nervousness may produce marked overaction of the heart, irregular and

Spurious form.

unequal inspiration, uneven muscular contraction and, therefore, "*misleading pseudo-cog-wheel breathing.*"

Title faulty.

The Genuine Cog-wheel Breathing.—*If genuine, it should be heard when the breathing is regular and deep, and the heart action neither unduly violent nor greatly accelerated. The term "cog-wheel" is not descriptive. The breathing is wavy, and marked by distinct breaks and the change almost wholly pertains to inspiration.* When genuine it indicates imperfect or irregular expansion of some portion of the lung.

Impaired expansion.

Normal over 7th cervical.

BRONCHIAL BREATHING.—Normal.—If a stethoscope be placed over the seventh cervical spine, bronchial breathing is heard *normally.*

It differs from vesicular breathing in almost every particular. Both inspira-

tion and expiration are high-pitched; between them is a distinct break. Expiration is greatly prolonged, its intensity being equal to or even greater than that of inspiration and its pitch usually higher.

Infiltration, compression, or cavitation.

Pathologic.—Such breathing indicates consolidation, cavitation or compression of lung tissue in close relation to a patent bronchus.

Thus it is heard in pneumonia and established or advanced phthisical infiltration, over lung compressed by mediastinal or other tumors or by

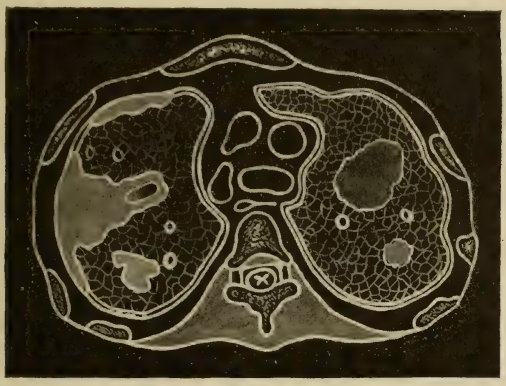


FIG. 124.—Malignant growth (left) and pulmonary abscess (right). The larger mass on the left side involves a bronchus, and would yield signs of consolidation. The anterior superficial mass would present only dulness, diminished voice and breath sounds, with defective lung movement on the affected side. Such abscesses as are here shown present few recognizable *physical* signs and are often overlooked. Malignant growths of the type shown are usually marked by hemorrhagic effusion.

massive pleural or pericardial effusion, but still maintaining communication with a patent bronchus.

Misleading variation.

Because of such compression probably, the locally intensified voice and breath sounds and especially the whispered voice are fairly well conducted from the condensed lung in some cases of effusion. In such instances the flat, toneless percussion note and usually (though paradoxically) a markedly impaired or wholly absent palpatory voice fremitus over the area of flatness suggests the correct diagnosis. The breathing is, moreover, usually *distant* though tubular in such cases. Occasionally, tense adhesion bands may act as transmitters of these sounds in cases of effusion.

Corrective value of percussion.

Broncho-vesicular Breathing.—Normal.—If the stethoscope be placed over the second intercostal space at the sternal border or is applied to the upper interscapular region of the normal chest, the sound conducted to the ear in *a curious mixture of vesicular and bronchial breathing*, the latter element being more marked in the expiratory phase.

Heard normally at 2d sterno-chondral angle.

Pathologic.—There is no more important clinical study than this modification, as it frequently represents early tuberculosis, compressed lung tissue, disseminated areas of infiltration or deep-seated central consolidation.

Deep or disseminated consolidation.

TUBULAR BREATHING.—Normal.—*Tubular breathing is that heard normally over the glottis and differs from bronchial breathing in being more intense and possessing a peculiar whiffing quality.*

Normal over glottis

Cavity or superficial consolidation.

Caution.

Pathologic.—*Its significance when heard over pulmonary areas is found in its association with cavity formation, bronchiectasis, and superficial consolidation of lung tissue.* It is important that this breathing should be carefully studied, because of its intimate association with, or near likeness to, cavernous and amphoric breathing.

Hollow and low pitched.

Cavernous Breathing.—*The sole important difference between cavernous and tubular breathing lies in the fact that in the former the pitch is relatively low and the quality distinctly hollow.*

Requisite conditions.

For its production a pulmonary or bronchiectatic cavity is necessary not less than an inch (2 cm. +) in diameter, empty or partially so, with resilient walls, and free bronchial communication.

Hollow metallic and musical.

Amphoric Breathing.—*Amphoric breathing is exactly the same as cavernous, save that it possesses a distinctly metallic or musical quality, the sound resembling that produced by blowing across the mouth of an empty bottle.*

Indicates a cavity.

It is heard over very large superficial cavities and in open pneumothorax and approximates in quality its more complex equivalent, the exquisitely bell-like tone of the coin sound as heard best in pneumothorax.

In all cases of cavity, free bronchial communication is necessary to the production of the typical sound.

Seitz' Metamorphosing Respiration.—Either or both elements in the cavernous or amphoric breathing may carry a simple tubular or bronchial element at its beginning which is transformed into the proper hollow or metallic tone before the phase is completed.

Veiled breathing.

Bruit Voilé.—Over infiltrated lung the breath sound may start as a vesicular sound and become broncho-vesicular or pure bronchial as it proceeds.

VOCAL RESONANCE

Cardinal facts.

Normal.—If the ear or stethoscope be applied to the axillary region of a healthy chest, and the patient slowly and clearly repeats the word "ninety-nine," in the lowest range of his speaking voice, the voice is heard but the words cannot be distinguished; moreover, the ear recognizes the fact that the sound is produced at a point some distance from the stethoscope.

Modified in disease.

Such normal vocal resonance is associated with a vesicular murmur and a normal percussion note, but in disease we find variations in vocal resonance to fit every variety of abnormal breath sounds and percussion tones.

Important Precaution.—*The head of the patient should be turned aside as the test words are spoken, or the misleading external sound may be further muffled by a handkerchief or towel held over the mouth.*

An important variation.

DIMINISHED VOCAL RESONANCE.—The following conditions usually result in diminution or loss of the normal vocal resonance:

Normal Causes.—(*a*) *Weak voice.* (*b*) *Excessively thick chest wall.*

Pathologic Causes.—(*a*) *Emphysema or relaxed pulmonary tension from any cause.* (*b*) *Effusion into pleural cavity (liquid or gaseous).* (*c*) *Pleural growth or thickening.* (*d*) *Massive pneumonia (large bronchi blocked).* (*e*) *Occluded bronchus due to foreign bodies or to pressure.* (*f*) *Certain phases of atelectasis and prolonged pulmonary edema.*

Increased Vocal Resonance of Heightened Pitch.—*Vocal resonance may be only slightly increased in intensity but show a rise in pitch in cases of imperfect or incomplete infiltration, or over cavities that are not surrounded by consolidated lung tissue.*

BRONCHOPHONY (*"Bronchial Voice"*).—**Normal.**—*Bronchophony is normally heard best over the manubrium. At this point the voice or whisper seems to be directly at the mouth of the stethoscope and the sense of remoteness, so marked in normal resonance, is altogether lost.* Heard normally over manubrium.

Pathologic.—*Bronchophony is heard in varying degrees under the same conditions that produce bronchial and broncho-vesicular breathing. Namely, consolidation cavitation or compression of lung tissue directly connected with a patent bronchus.*

A faint bronchophony is sometimes termed a *"bronchial whisper,"* but should be called *"distant"* or *"faint"* to avoid confusing it with *"whispered bronchophony."* Distant broncophony.

PECTORILOQUY.—(*Tracheal Voice*).—**Normal.**—*Pectoriloquy corresponds to the normal voice as heard over the trachea. It differs from bronchophony in that the words seem to be distinctly articulated and spoken directly into the ear.* Normal over trachea.

Pathologic.—*It is heard over superficial cavities, compression and consolidation.* Superficial lesions.

"Whispered pectoriloquy" *is the term applied when the whispered voice is thus transmitted and is always pathologic.** Whispered pectoriloquy.

EGOPHONY.—**Pathologic.**—*Egophony is a peculiar modification of the voice sound that gives it a bleating or distinctly nasal character. It is most frequently heard at the upper level of pleuritic effusion, and, sometimes, just above the area of advancing consolidation in pneumonia.* Nasal character.

Attention is not infrequently directed to small pleural effusions, acute or subacute, by encountering egophony at the inferior angle of the scapula. Accidental aid.

Bell Tympany (*Gairdner's Coin Test*).—*The coin sound proper is a peculiarly beautiful ringing bell-like note heard over the affected side in pneumothorax or some very large smooth-walled tense cavities when a coin is placed upon the chest and lightly struck with another.* *Bruit d' airain.*

In eliciting it in pneumothorax the auscultator usually applies his ear or stethoscope to the back and an assistant manipulates the coins upon the anterior surface of the chest. Technic.

In certain cases representing either very low or extremely high tension the coin sound may be absent in pneumothorax until the tension is changed.†

Amphoric Voice and Heart Sounds.—*Metallic or amphoric resonance* of the voice and heart sounds may be heard under conditions similar to those producing the corresponding breath sound and also at times because of the close proximity of a distended stomach. Misleading variant.

Metallic Chink (*"Signe du Sou"*).—A metallic "clicking" or "chinking"

* The whispered voice is not of glottic production and is almost inaudible over the normal lungs; wholly so as syllabic speech.

† The author has never encountered such an instance in high pressure cases, even though the dulled percussion note was present.

Pleural effusion.

may be elicitated over pleural effusions by the Gairdner coin test but is wholly different from the exquisite bell tone of pneumothorax.

Over a pneumonic consolidation the sound is usually merely a dull thud.

D'Espine's Sign.—This somewhat discredited sign is heard occasionally over the upper dorsal vertebrae in cases of tuberculosis of the tracheobronchial lymph nodes. It consists merely in a whispered prolongation of the final "e" of such words as "we" "three" or "tree" after the voice stops.

RÂLES

Their Genesis and Varieties.—*Râles are either dry or moist, and a consideration of the anatomic structure of the lungs and pleura makes it easy to understand the mode of their production, their significance, and the varying qualities dependent upon the condition of the lung itself and the character of respiration.*

Dry and moist.

Sibilant and sonorous.

DRY RÂLES.—Aside from the friction râle, dry râles may be divided into two groups: (*a*) *Sibilant or whistling.* (*b*) *Sonorous,* "at one time resembling snoring, at another the sound of a bassoon, and very frequently it is like the cooing of a turtle-dove."

Musical.

They occur in all grades of pitch and intensity and in passing from the very feeblest wheeze or whistle (sibilant) to the most raucous sonorous râle, the differences depend upon the size of the tube in which they are produced; its proximity to the surface; its relation to the consolidated areas; and the depth and character of the respiration itself.

Modifying factors.

Such dry râles alone or in association with others are most frequently found in bronchial obstruction, bronchitis, asthma and emphysema, but persistent dry râles are common in incipient tuberculosis and may be distinctly resonant or consonating in the presence of consolidation.

Significance.

Consonance refers to the peculiar quality by virtue of which the striking of a certain note upon a musical instrument will produce sympathetic vibrations in other sonorous bodies as seen in the strings of a piano. Râles of various kinds take a higher pitch and changed quality in the presence of consolidation or modify their qualities to accord with the peculiarities of vibration encountered in cavities.

Stridor.—Stridor is the loud, whistling sound heard when the trachea or a primary bronchus is obstructed by aneurysm, tumor or a foreign body, or in spasm or obstruction of the glottis by new growth, edema, false membrane, catarrhal swelling, abscess, or a foreign body. A similar sound is heard in paralysis of the abductors, such as may result from pressure upon the recurrent laryngeal nerve.*

Obstruction or glottic paralysis.

Cardinal points.

Moist Râles.—*In the consideration of moist râles the chief points to be noted are: their character, "size," pitch, and resonance.*

Causes.

A moist râle may be produced by respiration at any point from the glottis to a terminal alveolus and may be due to a tracheal "death rattle;" to the inflow and outflow of air through bronchi filled with the thin, serous exudate of edema; to the mucous or mucopurulent secretion of bronchitis; the thick tenacious sputum of pneumonia, or the purulent fluid of a pulmonary cavity or pyopneumothorax.

* See also "*Stertorous*" breathing.

Being oftentimes associated with consolidation of all kinds or cavity of any degree and originating in tubes of any caliber from that of the primary air passages to that of the terminal alveolar openings, it must vary with its surroundings in pitch, quality, size and intensity.

"Smallest" râle.

Crepitation.—*This, the "smallest," "finest," "tiniest," of all moist râles, is usually a "shower" of tiny sounds like the crackling of burning salt or the crepitations produced by rolling between the fingers a lock of the hair in front of the ear. It is heard characteristically at the end of a full inspiration.*

How imitated.

Crepitation is not truly a liquid râle, but signifies merely that air is entering into a collapsed vesicle and, at the end of inspiration, *i.e.*, at the moment of maximum pressure, is forcing apart the collapsed and slightly agglutinated infundibular and vesicular walls. The exact period for each of the group of vesicles affected varies by the fraction of a second and thus is produced the "*showers*" of crepitation.

Probable *rationale.*

"Showers."

The crepitant râle is one of the early physical signs of lobar pneumonia (crepitus indux); is not infrequently present in pulmonary edema, hemorrhage, infarction and incipient tuberculosis. It occurs as a precursor of resolution (crepitus redux) in pneumonia, and together with subcrepitant râles, or alone, may normally be heard with the first deep inspirations at the apex in persons who are habitually shallow breathers. It is present oftentimes at the base of the lung, when the first deep inspirations are taken, and, with considerable persistence, in persons suffering from exhausting disease, especially if they have been lying for some time in the dorsal position. Such râles are also heard *persistently* at the lung bases *early* in pulmonary edema and the hypostatic congestion of *cardiovascular insufficiency and constitute then a sign of importance.*

Associated lesions.

Significant.

It should be borne in mind that they are heard only at the end of deep inspiration, for at first they may be confounded by the student with a more superficial pressure crepitation produced by his own ear or stethoscope but lacking the specific timing of the true crepitant râle.

Time important.

Source of error.

Both crepitant and subcrepitant râles may be closely simulated by modified friction sounds.

Modified friction.

Friction sounds are unmodified by cough, peculiarly fixed in their apparent point of origin, and are usually distinctly superficial. Oftentimes they are manifestly intensified by firm stethoscopic pressure which may also reveal tenderness.

Differentiation.

"Crackling" Râles.—Three varieties of crackles are recognized by the diagnostician.

The Subcrepitant Râle.—*The fine crackling or subcrepitant râles are produced in the bronchioles, heard chiefly but not exclusively in inspiration and indicate, as a rule, commencing infiltration.*

Not limited to inspiration.

Bronchiolitic.

Heard at the apices, they always strongly suggest an incipient tuberculous process; at the base, temporary atelectasis, pneumonia, congestion, or incipient pulmonary edema. They are heard early in resolving lobar pneumonia and are the dominant finer râles of broncho-pneumonia.

Significance.

Medium Crackling Râles.—*These are of the same general nature as the foregoing, but coarser. They are heard over softening tuberculous areas, broncho-*

Occurrence.

pneumonia, and resolving lobar pneumonia, change of pitch and quality due to consonance being marked in the presence of infiltration.

Consonance.

Large Crackling Râles.—*These are still coarser and more distinctly fluid than the foregoing and, if consonating, point usually to extensive softening in a tuberculous infiltration area.*

Mucous Click.—This sound stands midway between the crackling and the bubbling or gurgling râles. Its name is its best description and it is usually single and oftenest heard at the apices in tuberculosis.

Bubbling Râles.—These liquid râles are true to name; may be small, medium, or large, and are usually readily recognized.

Gurgling Râles.—These, also sufficiently described in the title, are usually heard over large pulmonary or bronchiectatic cavities during cough or forced inspiration, occasionally in bronchitis with profuse exudate, and also over the drowning lungs in advancing pulmonary edema.

Pitch and Resonance of Râles.—*All varieties of râles have a certain pitch and degree of resonance, directly attributable to their surroundings.*

Resonant consonating râles.

*One that is produced in a bronchus or cavity surrounded by consolidated lung tissue or atelectasis will have many of the qualities of tubular or amphoric breathing, hence we speak of resonant râles, echoing râles, consonating râles, etc., and these may be either moist or dry and at times metallic or ringing.**

SOUNDS APART.—*There are certain special sounds of great interest, and chief among these are "post-tussive suction," "metallic tinkling," and "succussion."*

Post-tussive Suction.—This interesting physical sign is pathognomonic of cavity formation and has been aptly termed *"the india-rubber-ball* sound." It is an inspiratory moist "sucking" sound supposed to attend the expanding of a collapsed thin-walled cavity communicating with a bronchus.

Water-whistle Sound.—A coarse, gurgling sound either inspiratory or expiratory may be heard in pneumothorax if communication with the lung exists (pulmonary fistula).

Cavity or pneumothorax.

Metallic Tinkling.—*This term is applied to the exquisitely delicate and beautiful musical sounds sometimes heard over a very large wet cavity with dense smooth walls and, more commonly, in pyo- or hydropneumothorax.*

Tiny musical echoes.

The phenomenon is really due to the production of echoes, and when low-pitched is often termed amphoric tinkling.

Hippocratic Succussion.—This is the well-known "swashing" or "sloshing" sound, often metallic, usually elicited by the somewhat heroic measure

Pneumothorax.

of *shaking* the victim of a *pyopneumothorax or hydropneumothorax*, or rarely, of some huge lung cavity containing fluid.

A mere change in the patient's position or a gentle rocking of the body is usually a sufficiently effective maneuver. A similar sound may be heard

Rare source of error.

over the stomach *but is seldom so transmitted to the thorax as to cause confusion.*

A rub, squeak, creak or crackle.

FRICTION SOUNDS.—*True friction is produced by the attrition of the apposed pleural surfaces when rendered dry and harsh by inflammation; is usually unmistakable and often palpable.*

* As stated, it is well to remember that an overdistended stomach or colon may sometimes thus influence râles produced over or near the inferior boundaries of the lungs.

It is quite closely simulated as to its common phases by the sound heard when the palm of the hand is pressed firmly against the ear and the dorsum rubbed by the *flat* of the finger pulp under varying pressure. How imitated.

Distinctive Features.—*The quality of the sound is creaking, rubbing, shuffling, rustling, or, more rarely crackling. It is ordinarily distinctly superficial; commonly best heard in the axillary and inframammary regions and at the anterior lung margins; is usually jerky or interrupted in rhythm, associated with pain in deep inspiration in acute or subacute cases and unmodified by cough.* Superficial. Unmodified by cough.

If friction disappears from some site previously detected it may sometimes be elicited by flexion of the body, or raising the arm of the affected side. *Care should be taken lest a friction-like sound due to movement of the shoulder joint or scapula be confused with true friction.* Perez has described mid-sternal friction sounds associated with movements of the shoulder joints. The dependence of these pseudo-friction sounds upon movement and their ready localization will usually prevent serious error. Important point.

Friction râles may persist at the margins of the flatness due to a pleural exudate. Reappearance after disappearance indicates absorption.

Pleural Crepitation.—*This often occurs early or midway in the inspiratory phase of the individual breath sound, whereas true crepitation is late, but this distinction is not always possible.*

Pleuro-pericardial Friction.—A friction murmur of cardiac rhythm heard over the area of *superficial cardiac dulness*, or along its borders, synchronous with the heart beat but disappearing in forced expiration, *suggests* inflammation of the pulmonary pleura and apposed pericardium. Pulmonary pleura.

A similar murmur disappearing with full inspiration *suggests* inflammation and attrition of the pericardium and costal pleura. Costal pleura.

Such friction may also be persistent and yet vary in intensity with the respiratory phases. It is not a very rare sign in cases of pneumonia and indicates direct extension of the inflammatory process.

Pneumocardial Crepitations and Crackles.—These sounds *of cardiac rhythm*, with or without coincident or incidental friction sounds, are occasionally heard over the inner edges of the lung adjacent to the heart if infiltration is present. Show cardiac rhythm.

Pure crepitations along the anterior lung borders may also be associated with the fringes of air cells seen in advanced emphysema.

BRONCHOSCOPY.—The same apparatus can be used for bronchoscopy and esophagoscopy, and the instruments described elsewhere in this volume are suitable for this purpose.

The tubes carry a centimeter scale and measure for adults 7–10 mm. in diameter and 30–45 cm. in length; for children, 5–6 mm. in diameter and 15–25 mm. in length.

Thorough local anesthesia or in resistant individuals and in children general anesthesia will be found indispensable.

The position is practically that described under "Esophagoscopy" and "Gastroscopy," and the expert can render visible and obtain access to the interior of the tracheobronchial tree as far as the subdivision of the main bronchi into those of the second class.

INTRATHORACIC RADIOGRAPHY AND FLUOROSCOPY

THE ROENTGENOGRAPHIC EXAMINATION OF THE LUNGS AND PLEURA

FRANK S. BISSELL, M. D.,
Minneapolis, Minn.

A Region of Sharp Contrasts.—The thoracic cavity lends itself advantageously to roentgen investigation, the air within the lung affording the necessary contrast of the lung structure with the more dense tissue of the other viscera.

Appearance of thoracic field.

A sagittal view, on the fluoroscopic screen, is that of two large light fields, separated by a wedge-shaped shadow. The lateral areas are the lung fields and the central shadow is formed by the spinal column, sternum, mediastinal structures, heart and great primary arteries, and the venous trunks.

The Hilus Shadows.—On either side of the median shadow, within the lung fields, are the *"hilus shadows"* produced by the large blood vessels, bronchi, and the lymphatics, supplying the lungs. Usually, within the

Obsolete tuberculous foci.

hilus areas, even in persons not *actively* tuberculous, there may be several large nodules of greater density, evidences of some earlier but ineffectual tuberculous invasion.

Hilus radiations.

Radiating shadows, some short and broad, others fine and long, extend in all directions from the hilus toward the lung periphery. These, like the hilus shadows, are composite, being produced by bronchi, blood-filled vessels and lymphatics.

Fluoroscopy.

Method of Examination.—While the fluoroscopic method is excellent for the study of respiratory movements, the stereoscopic roentgenogram reveals the greater wealth of lung detail, and takes first rank in the roentgen diagnosis

Stereoscopy.

of lung diseases. In the stereoscopic roentgenogram, superimposition of the shadows of various structures lying in the axis of a given ray does not occur as it does on the single plate. Thus, with each shadow appearing in its

Defines relationships.

proper relation to every other shadow, the factor of error in interpretation is greatly reduced.

In the study of the roentgenogram, it is necessary to bear in mind that the objects appearing dark on the fluoroscopic screen are light on the photographic plate, while the lateral areas, light on the screen, appear dark in the roentgenogram.

Tuberculosis.—Mottling of the Pulmonary Field in Early Cases.—

Swollen peribronchial glands.

Normal peribronchial glands do not obstruct the rays sufficiently to cast a shadow, but when they become swollen, as the result of bacterial invasion, they appear as fine nodules, which, increasing in number, tend to give the lung field a mottled appearance.

While this mottling is observed early in many cases of tuberculosis, it is by no means characteristic of this disease but points only to some infection or irritation of the bronchi.

Tuberculosis of the adult type tends very early to involve the parenchyma

of the lung, peripheral to the terminals of the bronchial tree, and it is here that the most characteristic roentgen signs of the disease become manifest. The engorgement of the lymphatic and blood vessels which is an early reaction to the tuberculous invasion, produces in the stereoroentgenogram small cone-shaped areas of increased density, the apices of which are directed

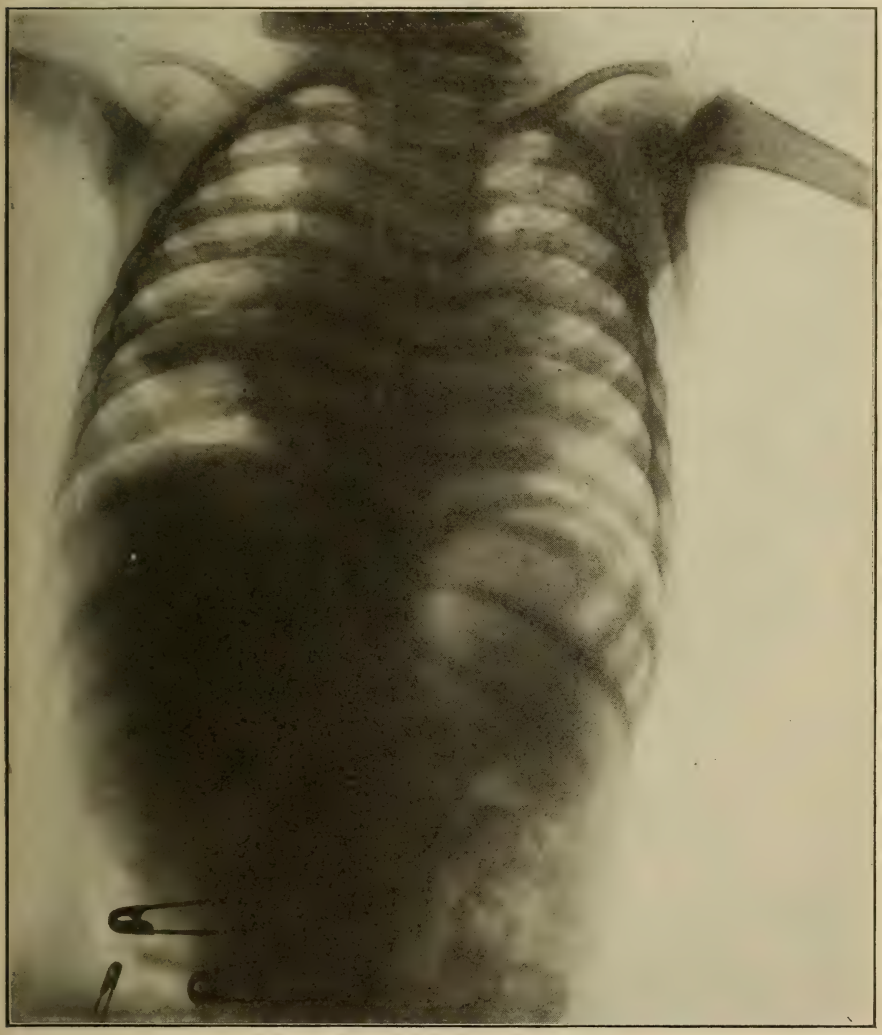


FIG. 125.—Interlobar empyema (right) with infiltration of left lung, closely simulating that of tuberculosis. (*Dr. Frank S. Bissell.*)

toward the hilus. The anatomical explanation of the "cone" is that each primary lung lobule (the anatomical unit) is triangular or cone-shaped and has its own complement of blood and lymph vessels.

Since, however, it is not always possible, even in known cases of tuberculosis to clearly delineate the "cone" it becomes necessary to rely upon less

typical areas of increased density, pointing to congestion or beginning infiltration in the periphery of the lung field. The more characteristic site of such areas is that beyond the extremities of the first and second interspace bronchial trunks. It is fortunate for differential diagnosis that these areas usually

FIG. 126.—Unilateral Pulmonary Tuberculosis. Note marked infiltration of right upper lung field. Pleuro-diaphragmatic adhesions. (*Dr. Frank S. Bissell.*)

remain clear and uninvolved until late in the progress of other chronic infections.

Basal tuberculosis, while it has the same general characteristics, is much more difficult to differentiate from other chronic infections. Tuberculosis

of the base, however, without concomitant involvement of the apex of one or more lobes, is relatively rare.

Advanced Tuberculosis.—Paradoxical as it may seem, the more advanced stages of tuberculosis sometimes present a roentgen picture less typical and hence less easy to differentiate than the earlier ones.

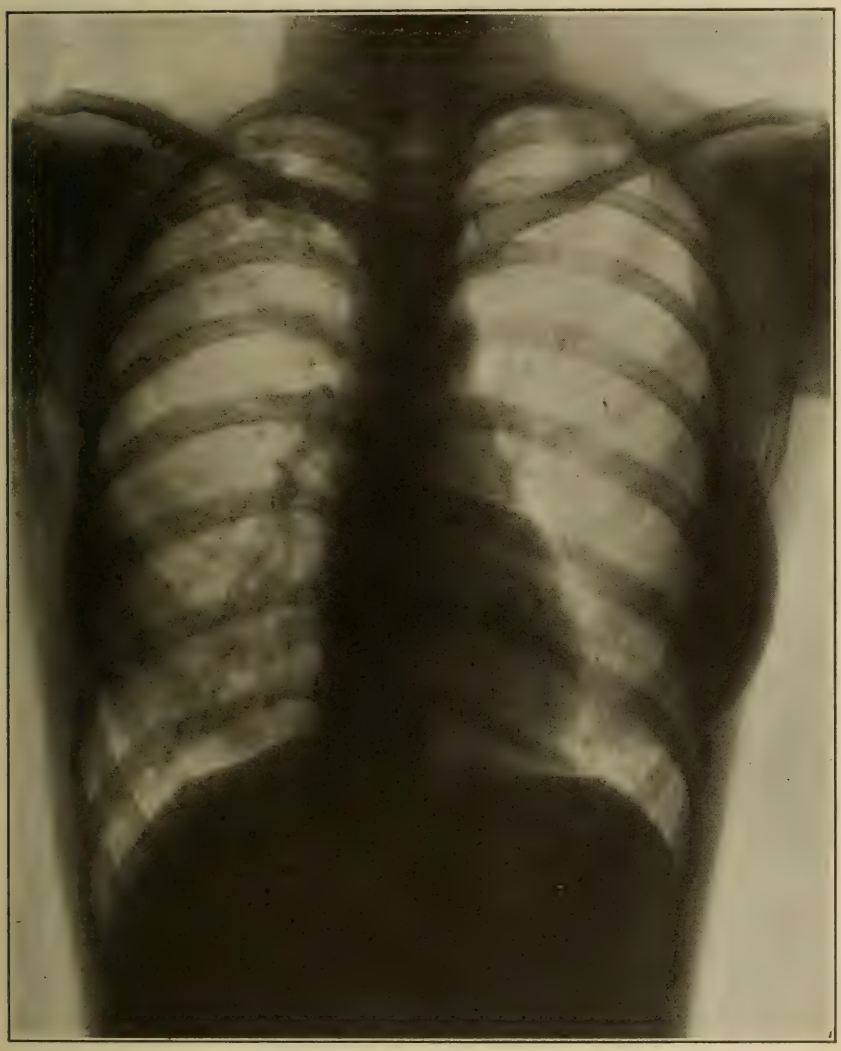


FIG. 127.—Apical Tuberculosis—Healed or Latent. Note that lesions are closely circumscribed by apparently normal lung. (*Dr. Frank S. Bissell.*)

This is true because the more characteristic changes tend to become masked by the effects of fibrosis and mixed infections. Usually, however, the distribution of the lesions points the way to a correct diagnosis or some area of slight involvement is found where the changes are more typical.

Compensatory emphysema frequently exists in some degree, manefesting **Emphysema.**

itself chiefly by increased translucency of the area so affected. This translucency, when the emphysema is extensive, aids materially in the study and recognition of the tuberculous foci by lending sharper contrast to them. *Thus the clinician's handicap becomes the advantage of the roentgenologist.*

Accuracy.

In advanced tuberculosis the greatest value of the roentgen examination

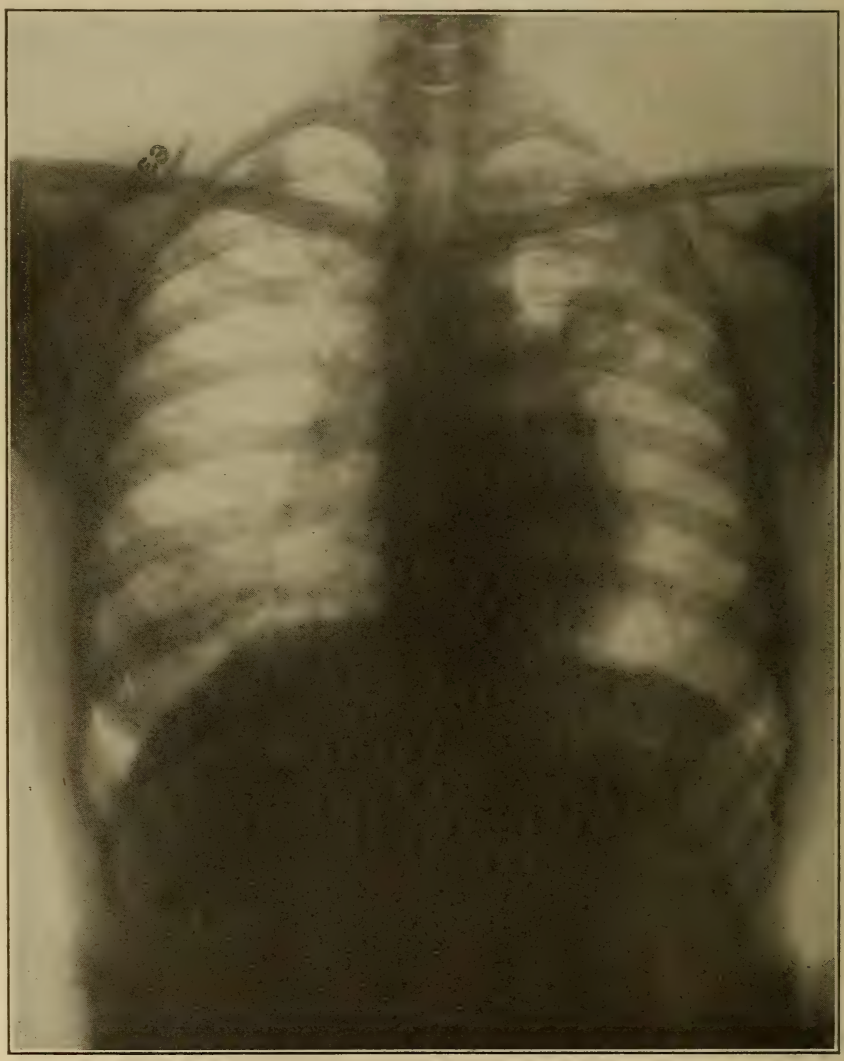


FIG. 128.—Pulmonary Tuberculosis with Cavitation. (*Dr. Frank S. Bissell.*)

lies in the accuracy with which may be demonstrated the extent of the disease, the presence and nature of complications, as well as other factors in prognosis. *The extent of involvement of a lung is usually found greater than physical signs or symptoms would lead one to suspect because so many of the lesions lie in the deeper portions of the lung where they escape the detection of the keenest clinician.*

Cavity signs.

Cavities.—Cavitation is easily recognized although one must be careful not to interpret all ring-like shadows in the lung field as cavities. There must be a total absence of normal lung structure within the ring and special care must be used to differentiate from bronchiectasis or partial pneumothorax. Areas of lung consolidation surrounding a cavity filled with exudate

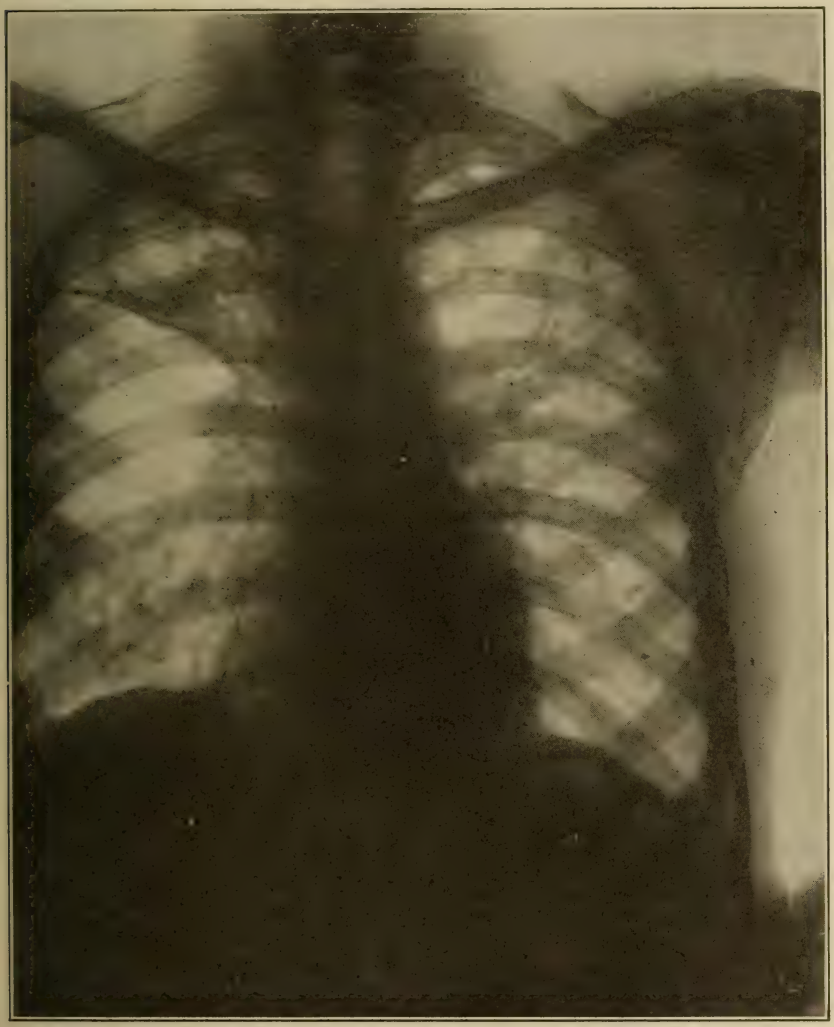


FIG. 129.—Chronic Pulmonary Tuberculosis with extensive fibrosis in both upper lung fields. Tendency is toward recovery but the determination of activity in this type of case must be made upon clinical evidence. (*Dr. Frank S. Bissell.*)

or pus may render its recognition temporarily impossible. Rarely, however, is a cavity so completely filled with fluid that it cannot be diagnosed.

Resolution.—In the process of recovery from a tuberculous invasion, the lung may return to normal or nearly normal in its roentgen appearance. An old focus which has become calcified, an apex more dense than normal

(thickened or retracted pleura) or a pleuro-diaphragmatic adhesion may alone remain to tell the tale of a previous infection.

Caution required.

There are, however, many cases clinically cured which show roentgen signs closely simulating those of active tuberculosis. Here both experience and caution are required and final conclusions as to activity should be based upon clinical, rather than roentgen evidence.

The shadows of inactive foci are more definite and circumscribed, while foci of active disease are softer in appearance and the lung immediately surrounding them is more dense than normal. This gives them a "woolly" appearance, their density shading progressively into the relative translucency of the normal air-filled alveoli.

"Woolly" foci.

Apical Tuberculosis.—*The roentgenologist meets with apical tuberculosis as described in text-books and at the bedside with comparative infrequency. A marked increase in the density of one or both apices may indicate consolidation, fibrosis, or thickened retracted pleura. When an active tuberculosis exists, however, the more characteristic signs previously noted are usually present.*

Isolated apex regions rare.

Prognostic Data.—Roentgen data which may aid in making a prognosis are: (1) *the extent of involvement;* (2) *the amount of fibrosis;* (3) *the presence of healed lesions;* (4) *cavitation;* (5) *the size of the heart.*

Aids in prognosis.

1. If the process shows a tendency to limit itself to one or two restricted areas, healing there as it spreads to others, the disease may continue active for many years without causing the lung tissue to "break down." If, on the other hand, many areas are involved in one or both lungs and these tend to become confluent, the prognosis is much more grave.

2. Extensive fibrosis, especially without cavitation, is a favorable indication.

3. So too, the presence of healed lesions, associated with the active ones, is evidence that the patient has an established immunity of some degree.

4. Cavities are always of grave prognostic importance. A case may become symptomatically cured, only to die with hemorrhage into a cavity which has escaped obliteration.

The "Drop Heart."

5. There is apparently a close association between the atrophic (drop) heart and a low degree of resistance on the part of the patient. Conversely, a large heart is a favorable factor in prognosis.

Confusing factors.

Differential Diagnosis.—*Certain chronic infections of the mediastinal and pulmonary lymphatics, secondary to tonsillar, peridental, and antrum or sinus infections, may closely simulate the roentgen picture of tuberculosis.*

The lesions, however, are prone to be more diffuse throughout both lung fields; there is not so much tendency to fibrosis and there is complete absence of any of the complications of tuberculosis such as cavitation, pleurisy, and the like. Some of these cases cannot be differentiated roentgenologically from miliary tuberculosis.

Pneumonoconiosis, Chalicosis, Anthracosis.—Men engaged in certain occupations, of which marble-cutting is the most conspicuous example, frequently show lung changes in the roentgenogram which so closely resemble those of tuberculosis that differentiation is impossible. That these are not

actually tuberculous is proven by the fact that the lungs regain their normal appearance when a temporary change of occupation is made.

Syphilitic Pneumonia.—Certain cases of tertiary syphilis show lung changes closely resembling chronic pulmonary tuberculosis. The history and other clinical evidence must be relied upon for differentiation. Value of clinical data.

Malignant Metastases.—Metastases from malignant tumors of the mediastinum and elsewhere produce shadow changes in the lung fields not greatly unlike those of advanced tuberculosis. The individual lesion, however, is usually much more conspicuous and massive in appearance. More distinct and massive.

CHARACTERISTICS OF OTHER PULMONARY LESIONS.—Chronic Bronchitis.—In many cases of long standing, one notes the marked translucency of basal emphysema in distinct contrast to the increased density of the poorly aërated lung above. An *accentuation* of the normal lung markings may be observed and the bronchial tree shadows may have a broken appearance, like the network described by certain authors as characteristic of tuberculosis.

Bronchiectasis.—Demonstration of bronchiectatic cavities is relatively easy when they are empty and surrounded by thickened lung tissue. The examination should be made in the morning after the paroxysm of coughing has emptied the cavity or cavities. Repeating the roentgenization on the succeeding day, one may note that a light center has appeared in certain areas which were homologous shadows the day before, the secretory contents of the cavity having been coughed up in the interim. The entire lung field may be sponge-like in appearance, at first glance, but upon more careful study in the stereoscope we may differentiate a multitude of small bean-shaped cavities, connected together by dense strands of tissue. Necessary precaution. Multiple bronchiectases.

Tumors.—*Primary carcinoma of the bronchial mucous membrane has been difficult to diagnose by percussion or auscultation because its usual location is at the hilus, whence as a dense fan-shaped shadow it spreads toward the periphery.* Characteristic shadow.

Metastases to the lung usually appear as rather dense shadows, often multiple, surrounded by areas of lesser density. After heavy therapeutic radiation, the involved lung area sometimes assumes a heavily striated appearance, suggesting fibrosis. Whether this is a reaction to the radiation or the disease is not yet known. Metastases.

Foreign Bodies in the Bronchi.—These may be localized with considerable accuracy in the stereoscopic roentgenogram. Even when not opaque to the X-rays and hence not to be directly visualized, Willis Manges working with the clinical material of Dr. Chevalier Jackson, has demonstrated that it is possible to diagnose foreign body upon the presence of an emphysema on the affected side. Localization.

Exudative Pleuritis.—(*Pleuritis exudativa*).—This presents a variable roentgen picture, according to the size and position of the exudate, its character, and many other factors.

There may be marked displacement of the neighboring organs with relatively little exudate, or the reverse may be true. Visceral displacement.

While easy to recognize in typical cases, one may meet with some difficulty

Surmounting diagnostic obstacles.

Small effusions.

in those complicated by old fibrous thickening of the pleura the shadow of which may cover that of the fluid. Here the fluoroscopic screen is much superior to the roentgenogram, because one may observe the effect of respiration and of change of position upon the density of the shadow. Even a small

FIG. 130.—Pleurisy with effusion. Note density of fluid shadow and its *horizontal* upper border. Note also cardiac displacement. (*Dr. Frank S. Bissell.*)

exudate may usually be recognized early as it fills the complementary pleural sinuses, obliterates the phreno-costal angle, and interferes with the normal excursion of the diaphragm of the side affected. As the fluid increases in quantity, one notes its tendency to climb along the lateral wall toward the axilla. This is the so-called "curve of Damoiseau." This curve tends to

disappear as the upper level of the fluid descends to a point below the lung hilus. It also disappears when the patient is in the horizontal position.

Early visceral displacement.

The heart and other mediastinal organs may become displaced toward the opposite side before the exudate has become large enough to exert direct pressure.

Pull and counterpull.

This displacement is the result of an elastic pull of the normal lung, the compressed lung of the affected side having ceased to exert its normal counter-pull. When the exudate is sufficiently large, the displacement of the mediastinum including the heart and great vessels produces an elongated, triangular shadow with its base on the diaphragm and its apex at the sterno-clavicular articulation. This shadow furnishes information as to the amount of effusion and the expediency of puncture.

Triangular shadow.

Differentiation from pneumothorax.

The line of separation between exudate and lung is not a sharp one, there being a noticeable difference between the sharp straight upper border of the usual pneumothorax exudate and the more indistinct washed-out border of a pleuritic exudate. While a very large pneumothorax exudate, such as may follow spontaneous rupture of the lung, may be recognized as such with great difficulty, the air above the fluid having been fully reabsorbed, careful search will usually reveal a small pneumothorax bubble.

Unless the roentgenologist is fortified with a suggestive anamnesia, however, he may mistake such an exudate for one of pleuritis. When air still remains above the fluid level, the latter's sharp distinct line readily suggests the diagnosis.

Distinct line of fluid level.

A point of great importance.

The recognition of the character of this exudate is highly important because its complete removal by paracentesis, as in pleurisy, is a grave technical error.

Pleuritic Adhesions.—*Pleuritic adhesions occur either as broad laminæ or as fine bands.* When the former occur in such a manner as to obliterate the costophrenic sinus, their differentiation from a small exudate is difficult but unimportant. A small exudate retained within the thickened pleura may entirely escape detection. If the thickened pleura lies near the plate or screen, its projected shadow is much more distinct than when it is on the opposite wall of the chest cavity.

Adhesions.

Fine pleuro-diaphragmatic adhesions are rarely if ever seen, but in some cases they may be recognized by the characteristic manner in which they cause the diaphragm to kink, tent-like, during deep inspiration.

Fixation of diaphragm.

Fixation of the diaphragm is usually present with pleuritis even though no fluid or adhesions are present. This immobilization may be difficult to determine when there is an effusion on the right side since the diaphragm itself cannot be visualized. On the left side, however, the presence of gas or air within the stomach renders the lower side of the diaphragm visible. The immobilization may persist for a long time after the fluid has become absorbed.

Pneumothorax.—*Uncomplicated by adhesions, exudate, etc., pneumothorax is recognized chiefly by the absence of normal lung markings, within the thoracic cavity. The unilateral area involved is much more translucent than its normal fellow, and the compressed lung may be observed, reduced in volume to a variable degree, and crowded sharply against the median shadow.*

Differentiation of types.

Displacement of the mediastinal organs occurs as a result of the elastic pull of the normal lung and of the pressure from the accumulation of trapped air in the pneumothorax cavity.

Artificial Pneumothorax.—Without roentgenographic examination, artificial pneumothorax is not practicable. The roentgenologist is usually asked to determine the two most important questions: first, whether pneumothorax is possible, and second, whether it is indicated.

Two important questions.

While it is not possible to determine these points with certainty, it is possible to do so with a considerable degree of probability. Are there pleuro-pulmonary adhesions, and if so, what is their topography, extent and firmness? Diaphragmatic adhesions can at least be excluded when not present. When there is a partial or complete effacement of the costo-diaphragmatic sinus, immobilization of the diaphragm, or suppression of the respiratory movements, the base of the lung is probably adherent.

The question as to the indication for pneumothorax is determined by the presence of cavitation and the absence of involvement in the other lung. Examinations made after an attempt at pneumothorax determine the degree of success and thus whether the procedure should be continued or abandoned. Serial examinations may demonstrate a gradual giving way of adhesions with final pulmonary collapse.

Special technic.

Pneumonia.—*In the study of pneumonia with the X-ray, technic is of the utmost importance. The lobe affected must be brought as close as possible to the roentgen plate, and tubes af slight penetration must be employed.*

Early signs.

Early in the disease, only a very faint diffuse density, due to decreased air content is noted. In a few hours this may have become a distinct shadow, sharply defined by the boundaries of the lobe involved. The subsequent course is very variable. The shadow of a pneumonic lung rarely reaches the density seen with tumor or effusion. The so-called pneumonic triangle, a dense shadow with its base toward the axilla, occurs most frequently in childhood and when present is very characteristic.

Pneumonic triangle.

Hilus pneumonia in influenza epidemic.

Hilus pneumonia was a common type during the influenza epidemic of 1918 and could be recognized only by X-ray examination. This type was often hemorrhagic in character, and very accurate prognoses could be based upon a study of serial plates made during the first days of the disease. When the process became bilateral it was almost invariably fatal.

Usually soon after the crisis a clearing-up process is perceptible, proceeding most frequently from the hilus toward the periphery. *Certain veil-like shadows usually persist, however, and these may be observed many days or weeks after resolution.*

Broncho-pneumonia.—Here the process is not limited to a single lobe but presents disseminated shadows usually obscure and poorly-defined and involving different lobes of one or both lungs. This disease is characterized by the most bizarre roentgen manifestations, quite unlike those of any other acute infection. However, the chief value of X-ray studies here lies in the aid afforded to prognosis, which apparently varies directly with the number of lobes involved and the rapidity with which the process extends.

The Heart in Pneumonia.—Certain German observers (Holzknecht, Steyrer) have called attention to the condition of the heart during pneumonia. Holzknecht noted early in the disease a distinct enlargement of the right side of the heart and a marked pulsation of the middle convexity of the

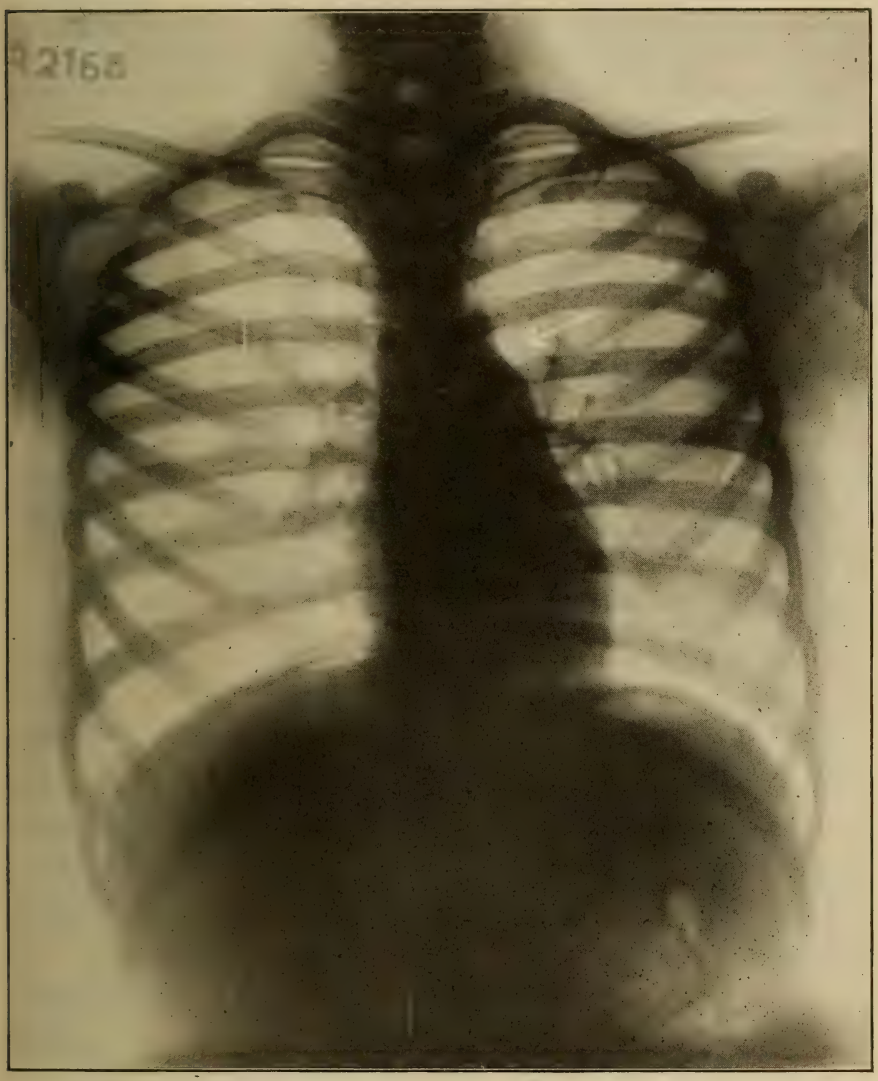


FIG. 131.—Resolving pneumonia. Note marked prominence of the intermediate convexity of the heart border. This is frequently observed in acute pneumonic conditions; probably represents residual dilatation of the pulmonary artery.

left border, representing left auricle and pulmonary artery. This indicates, in his opinion, a dilatation of the right heart and an over-distention of the pulmonary artery, conclusions which are wholly in accord with long-established bedside observations. Soon after the crisis these signs entirely disappeared.

Lung Abscess.—A more or less localized area of increased density situated entirely within the lung field is suggestive of lung abscess. The case history and clinical evidence are necessary to complete the diagnosis. Dr. William H. Stewart has reported successful diagnoses and localizations of abscess by means of bismuth paste injections through the bronchoscope. A pure roentgenological differentiation between abscess and infarct is probably not possible since both occur within the lung and have ill-defined borders by reason of the inflammatory process surrounding them.

Gangrene.—Gangrene has no characteristic roentgen signs but the presence of an hydropneumothorax together with clinical evidence, may make the diagnosis.

Empyema.—An empyema resembles a simple pleuritic effusion in the intensity and homogeneity of its shadow. Its borders assume various shapes dependent upon the adhesions which surround it. In the interlobar form, one depends upon the location of the shadow with relation to various lobes for its identification. Its most characteristic appearance is that of a transverse opaque band completely bisecting the lung field.

Emphysema.—With an increase in the air of the lung there is a proportionate and usually striking increase in its penetrability to the ray, *i.e.*, the brightness of the pulmonary fluoroscopic fields. While in the normal lung there is a marked difference between the density during inspiration and expiration, in the presense of a general emphysema this variation is not demonstrable. Other roentgenological signs of emphysema are, horizontal ribs, broad lung fields, and a flat diaphragm.

COUGH AND SPUTUM

COUGH.—Definition.—*The term cough covers single or multiple, consecutive, explosive, expiratory acts immediately following glottic closure.*

Diverse Causes.—It may be voluntary, involuntary or truly paroxysmal and arise from any of a multitude of causes and from the irritation of widely separated and diverse regions, though commonly purposeful and intended to remove irritating material from the bronchi or throat. This irritant is ordinarily the secretion accompanying acute or chronic disease of the air passages, or, foreign bodies.

Sensitive areas.

The most sensitive regions are the interarytenoid space, the tracheal bifurcation, pharynx, base of the tongue, naso-pharynx and certain areas in the nasal passages proper.

Reflex Cough.—Many forms of *reflex cough* are observed and these should always receive consideration in obscure cases.

Arnold's branch of the pneumogastric nerve is accountable for a rare cough connected with irritation of the external auditory canal.* So also chill-

* Such a case recently observed by the author was instantly and permanently relieved by the removal of impacted cerumen. The patient had been for several months under the treatment of a quack and, when bankrupt, was told, with engaging delicacy and frankness, that his lungs were "all rotted" and his case hopeless.

ing of the surface, pressure upon or diseases of the spleen or liver, when associated with involvement of the diaphragmatic pleura, may be the source of obstinate coughs. In women, chronic disease of the pelvic organs is an occasional cause and such a cough frequently disappears entirely after operation.

Hysteric, Dyspeptic and Tobacco Cough.—Other coughs are purely neurotic in origin, such being the obtrusive, forced, *hysterical cough* and the *barking cough* of puberty. *Dentition cough* in infants is apparently purely reflex in character but the so-called "*stomach cough*," associated ordinarily with chronic gastritis and most frequently observed in drinkers, is no doubt due to the accompanying pharyngitis and the same statement applies to the "*smoker's cough*" and its associated laryngeal irritation and hyperesthesia.

Smoker's cough.

Many cases due to excessive irritability of sensitive turbinate areas are observed, particularly in young people and neurotic individuals, the conditions being very similar to those associated with certain asthmatic paroxysms or with hay fever.

Nasal cough.

Direct Sources.—*It should be remembered that irritation of the lung parenchyma itself does not cause cough, but unquestionably irritation or inflammation of the bronchial mucous membranes or pleura does.*

Bronchial cough.

Pleural cough.

Therefore, in pulmonary disease, acute or chronic, the cough is attributable either to bronchial or pleural irritation. The assumption that any cough is purely reflex or neurotic should be postponed until all other channels have been thoroughly investigated.

Source of error.

"*Adenoids*," enlarged tonsils, impacted cerumen, granular pharyngitis, hypertrophy of the lingual tonsil, enlarged turbinates, chronic disease of the accessory sinuses, goiter, chronic heart disease, diseases of the liver, enlarged bronchial glands, aneurysm, mediastinal growths, dorsal caries, occupation, habit, and imitation, are some of the many other *potential or possible* conditions to be considered aside from affections of the bronchi, lungs, and pleuræ.

A host of possibilities.

Dry and Moist Cough.—In incipient phthisis and the early stages of bronchial or broncho-pulmonary inflammation the cough is wholly or relatively unproductive, *i.e.*, dry.

Such a cough may be extremely *urgent*, *paroxysmal*, or *painful* and acute bronchitis, pneumonia, whooping cough, asthma, and pleurisy furnish the best examples. In their early stages, practically all of the nervous, reflex, or pressure coughs are dry.

Dry cough.

When pain is present an effort is made to *suppress the cough*, as is the case especially in lobar pneumonia and pleurisy.

Suppressed cough.

Paroxysmal cough, best exemplified by whooping cough, may be encountered in many other conditions. Whether due to direct irritation, as in the case of a laryngeal tumor, or to reflex causes, as in impacted cerumen or turgescence of the nasal passages, and particularly in pertussis, the cough paroxysm may be associated with vomiting and even with epistaxis and hemorrhages under the skin.

Many sources.

Vomiting, nosebleed, and ecchymoses.

A paroxysmal cough may also result from a gradual, silent, accumulation of secretion and be attended by a profuse and perhaps sudden discharge, as,

Emptying of cavities.

for example, in bronchiectasis, certain pulmonary cavities, or the rupture of abscesses into the bronchi.

Pressure cough due to mediastinal tumors, pericardial effusion and like causes, is not usually paroxysmal but may be most misleadingly so, even to the simulation of asthmatic seizures.

Hacking cough.

The so-called *hacking cough* is especially common in incipient tuberculosis, but may occur also in acute or chronic irritation or inflammation of the upper air passages.

VARIOUS TYPES.—"Barking," "Hollow" and "Brazen" Coughs.—*Barking cough*, if not neurotic, is usually associated with inflammation of the glottis; a *hollow cough* with advanced tuberculosis; and *ringing, metallic or brazen cough* with mediastinal pressure from whatever cause. The author has observed as good examples of the brazen cough in massive pericardial effusion as from its commoner causes, aneurysm and mediastinal growths.

Diminuendo cough.

In emphysema the cough is peculiar in its prolongation of the individual expirations and the manifest forcing and prolonging of the series *diminuendo*.

"Crowing."

The "hoarse cough" of croup and the *inspiratory crowing* during the paroxysm are but too well known, and a *noiseless cough* may occur in certain

Toneless cough.

forms of glottic paralysis or a *toneless cough* in terminal cases of pulmonary disease.

The inspiratory whoop may follow various forms of paroxysmal cough, but is occasional and unusual, whereas in established whooping cough it is persistent and practically pathognomonic.

Significant variations.

An Ominous Sign.—*Absence or cessation of cough, with persisting physical signs, may indicate profound toxemia, excessive weakness or approaching death and is seen especially in fatal pneumonias of infancy, childhood, and old age.*

Localized momentary protrusion normally occurs in the apices and upper intercostal spaces during cough. The condition is exaggerated in emphysema and diminished in infiltration of the lung apices.

Pulmonary cavities and bronchiectases.

Postural Modifications.—In certain conditions posture markedly modifies the tendency to cough and in bronchiectasis, open pulmonary abscess and certain large phthisical or gangrenous cavities, a severe paroxysm of cough may be induced by a change of position which initiates and facilitates a flow of secretion from the cavity to the bronchi. Such patients may of their own volition assume a posture which, by lowering the head and thorax, drains the cavity by gravity assisted by the cough.

THE SPUTUM

Definition.—The sputum proper is that content of the air passages obtained by "clearing the throat" or coughing. The term embraces nasal, pharyngeal, laryngeal, bronchial, alveolar and, inevitably, a certain admixture of oral secretion.

All sputum save that representing pure pus or blood is mucoid in consistence and the general term embraces all similar material derived from fistulous communication with adjacent structures, or destructive processes

within the respiratory tract, *i.e.*, perforating abscess, echinococcus cysts, cavities, etc.

The Important Factors.—*One must observe: (a) Reaction. (b) Color and transparency. (c) Air content. (d) Consistence. (e) Amount. (f) Odor. (g) Albumin content. (h) Microscopic findings.*

The reaction is alkaline save after certain decomposition processes outside the body.

Color.—*Aside from misleading tints derived from food or medicines, the color* varies, according to its source and composition, from the colorless mucoid or serous sputum to that of pure blood. Slight purulent admixture (*mucopurulent sputum*) or predominance of pus (*purulent*) produces various degrees of yellow or greenish yellow. *Dust and soot* inhalation gives a dingy or grayish tint, and bile pigment or the development of certain germs, may produce a faint or even vivid green. *Blood* may be pure or represented only by faint pink, light brown or salmon color, iron rust shading, faint yellows and greens or the thin serous so-called "prune-juice" sputum. *True icteric sputum* appears only when actual jaundice or perforating hepatic abscess is present, though many blood-containing specimens of unusual tint react to the test for bile pigment. Greenish tints may occur in carcinoma, chloroma and various other conditions, and various occupational sputa are encountered, as in mirror polishers, aniline dye workers and others.

Soot.

Blood.

Bile.

Heart Disease Cells.—*In disease of the mitral valve with brown sputum peculiar pigment-holding cells are present.* In the perforating abscess of amebic dysentery the "*anchovy sauce*" *sputum* may be encountered, and in hysteria there is occasionally a viscid or jelly-like sputum exactly like *crushed raspberries*, which may create much unnecessary alarm.*

Bizarre forms.

A similar "raspberry-like" sputum is said to occur in certain cases of tumor of the lungs.

Air Content.—Foaminess and low specific gravity measure the air content and *all serous sputum is frothy.*

Cavity sputa dropped in water appear as globules, flattening to coin-like bodies (nummular sputum) as they sink to the bottom and, in general, the greater the pus content the less the amount of contained air, and the smaller the tubes of origin the greater is the air content.

Nummular sputum.

In bronchiectasis and gangrene one finds three distinct layers because of the varying specific gravity of the constituent elements.

Consistence.—*Pus or serum content determines fluidity, and mucus and fibrin (croupous pneumonia) the viscidity and tenacity of sputum.*

Fluidity vs. Viscidity.

Macroscopic Appearance.—A proper background for macroscopic examination is readily obtained by laying a piece of glass or a Petri dish

* A typical example was encountered by the author at the country place of some old friends whose sympathies and ministrations were in process of bestowal upon a most unworthy object. An hysterical young female degenerate had entered the grounds and gracefully cast herself down under the bellies of a sedate team of horses. She then attempted to simulate coma and convulsions with considerable skill until the doctor arrived. Her "crushed raspberry" sputum had excited even more alarm than her other spurious symptoms.

over black paper or cloth or employing a special glass plate with half its under-surface black.

One may find (*a*) **"Curschmann's spirals,"** which are not pathognomonic of asthma, but most frequent in that disease. They are refractile, visible to the naked eye and represent probably a bronchiolitic exudation. Though interesting they have no great diagnostic importance.

(*b*) **"Dittrich's plugs"** are yellowish-white, mustard-seed sized, *foul-smelling* aggregations of fatty acid crystals and bacteria, closely resembling the tonsillar plugs of follicular tonsillitis and are found in decomposition processes (pulmonary gangrene, foul cavities, etc.).

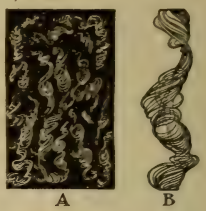


FIG. 132.—Curschmann's spirals. A, unmagnified. B, magnified.

(*c*) **Misleading food particles** may be encountered which are usually starchy and strike a blue color with Lugol's solution.

(*d*) **Fibrin masses** are white and extremely tenacious and are rendered clearer and bulkier by the addition of acetic acid. They vary in size from the tiniest plugs to casts of the bronchial tubes (croupous pneumonia, fibrinous bronchitis), or the diphtheritic membrane.

Fibrinous casts.

(*e*) **The Fibrinous Casts.**—These show best if shaken up in water.

(*f*) *Rarely and especially in old cases of tuberculosis,* **calcareous plugs** *or definite casts, often of considerable size are expectorated,* or* **foreign bodies** *of recent or ancient introduction may appear.*

All sorts of substances may be introduced by fistulous communication with abscesses, echinococcus cysts, etc., etc.

Collecting the sputum.

Amount.—Whenever possible the twenty-four hours' sputum should be obtained, and the transparent sputum cups are far better than the metallic and paper cups so generally used. Early stages of inflammation yield scant secretion. The morning is the most productive period in chronic processes though in pulmonary cavity and especially bronchiectasis large amounts may be raised at irregular times, often when assuming a special posture. Advanced pulmonary tuberculosis, bronchorrhea, bronchiectasis, perforating abscesses, pulmonary edema and resolving pneumonia furnish large amounts.

Foul odors.

Odor.—Ordinarily odorless when first raised, it may be extremely foul in any process attended with decomposition within the lung (bronchiectasis, certain tuberculous cavities, abscess of liver, communicating empyema) and characteristically so in gangrene.

Slight differential value.

Albumin Content.—The albumin content of the sputum constitutes a rough measure of the severity of an inflammation and is said to be absent in simple bronchitis. Possibly some slight value may be attached to a decided percentage as differentiating simple bronchitis from pneumonic processes and from pulmonary tuberculosis.

Zenoni's Differential Color Test.—This pretty and simple test for albu-

* In a case observed some time ago a large lung stone was ejected after the sudden onset of a protracted and violent coughing seizure and no symptoms of the old disease had been manifest for over twenty years.

min and mucin consists in spreading the sputum on a slide, fixing it with alcohol (fifteen minutes) and staining with a half-saturated aqueous solution of safranin. The albumin is stained red and the mucin yellow. Albumin and mucin.

MICROSCOPIC FINDINGS.—Staining.—The sputum should first be examined as a flat preparation without staining and then by making a smear, drying in the air and staining for three minutes with Wright's stain, thoroughly washing, and mounting. General stain.

Cells.—One may find:

(*a*) **Flat pavement cells** from the mouth and pharynx.

(*b*) **Columnar ciliated cells** from the larynx, trachea and bronchi.

(*c*) **Mono- or polynuclear cells** from the alveoli which may contain

(*d*) **Irregular highly refractile masses of myelin,** showing concentric layers.

(*e*) **Blood-pigment-bearing** (heart disease) cells.

(*f*) **Hematoidin crystals.**

(*g*) **Fat.**

(*h*) **Carbon.**

(*i*) **Free myelin bodies.**

(*j*) **Elastic fibers.** If present these last structures may be obtained sometimes by merely pressing a cheesy, granular, or especially dense, portion of sputum between two glass slides and using a low-power lens, but a more elaborate procedure is usually required.* Such fibers indicate actual destruction of lung tissue, most frequently advanced tuberculosis or pulmonary abscess, and usually show the alveolar arrangement. Staining is quite unnecessary, the only difficulty being the possible derivation of the fibers from retained food in specimens not properly safeguarded in the process of collection. *Owing to the presence of a peculiar ferment, they often are absent in destructive pulmonary gangrene.* Significance. Spontaneous disappearance.

(*k*) **Leucocytes.** Eosinophiles are abundant in asthmatic sputum and neutrophiles in all sputa. These may show phagocytic inclusions of fat, carbon or hematoidin.

(*l*) **Erythrocytes.** Microscopic blood exists in most sputa associated with severe cough, but macroscopically it is present only in violent paroxysmal cough, accidental hemorrhage and true inflammatory or necrotic processes. Fibrin, if present, may be demonstrated by Weigert's method.

(*m*) **Crystals.** The rhomboidal crystals of *cholesterin* indicate emphysema or lung abscess, seldom phthisis. The sharp slender fasciculated *fatty acid crystals* suggest gangrene or advanced tuberculosis, while the colorless, octahedral *Charcot-Leyden crystals* point to bronchial asthma. Yellow or brown amorphous masses or rhomboidal crystals of hematoidin point to blood retention and ulceration within the alveoli, and leucin and tyrosin may be present in emphysema. Emphysema or lung abscess. Asthma crystals.

(*n*) **Bacteria.** Among those occurring in sputum are: *tubercle bacilli* Simulators of tubercle bacilli.

* Boiling a mixture consisting of equal parts of sputum and 10 per cent. NaOH or KOH, setting aside for twenty-four hours and then examining selected portions of the precipitate is a useful device and search is seldom successful without it.

and its simulators, the smegma and timothy bacilli, and, those of pneumonia, anthrax, influenza, typhoid, glanders, plague and leprosy as well as the Friedlander bacillus, micrococcus tetragenous and others.

Animal inoculation.

THE TUBERCLE BACILLUS.—*The tubercle bacillus is by far the most important, and for its determination most careful work and even animal inoculation may be necessary.* In most instances it is easy to find the germ in the cheesy particles or thicker portions of the specimen, but at other times they must be obtained by centrifugation of the sputum.

The bacilli are often readily found in sputum thinned by spontaneous decomposition.

Rapid preliminary examination.

To Secure a Concentrated Sediment.—The Antiformin Method.—If the specimen yields none of the acid- and- alcohol-fast tubercle bacilli to the first rapid examination by the Gabbett or Ziehl-Neelsen method, one can

Centrifugation.

procure a concentrated centrifugated sediment by the use of *antiformin* (a trade mixture of sodium hypochlorite and sodium hydrate solutions), which keeps fairly well and is readily obtainable.

Loeffler's Modification of Uhlenhuth's Antiformin Procedure.—(*a*) *Boil, for a period not exceeding fifteen minutes, equal parts of sputum and* 50 *per cent. antiformin.*

(*b*) *Make a mixture of* 1 *part chloroform to* 9 *parts alcohol.*

(*c*) *Add* 3 *c.c. of this mixture to* 20 *c.c. of the original boiled mixture of sputum and antiformin.*

(*d*) *Shake to form a fine emulsion.*

(*e*) *Put required portions in corked sedimentation tubes and centrifuge for fifteen minutes.*

The original process destroys all save the acid-fast organisms. The chloroform in the centrifugated mixture holds the tubercle bacilli in a film at its upper margin.

(*f*) *The supernatant fluid is decanted and the film and its contained materials is then spread on slides, dried, treated with egg albumin in the usual manner, to insure subsequent secure fixation and is then ready for the usual stain, that most generally used being the Ziehl-Neelsen.*

The various steps of the staining process beginning with the removal of the centrifuged organisms in the film above the chloroform (stage "*e*") would be as follows:

Ziehl-Neelsen Method.—(*a*) Making a thin, even, smear after adding a little egg albumin or, better still, some of the original sputum, in order that subsequent firm fixation may be attained.

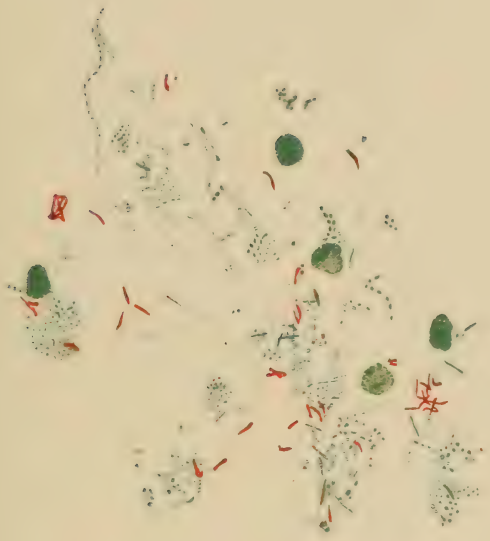
(*b*) *Drying* by gentle heat or passing to and fro through the air.

(*c*) *Fixing.*—The cover-glass smear is then fixed by passing it deliberately but steadily through an alcohol (or small Bunsen) flame four or five times, or ten or twelve times, if slides be used.

(*d*) *Staining.*—It is then completely covered with the fuchsin* (acid)

* To make this stain, a saturated alcoholic solution of fuchsin or gentian violet is added drop by drop to 5 per cent. solution of carbolic acid until a surface sheen appears.

PLATE II.



Tubercle bacilli in sputum (Ziehl-Neelsen method). The plate is an unusually good presentation of the staining (red), form, and usual microscopic field relationships, of the tiny rods. (*Webster.*)

stain, and is gently heated over a flame for three or four minutes to such a degree as to cause sustained steaming but not boiling. A few more drops of stain may be added as evaporation takes place.

(*e*) *Washing and Decolorization.*—It is then washed thoroughly and decolorized with a 10 per cent. solution of sulphuric acid in 95 per cent. alcohol until only the faintest pink remnant of the fuchsin can be seen on well-spread sections of the smear.

(*f*) *Washing and Contrastaining.*—It is then washed and stained one minute with Löffler's methylene blue* as a contrast stain—washed, dried and mounted for examination.

The tubercle bacilli appear as red rods in a light blue field.

Advantages of the Antiform in Methods.—(*a*) *Greatly increased power to detect small numbers of the acid-fast bacilli in sputum or other secretions and excretions.*

(*b*) *Destruction of other organisms with preservation of the acid-fast bacilli.* Inasmuch as a solution may be obtained in twenty-four hours without boiling and at room temperature, or in six hours if the incubator be used, it follows that—

(*c*) *It facilitates the making of pure cultures if this step be desirable.*

(*d*) *It permits direct animal inoculation in urgent cases for in such preparations all but the acid-fast bacilli are destroyed and these remain living and concentrated.*

Gabbett's Method.—This somewhat coarse but extremely rapid and convenient method may be used, but negative results should count for nothing because of its liability to decolorize certain of the tubercle bacilli and the fact that it does not exclude the smegma or timothy bacillus for the decolorization of which the acid alcohol is essential.

Convenient.

Uncertain negation.

It does, however, give rapidly a very clear and definite picture with the limitations mentioned above.

Procedure.—After spreading, drying and fixing one proceeds as follows:

1. *Stain.—Carbol-fuchsin as in Ziehl-Neelsen.*

2. *Throw off stain and add Gabbett's solution* (methylene blue 2.0; sulphuric acid 25, and distilled water sufficient to make 100) *without washing slide or cover-glass and allowing but one-half minute of contact.*

3. *Wash in water.*

4. *If thinner areas are still red repeat the Gabbett's stain and washing.*

5. *When they are become blue, dry and mount in balsam.*†

Caution.—*The greatest patience is required in these examinations and diagnosis should never be based upon a doubtful finding.*

Wherever there is one germ, more can and must be found and terrible mistakes result from hasty conclusions though these are far less deadly than the too common failure to find the germ when present.

* Löffler's alkaline methylene blue—sat. alcohol sol. methylene blue added to 100 c.c. of a 1–10,000 solution of potassium hydrate.

† Prolonged single applications may decolorize the "acid-fast" bacilli.

RHINOLOGY, PHARYNGOLOGY AND LARYNGOLOGY

A surgical specialty.

Diagnosis at Sight.—*The modern specialty which embraces these subjects has the advantage of permitting practically all its diagnoses to be made at sight through the use of highly specialized appliances.* Furthermore, it is almost wholly surgical; hence the student must consult special text-books for any adequate description.

Essentials.

Technic.—The essentials are (*a*) *strong, steady light and a good reflector.* (*b*) *Properly heated laryngoscopic and rhinoscopic mirrors.* (*c*) *A steady hand and a thorough knowledge of the anatomy of the parts and of the regional pathology.*

Technic.

The long-handled mirrors are held in the pen position and in the case of the larynx are introduced with the free edge downward and *without touching any structure* until they press back the soft palate. The tongue is grasped in a dry napkin between the index-finger and thumb of the unoccupied hand and held firmly forward either by the operator or the educated patient himself. The index-finger of the examiner or the thumb, in the case of the patient, should rest against the lower teeth forming a support to steady the hand and prevent painful pressure upon the median raphé.

Larynx.—If the mirror has been warmed to prevent misting and is held at, but not touching, the upper angle of the pharyngeal vault, an excellent view of the larynx is obtained.

Vocal cords.

The upper portion of the image represents anterior, the lower, posterior structures. Deep *breathing* and *voice production* reveal the action of the vocal cords.

Naso-pharynx.—*In examining the naso-pharynx a* smaller mirror is introduced with its free edge upward well back to, but not against, the posterior pharyngeal wall. If steady, deep breathing (through the nose, so far as possible) and passivity maintains a relaxed soft palate the parts may be seen without difficulty, but oftentimes some one of the special forms of the palate retractor must be used to pull that structure firmly forward and slightly upward.

Obstacles to examination.

Hesitancy, fumbling and tremor on the part of the examiner invariably cause retching, and occasionally hyperesthesia may be so marked as to necessitate the preliminary administration of a bromide or the use of a dilute solution of cocain.*

The small mirror should invariably be surgically clean, repeatedly warmed to prevent misting, and tested for excessive heat by laying its back against the skin of the operator's hand.

Position of head.

The patient's head should be slightly tilted backward to best show the larynx and held perpendicular or inclined a trifle forward in examining the naso-pharynx.

The anterior nasal chamber is examined by direct light, the patient's head being tilted backward and the alæ widely separated by means of one

* Decided *anesthesia* is a valuable sign of hysteria.

of the various nasal dilators, bringing into view the vestibule, septum, inferior meatus, inferior turbinate, middle meatus and middle turbinate. Obstructing secretion is sprayed or wiped away and any interfering congestion is readily reduced by a weak solution of cocain and suprarenalin.

DISEASES OF THE NOSE

ACUTE CORYZA (*Acute Rhinitis*).—A description of this universally known ailment is unnecessary. The susceptibility of an individual is markedly increased by the existence of a chronic catarrhal inflammation of the nasal passages, exhaustion, chronic diseases and lowered vitality from whatever cause. The mucous membrane in such cases shows primarily congestion, swelling and diminished secretion, secondarily, the characteristic profuse mucopurulent discharge.

Predisposing factors.

Chronic rhinitis (chronic catarrh) resulting from repeated acute attacks or due to general impairment of nutrition, presents much the same appearances as acute rhinitis.

HYPERTROPHIC RHINITIS.—Through chronic catarrhal inflammation the vascular turbinate tissues become permanently distended and the epithelial layer thickened and infiltrated.

This condition is frequently associated with *exostoses* or *ecchondroses* of the nasal septum and is most marked at the anterior and posterior portions of the middle turbinate and the posterior portion of the inferior turbinate.

Nasal obstruction and catarrh resulting from such conditions, lead to mouth breathing and frequent acute or subacute coryzal attacks, and there is a troublesome recurrent obstruction especially marked in recumbency, such patients awaking in the morning with dry lips, coated tongue and oftentimes a foul breath. The imperfect drainage of secretion may cause obstruction of the nasal duct; the senses of smell, taste and hearing are impaired, and the Eustachian tube may be the seat of a catarrhal process. Headaches are common either as the result of infundibular occlusion or actual disease of the accessory sinuses.

Mouth breathing.

Deafness common.

Headache.

EXOSTOSES AND ECCHONDROSES.—These formations are recognized at sight and their consistence determined by the use of the probe.

SEPTAL DEVIATION.—This condition, rarely absent in slight degree, is pathologic only when it produces pressure or absolute obstruction. Marked deviations lacking a history of traumatism are usually associated with a high-arched palate and probably with previously existing adenoid growths of the naso-pharynx. Deviations may be partial or general, angular or curved, are readily recognized upon examination and usually associated with chronic catarrh.

When pathologic.

SEPTAL HEMATOMA is a blood-red tumor, usually the result of an injury.

SEPTAL ABSCESS forms a fluctuating swelling and is a frequent source of septal perforation.

Foul breath.

ATROPHIC RHINITIS (*Dry Catarrh*).—This condition usually follows a chronic hypertrophic rhinitis and is characterized by foul breath and a dry glazed mucous membrane carrying scanty and foul secretions which dry into adherent scabs and scales. Not infrequently, there is ulceration of underlying tissues and, in long-standing cases, actual structural atrophy, the turbinate bones being shrunken and the septum thin.* In delicate children the condition may co-exist with an excessively fetid, thin, watery secretion (ozena).

NECROSING ETHMOIDITIS.—Inflammation of the middle turbinate may cause necrosis of the inferior turbinate and ethmoidal structures. Periostitis and necrosis are usually followed by the exfoliation of a limited amount of bone. The symptoms are those of a suppurative inflammation specially involving the middle turbinate. Pressure deviation of the septum is common and nasal polypi frequently result and may remain as evidence of past disease. The probe introduced at the site of the suppuration usually detects the necrotic bone.

HAY FEVER (*Periodic Vasomotor Coryza*).—However obscure the causal factors, the disease offers few diagnostic difficulties.

The symptoms are merely those of an unusually obstinate or persistently recurrent acute coryza with a profuse watery, rather than mucopurulent, discharge, coming on periodically often at a stated day or week in each year

Periodicity.

and not infrequently associated, alternating with, or substituted by, attacks of pure bronchial asthma. The ailment is most frequent in the later spring months (rose cold) and in August and September (hay fever) and in some instances occurs irregularly or as the result of such influences as are described under the exciting causes in asthma. In certain cases a violent harassing and markedly paroxysmal cough may be present, slight fever may occur at intervals and the usual duration is from four to six weeks. This disease boasts a wonderfully voluminous literature bearing upon its etiology and treatment.

NASAL POLYPI.—These may be either mucous or fibrous, the former being soft and gelatinous, the latter hard. They are most common in the posterior chamber, may cause necrosis, invade the adjacent cavities, deform the nose and, in the case of fibrous growths, become sarcomatous.

Friable and vascular.

SARCOMA AND CARCINOMA.—The sarcomata are light red, usually sessile, bleed at the slightest touch, and are associated with ulceration and offensive discharge.

Usually secondary.

Carcinomata are usually epitheliomatous or encephaloid in type and are excessively rare as primary growths.

NASAL SYPHILIS.—Primary lesions are almost unknown, secondary lesions coincide with the appearance of the exanthem upon other parts, but tertiary lesions may appear as nodular swellings on any part of the nose.

Behaving here as elsewhere, they may undergo absorption if recognized and treated, or, a destructive necrosis, attended by profuse filthy secretion

Necrosis cases.

and a foul breath due to a rapidly spreading involvement of the surrounding

* The nasal chambers look extraordinarily and characteristically roomy in such cases.

and underlying tissues. Few more terrible pictures are presented than those of the malignant form of rapidly progressing syphilitic necrosis involving the nasal structure. Horrible cases.

EPISTAXIS.—*In nine cases out of every ten, any spontaneous nasal hemorrhage occurs as the result of a small erosion affecting a tiny group of veins on the anterior portion of the septal walls.* Most frequent source.

It is associated also with excessive physical and mental exertion, injuries, operations, ulcerations, tumors affecting the nasal mucous membrane, and venous trunk obstruction, as in the case of tumors of the throat and mediastinum. It occurs in cases of mitral and tricuspid disease and, with considerable frequency in cases showing high arterial pressure associated with interstitial nephritis, left ventricular hypertrophy and arteriosclerosis. In some of these instances no doubt, it acts as a safety valve and a substitute for phlebotomy. Widely varying causes.

Nasal congestion alone or occurring as a part of a cerebral congestion gives rise to single or repeated hemorrhages. It occurs as a prodromal symptom of acute infectious diseases, especially typhoid, and is a complication of profound dyscrasias, such as leukemia, hemophilia, pernicious anemia, scurvy and purpura, in which it may be fatal. Rarely it represents *vicarious menstruation* or substitutes hemorrhoidal bleeding, and it should be remembered that repeated hemorrhages, often disregarded or overlooked in case-taking, may be the source of a profound secondary anemia. Prodromal. Dyscrasic. Useful blood-letting.

Foreign Bodies in the Nose.—*In a child or insane person a mucoid discharge becoming purulent or perhaps fetid and bloody should suggest a foreign body in the nose, and demands a rhinoscopic examination.*

THE ACCESSORY SINUSES.—*Until the past few years the great and far-reaching importance of these cavities has been little appreciated.* Great importance.

They consist of the maxillary antrum (antrum of Highmore) and the frontal, ethmoidal and sphenoidal sinuses, any one of which may be the seat of inflammation and an acute or chronic sepsis which may lead to serious secondary septic absorption of either the acute of chronic type. Sepsis and septic absorption.

We have learned recently that these cavities play a large part in severe chronic or recurrent headaches and that many cases of chronic and persistent ill health with nebulous findings and intractable relapsing secondary anemias have their origin in these sinuses. Headache and anemia.

The Antrum of Highmore.—This, the largest of the accessory sinuses, opens into the middle meatus about 1 inch above its floor. Important anatomic relationships.

The roots of the teeth of the upper jaw and especially those of the molars are separated from the antral cavity by only a thin plate of bone.

If acute infection occurs through ordinary acute coryza, influenza, peridental abscess, necrosis, or what not, the antral outlet is readily blocked and retention or even abscess formation may follow. This condition is but too often overlooked and neglected whether in its acute or its chronic form. Retained secretion. Abscess.

Transillumination.—Most valuable information is afforded by the use of a small electric bulb of special pattern held within the mouth with the

Eye reflex.

lips closed. In a dark room the sound cheek and corresponding pupil show a dull red glare and if the eyes are closed the lower lid on the sound side and lower margin of the orbit show the same illumination.

Diagnostic shadow.

The affected side will show usually a more or less decided shadow or deficient illumination if the cavity contains a large amount of secretion or is the seat of inflammatory thickening, acute or chronic.

Special diagnostic procedure.

Inspection.—Inspection may show some drainage from the sinus, especially if the head be bent far forward, but in many instances retention is complete and direct exploration of the opening and the establishment of lavage or artificial drainage through the wall of the inferior meatus is necessary.

Special suction device.

In all sinus cases the use of suction apparatus applied to one nostril with the other nostril held closed while the patient repeats the word "tick," will often draw pus from the affected sinus.

Vasoconstrictor sprays.

In all sinus inflammations the use of vasoconstrictor sprays or douches may temporarily reëstablish drainage and aid in diagnosis both by this result and by the prompt but transient relief or amelioration of pain so often experienced.

THE FRONTAL SINUSES.—These like the maxillary antra open into the middle meatus and blocking of the outlet by mucus (mucocele) or mucopus (empyema) is an extremely common complication or sequence of various infections affecting the nasal passages or even of hypertrophy of the middle turbinate or the presence of polypi.

Mucocele and empyema.

Transillumination.—This maneuver is of service here as in the case of the maxillary antrum but the more direct method of inspection of the nose and, if necessary, the introduction of a probe or catheter into the sinus after a primary and thorough shrinking of the tissues by local applications, is more satisfactory.

Inspection.

Radical procedure.

If necessary, a resection of the anterior extremity of the middle turbinate may be performed and the results of direct inspection or exploration together with the subjective symptoms and the external signs, will aid in making a positive diagnosis.

Neglected cases.

The anterior wall of the sinus is relatively thin and neglected empyema with retention may cause actual necrosis and external pus accumulation. A decided redness, swelling or edema of the overlying tissues is sometimes present before necrosis is far advanced.

Important relationships.

THE ETHMOIDAL SINUSES.—*These lie along the inner orbital walls in dangerously close relation to the meninges and the cavernous sinus.*

Serious possibilities.

In neglected empyema the thin orbital boundary wall may yield and exophthalmos, strabismus, retrobulbar neuritis, ptosis, or even cavernous sinus thrombosis or basal meningitis result. The anterior cells, like the sphenoidal and nasal sinuses, open into the middle meatus, the posterior cells into the superior meatus.

Catheterization of sinus.

THE SPHENOIDAL SINUSES.—These lie at the base of the skull posteriorly and open into the superior meatus by a passageway which may be traversed by a curved probe passed back over the middle turbinate at the junction of its anterior and posterior halves if the instrument is in

specially skilled hands and proper preliminary cocainization and shrinkage of the tissues precedes the maneuver.

The intimate deep relationships of the sphenoidal cells indicate clearly the dangers accompanying empyema of these structures.

Symptoms.—In both ethmoidal and sphenoidal cases more or less severe deep-seated pain is felt in the orbit and back of the nose and often a deep boring pain in the occiput. Pain localization.

It is usually possible to demonstrate a discharge of mucus or mucopus into the middle meatus if the anterior ethmoidal cells are involved and from the inferior meatus if the posterior ethmoidal cells or the sphenoidal cells are affected. Nevertheless, in many instances, sphenoidal and posterior ethmoidal retention cases may be very obscure. Localization.

Pain in Disease of the Accessory Sinuses.—The pain of a sinusitis, mucocele or empyema with complete or intermittent retention may be very severe.

It is usually worse in the morning and this is especially true of cases of frontal sinus retention. "Sun pain."

Its maximum localization and its character are fairly definite or strongly suggestive in both frontal and maxillary sinus lesions but extremely indefinite in cases involving the sphenoidal regions.

It is evident that in all cases the specific diagnosis must depend chiefly upon direct inspection of the districts into which the various sinuses normally drain their secretion and by means of preliminary shrinkage of tissue, the use of the probe, and even of a special suction apparatus, the skilled specialist can usually make a positive diagnosis. Specific diagnosis.

Every physician should consider these regions in relation to the etiology of asthma, obscure chronic headache, rheumatism, anemia, arthritis deformans and even endocarditis, myocardial insufficiency and the nephritides, for they, like the tonsils though in less degree, offer exceptional opportunities for the continued growth and reproduction of pathogenic organisms. Etiologic importance.

DISEASES OF THE PHARYNX

ACUTE PHARYNGITIS.—In this ailment the throat is sore, stiff and dry. There is dysphagia, hoarseness and, in severe cases, swelling of the cervical glands. The patient is harassed by the recurrent cough and persistent pharyngeal irritation. Any secretion present is usually viscid and tenacious and causes persistently repeated but vain efforts to "clear the throat." "Hawking."

CHRONIC PHARYNGITIS.—This presents much the same symptoms during its period of exacerbation and frequently shows a special involvement of the follicles, giving the throat the granular appearance sometimes separately described as "*follicular pharyngitis.*"

ATROPHIC PHARYNGITIS.—The *atrophic form* shows a thin, glistening mucous membrane. The throat is dry and stiff. It may be dotted with islands of dried secretion and an harassing cough is common. The nasopharynx may be chiefly involved and cause a burning sensation. Most troublesome and irritating efforts to dislodge secretion result.

In acute conditions the secondary increase of secretion may be profuse. The atrophic form is especially disagreeable if it involves the naso-pharynx and a hypertrophic form may involve the lateral walls of the pharynx.

Importance.

POST-NASAL ADENOIDS.—*"The Curse of Childhood."—These lymphoid structures may greatly impair the health, physical and mental development, and future life, of children.*

They represent hypertrophied lymphoid tissue and form vascular growths, broad-based, sessile or pedunculated and sometimes moderately firm and fibrous. They are astonishingly frequent and may affect whole families. Excessively cold or damp and changeable climates seem to increase their frequency and, though the condition may be congenital, it is seldom symptomatically declared before the age of two years.

Frequency. Climate.

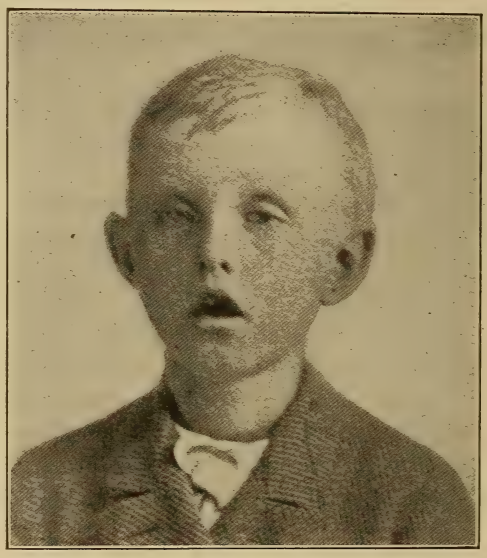


FIG. 133.—Adenoid Facies. The condition may exist with little or no evidence of this kind

The Symptoms.—These are varied and interesting. *In the typical and pronounced cases* the child is a mouth breather with the physiognomy shown in our illustration and in such cases the growth is usually large.

Mouth breathers.

Smaller bodies may nevertheless produce marked irritability, nocturnal restlessness and a complete change in the disposition and mental alertness of the child.

Invite disease.

In all cases there is an increased liability to acute coryza, otitis media, croup, tonsillitis, laryngitis, bronchitis and middle-ear disease, as well as added danger from attacks of measles, scarlet fever, whooping cough and diphtheria.

Sequelæ.

If unremoved, they may undergo atrophy as adult life approaches, usually leaving behind chronic naso-pharyngeal catarrh, *a high-arched palate, a deformed chest, and imperfectly expanded lungs.*

Mouth breathing, a muffled nasal tone, snoring, and the evident increase of respiratory obstruction in the dorsal recumbent position should call attention to these cases.

Impaired hearing is extremely common and is due to involvement of the Eustachian tube.

Reflex cause of disease.

Adenoids may cause asthma, enuresis, night terrors, teeth grinding, gastrointestinal disturbances, croup and possibly pseudo-epilepsy and choreiform manifestations.

Mental effect.

Retardation in mental development is often attributable to "adenoids" and this statement is equally true in relation to certain vicious traits in unruly children.

Direct Diagnosis.—A positive diagnosis is readily made by slipping the index finger behind the palate and exploring the post-nasal vault or, by the use of the mirror and retractor.

SYPHILITIC PHARYNGITIS.—A secondary syphilitic erythema is diffuse or takes the form of a *vertical red band* on the anterior pillars which ends abruptly at the uvula. Mucous patches, if present, tend to be symmetrical, but tertiary lesions are frequently unilateral and there is usually an associated laryngitis.

Usual site.

RETRO-PHARYNGEAL ABSCESS.—Such abscesses are *unilateral* and most often in the oro-pharynx, but may be opposite either the nasal or laryngeal portion and are often to be seen only with the mirror. Injury and caries of the vertebræ are the usual causes, though they may complicate tonsillar abscess, syphilis and tuberculosis. According to their location, they cause obstructed nasal breathing, spasmodic dyspnea, deafness, or tinnitus aurium, combined with symptoms of severe pharyngitis and the local and general symptoms of abscess.

Causes.

Symptoms.

PARALYSIS AND TUMORS.—Paralysis of the pharynx need not be separately considered and the tumors of this region may take any form. Paralysis of the soft palate may follow diphtheria and cause characteristic changes in the voice and difficulty in swallowing liquids.

DISEASES OF THE TONSILS

ACUTE TONSILLITIS.—This common disease of young people is rare in infants and in elderly persons. The relation of tonsillar infections, acute and chronic, to rheumatism, chorea, endocarditis and myocarditis is now definitely established and the remarkable instances in which chronic ill health, recurrent anemia and obscure albuminurias have been relieved by *complete* tonsillar enucleation are so frequent as to enormously increase the legitimate indications for that procedure.

Related ailments.

Importance of complete enucleation.

The recent studies of their bacterial flora show that tonsils which have been the seat of repeated attacks of tonsillitis are, as a rule, dangerous possessions and, unfortunately, one cannot always estimate their evil potency by the external appearance of the glands. Acute tonsillitis prevails chiefly in the spring in this country and affects both sexes equally.

Dangerous possessions.

Symptoms.—*A chill or chilliness and severe muscular and bone pains are followed by rapidly rising fever, sore throat and dysphagia. The temperature reaches* 103 *to* 105°*F., and the voice may be thick and nasal. The tonsils are swollen and dotted with a readily detachable exudate, which may become confluent, but tends to be confined to the limits of the tonsil.*

The disease lasts but a few days and is usually uncomplicated. In many instances the diphtheritic membrane is so closely simulated as to demand cultural tests and in certain cases of diphtheria the first appearance is that of follicular tonsillitis.

Exudate.

Despite the fact that most cases terminate favorably, one must remember that this ailment almost invariably precedes any attack of acute rheumatism.

Warning.

Furthermore, it is often the first known manifestation of many other ailments, the true nature of which may be revealed only after several or many days.

SUPPURATIVE TONSILLITIS (*Quinsy*).—*This differs from the preceding form chiefly in the predominatingly unilateral primary involvement of the peritonsillar tissues, the more violent onset, higher fever and pulse rate, the greater prostration present and its tendency to abscess formation.*

Usually unilateral.

The staphylococcus pyogenes is usually the dominant and predominant organism.

The local symptoms are those of pain, dysphagia and marked swelling of both the tonsil and the peritonsillar tissues. The cervical glands are enlarged, salivary secretion is increased and within two or three days fluctuation is evident and through rupture or, preferably, by the use of the knife the disease passes.

Dangers.

Both tonsils may be involved, the pus burrow with unexpected rapidity, and even an edema of the glottis may occur.

Quinsy should not be allowed to go on to spontaneous rupture.

CHRONIC TONSILLITIS.—Chronic hypertrophy, often associated with dense adhesions, is closely associated with so-called lymphatism, and the significance and effect of this lesion and of naso-pharyngeal adenoid growths is somewhat the same.

Furthermore, as stated previously, such tonsils are usually veritable commissaries and incubating ovens for the various strains of streptococci responsible for acute and chronic arthritis, endocarditis and myocarditis.

Incubators and commissaries.

Hypertrophied tonsils should not be allowed to remain in the throat of children or adults unless a distinct contraindication to their removal exists.

DISEASES OF THE LARYNX

SIMPLE, ACUTE AND SUBACUTE LARYNGITIS is too well known to need extended description, its chief characteristics being *varying degrees of hoarseness, painful and distressing cough, general congestion of the mucous membrane and vocal cords and scant viscid secretion.*

Chronic laryngitis is a common complication of other diseases of the respiratory tract and a very frequent form of *fatigue neurosis*, such as is experienced by singers, auctioneers, hucksters, army officers and others. In its milder forms it is associated with the excessive smoking of tobacco, overuse

A complication, sequel or neurosis.

of alcoholics, and the rheumatic or gouty diathesis. It is particularly frequent in all poorly nourished persons, especially mouth breathers, and such as live or work in dusty, steamy or vitiated atmospheres.

Symptoms.—*Chronic or recurrent hoarseness of varying degree, scanty tenacious secretion, local irritation, laryngeal congestion and more or less cough are the chief symptoms.* The vocal cords are sometimes unaffected and even whiter than normal, though occasionally their movement is restricted by swelling of adjacent structures. *The cough may be paroxysmal or violent and projectile*, the dried secretion acting like a foreign body. A form precisely similar to *atrophic* rhinitis and pharyngitis may occur.

Cords.

Cough.

Atrophic form.

Singer's Nodes.—These are papillary excrescences or nodules such as may be encountered on the true vocal cords of any victim of inveterate chronic laryngitis.

Edema of the Glottis.—*Cases of sudden onset and rapidly fatal termination may occur, but usually it is preceded by progressive inspiratory dyspnea with hoarseness or aphonia. The laryngoscope or even the finger at once reveals it and no condition more urgently demands prompt, often immediate and radical, treatment.** It should be remembered that this condition may complicate actual traumatism, especially burns, or the inhalation of poisonous and irritating gases, the ingestion of corrosive poisons, diphtheria, erysipelas, quinsy, scarlet fever, typhus, Bright's disease, syphilis, tuberculosis and diabetes mellitus. It is very rare in association with primary acute laryngitis.

Prompt relief demanded.

CROUP.—This well-nigh universal ailment of children, while possibly, quite distinct from the spasmophilic form known as laryngismus stridulus, is distinctly of a spasmodic type and, in the author's opinion, cannot properly be called merely an acute laryngitis. It occurs between the ages of six months and three years, rarely later. Deficiency of calcium in the diet, rickets, the irritation of teething, large tonsils and adenoids are important exciting or contributing factors. It is a distinctly spasmodic ailment associated with a mild and usually transient laryngeal catarrh and in many instances seems to indicate a close relationship to spasmophilia both as to exciting causes and basic etiology.

Symptoms.—The symptoms are those of spasmodic inspiratory dyspnea and laryngeal congestion. It is preceded by marked hoarseness, and during the spasm the child is cyanotic and asphyxiation may seem imminent.

The peculiar "crowing" sound attending the forced indraft of air through the glottic chink and the hoarse metallic cough are characteristic of the disease and all too familiar to both physician and layman.

"Crowing."

In many cases the attacks last only for a short time, usually coming on about midnight; in others, they may recur on several successive nights and

* In more than one instance a timid, hesitant physician has watched the slow suffocation of these patients. Cocain and adrenalin, on a swab or as spray, and scarification of the tissues may serve at least for temporary amelioration, but, as in the cases of sudden extreme laryngeal stenosis in the diphtheritic child, an emergency tracheotomy with a razor for scalpel and hair pins for retractors, if no better means offers, may be imperatively demanded.

the child is hoarse and has a croupy cough during the day. Convulsions rarely occur and death during the attacks is almost unknown.

A spasmophilic form.

LARYNGISMUS STRIDULUS is a term applied to an adductor laryngeal spasm which may be either nocturnal or *diurnal* and lacks the hoarseness and cough of croup. It corresponds to "passion fits," "child crowing," "holding the breath," of the laity, and convulsions, carpopedal or general, may occur.

It is a prominent member of the group of syndromes included under the term "*spasmophilic diathesis.*"

MEMBRANOUS CROUP (*Laryngeal Diphtheria*).—In rare instances, especially in measles and pertussis, this terrible disease is said to occur unassociated with diphtheria, and is credited to streptococcus infection, but for practical purposes any persistent membranous laryngeal stenosis must be considered and treated as diphtheria and demands prompt intubation and large doses of diphtheria antitoxin.

Probably always diphtheria.

Usually tertiary.

SYPHILITIC LARYNGITIS.—This is ordinarily a tertiary manifestation and may occur two or three decades after the primary infection. *The secondary stage may, however, give rise to hyperemia, symmetrical ulceration and condylomata which may resolve or ulcerate. The gumma may cause deep ulceration or necrosis with resulting stenosis from cicatricial contraction.* The specific tests are often necessary. Congenital laryngeal syphilis usually appears in infancy, more rarely at the age of puberty (*hereditaria tarda*).

Therapeutic test.

Seldom primary.

TUBERCULOUS LARYNGITIS.—Primary tuberculosis in this region is extremely rare and usually, though not always, the lesion indicates and is found to be associated with an advanced pulmonary lesion.*

Hoarseness, cough and aphonia.

Cords ashy gray and "moth-eaten."

Pyriform swelling and epiglottic turban.

Symptoms.—*The symptoms are those of a persistent chronic laryngitis, hoarseness or aphonia being a marked and early symptom. Swallowing is painful if there is epiglottic or pharyngeal ulceration. The laryngeal mucous membrane is at first pale and later an ashy gray, the arytenoids showing a pyriform swelling and the epiglottis becoming turban-shaped.*

The ulcers themselves are shallow and broad with gray granular bases and irregular, sharply cut outlines, and the vocal cords usually appear "moth-eaten" from ulceration. The tubercle bacillus can usually be demonstrated easily in the sputum and makes the diagnosis positive.

A *lupoid form* may be encountered, characterized by small gray nodules centered in a zone of redness and non-ulcerative.

TUMORS OF THE LARYNX.—*The general symptoms of laryngeal tumors are dysphonia and aphonia, intermittent or persistent dyspnea, cough, and sometimes laryngeal spasm.*

Certain growths above the cords are subjectively symptomless and some sessile growths produce strikingly intermittent and irregular symptoms with violent paroxysmal cough and are often difficult to see with the laryngoscope. Any extended discussion belongs to surgery.

* Nearly all of the supposedly primary cases prove to be secondary to advanced but latent or partially arrested pulmonary lesions. They will become still rarer as the X-ray is more generally used in diagnosis.

DISEASES OF THE BRONCHI, LUNGS AND PLEURA

BRONCHITIS

Etiology.—The general etiologic factors are essentially the same in all forms of simple bronchitis.

Exciting Causes.—In all forms of bronchitis bacteria play a prominent part: "*Micrococcus catarrhalis*," pneumococci, the streptococcus and staphylococcus, Friedlander's bacillus and many others are found, quite aside from specific infections in which a bronchitis is a secondary factor.

Predisposing Causes.—The extremes of life, a changeable, damp climate, whether temperate or tropic, and general low vitality, primary, or secondary to disease, alike invite it. Age, climate and previous health.

Chronic malaria, gout, alcoholism, Bright's disease, incompensated cardiovascular lesions, especially those associated with pulmonary stasis, cause or predispose to it and it invariably accompanies active tuberculosis, emphysema and asthma. Antecedent diseases.

In certain acute diseases, such as measles, typhoid, smallpox, influenza, and whooping cough, it is a common or invariable complication, but in so far as acute bronchitis is concerned, the common "coryza" is ordinarily the primary factor. Acute infections.

Males are chiefly affected by reason of their greater exposure both to climatic variations and the diseases of etiologic relationship. Sex.

Steel grinders, stone cutters, bakers, millers, stokers, felt workers, employees in flax and cotton mills and such as must be exposed to irritants of any sort show a special tendency to this disease. Occupation.

The foolhardy process of "*hardening*" young infants by excessive and reckless exposure to harsh weather and the irrational use of insufficient outdoor clothing helps greatly to swell the infant mortality though an increasing use of *intelligently regulated* open-air living for children and adults alike is one of the most hopeful advances in preventive medicine. "Hardening" infants.

Morbid Anatomy.—**In acute bronchitis,** the mucous membranes are at first inflamed, dry and irritable, later there is a reactive mucopurulent secretion, the membranes are swollen or edematous and, should the bronchioles become affected, a disseminated broncho-pneumonia ("*capillary bronchitis*") results.

In chronic bronchitis the long-continued inflammatory changes, often associated with and increased by chronic venous congestion, lead to permanent thickening or atrophy of the mucous membrane, with granulation or even ulceration, and it is one of the causes of the fusiform or saccular dilatations known as bronchiectases.

ACUTE TRACHEO-BRONCHITIS

Symptoms.—These are: *fever, usually moderate* (99.5° to 103°*F.*); *substernal pain, soreness or oppression*; *hoarseness, general malaise and cough, at first urgent, dry, ringing, harsh and somewhat painful, later moist and hollow.* Fever, substernal distress, and cough.

Sputum.

The sputum, at first mucoid and scant, soon becomes mucopurulent and sometimes profuse, and if the cough is violent may show blood streaks in the early stage.

Râles chiefly.

Physical Signs.—The only important physical signs are the *sibilant and sonorous râles* of the first stage, followed by the *mucous bubbling or even gurgling râles*, of the stage of exudation. Slight dyspnea and cyanosis *may* be evident, and palpation may reveal rhonchal fremitus. Marked cyanosis suggests disseminated broncho-pneumonia, a preëxistent emphysema, or a complicating cardiovascular lesion. In the bronchitis of influenza or typhoid fever, toxemia may be profound even in the absence of serious pulmonary lesions. Broncho-pneumonia is suggested by a prostration disproportionate to the physical signs.

The Prognosis in Acute Bronchitis.—In this form it lasts from a few days to several weeks and one may anticipate a prompt and complete recovery and freedom from relapse save in those persons exhausted by antecedent disease, badly nourished or suffering from emphysema, cardiovascular or renal disease.

Its conversion into a broncho-pneumonia is to be feared in patients representing the extremes of life.

RATIONALE OF ACUTE BRONCHITIS

Lumen of bronchi reduced.

Obviously, infection, irritants or foreign substances must induce varying degrees of congestion, together with edema of the bronchial mucosa in the severer cases, resulting in various degrees of stenosis, especially affecting the tubes of relatively small caliber, but not the terminal bronchioles.

Dryness followed by overexertion.

The surface of the inflamed mucous membrane is at first dry from the checking of normal secretion; later excessively moist from the usual reactive oversecretion characteristic of congested mucous (or serous) membranes.

Brief duration.

Desquamation of varying degree is present but pronounced round-celled infiltration of the deeper structures of the bronchial tissues is usually absent and prompt regeneration follows. Certain cases are primarily so severe or

Prolonged attacks.

meet such conditions of impaired resistance as furthers the establishment of a chronic process. The lung tissue is practically unaffected.

Fever, local discomfort.

Results.—Fever results if there be an acute infection, either primary or secondary, together with distress over the region of the inflamed and hypersenstitive trachea* and main bronchi, and racking and unproductive cough is followed by one productive and "softer," but no physical signs are present

Dry and moist râles.

save dry whistling or snoring râles in the first stage and both dry and moist mucous râles in the second.

The production and intensification of some of the smaller râles is favored by certain areas of dilatation of the lesser bronchi. The quality, pitch and intensity of the various râles will vary with the calibration of the tubes and the amount and character of exudate present at the site of their production.

* Certain ganglia at the tracheal bifurcation are chiefly responsible for the substernal distress so commonly present.

CHRONIC BRONCHITIS.—*This is essentially a secondary disease.* It may result from repeated acute attacks or long-continued exposure to irritating substances; is relatively common in connection with gout, the cardiovascular degenerations and circulatory inadequacies of advanced age; is an invariable accompaniment of asthma, incompensated mitral disease or emphysema, and in many instances is associated with chronic nephritis.

Seldom primary.

Common associations.

The diagnoses of "chronic bronchitis" and "winter cough" have diminished, as more exact methods have proven many to be cases of pulmonary tuberculosis, chronic cardiac or cardio-renal insufficiency, or bronchiectasis.

Diminished incidence.

The bronchitis may be dry, moist, or actually bronchorrheic, in which event more than a liter of sputum may be raised daily. In all persisting severe cases an emphysema results and the physical signs of the disease are in direct accord with this fact.

EOSINOPHILIC BRONCHITIS.—This condition differs in no respect from other forms of chronic bronchitis save in the obstinate severity of the cough, the special tendency to decided dyspnea and the almost exact likeness of the sputum to that of "bronchial asthma." This secretion is tenaciously mucilaginous, is streaked with yellow, is rich in eosinophile cells and often contains Curschmann's spirals and the Charcot-Leyden crystals. The eosinophiles of the blood may also be greatly increased.

A dubious entity.

OBLITERATIVE BRONCHIOLITIS.—This rare and curious condition may result from the inhalation of irritant gases as in the horrible "chlorine gassing" of the European war, but also occurs without assignable cause as a sequel to an apparent simple bronchitis. Acute emphysema, dyspnea, cyanosis and a failing heart with moderate fever may progress to a fatal issue, pursue an extremely chronic course or even terminate in recovery. In the typical case the lungs are filled with gray nodules which resemble miliary tubercles. These are found to respresent cross-sections of obliterated terminal bronchioles.

PUTRID BRONCHITIS.—This form is chronic and characterized by foul-smelling sputum, but hardly deserves a separate place because of its almost invariable association with bronchiectasis, gangrene, ruptured abscess, or empyema.

Physical Signs.—Unless, as is commonly the case, one finds superadded symptoms of emphysema or other primary conditions, the physical signs of this ailment are limited to sibilant, sonorous, mucous or bubbling râles with more or less profuse expectoration.

Chiefly auscultatory

FIBRINOUS BRONCHITIS.—In this ailment fibrinous casts are formed and expelled; these are often extensive and sometimes produce bronchial blocking and atelectasis of lung areas. Dyspnea and cough are much more urgent than in ordinary bronchitis and the attacks may be single, recurrent, acute or chronic. *It is a clinical curiosity.*

Fibrinous casts.

Urgent symptoms

SYPHILITIC BRONCHITIS.—It is necessary to remember that syphilis may and often does affect the bronchi and may produce any and every variety of bronchitis. In all cases of persistent intractible bronchial inflamma-

tion especially, one should remember this fact and endeavor to ascertain by direct or indirect diagnostic methods whether or not lues is present.

Hemoptysis occurring suddenly and without apparent cause or in association with what appears to be a simple bronchitis, acute or chronic, should always suggest the possibility of a syphilitic origin. This is an important diagnostic point very frequently overlooked. Such hemorrhages may occur in syphilitics in whom bronchitis or any signs of pulmonary disease are wholly absent and may be profuse and recurrent.

TRENCH GAS POISONING (CHLORINE "GASSING")

Probable agent.

A Horrible Agent of Warfare.—The most terrible of the agents of destruction used in the European War, is concentrated chlorine gas, introduced by the Germans as a means of trench warfare.

Peculiar adaptability.

This substance is perfectly adapted to its horrible purpose in that even when wind borne it hugs the ground, being more than twice the weight of air; is readily compressed and safely transportable in metal cylinders; and even in high dilution (1–10,000) exerts a most violent irritative and asphyxial action.

Respirators.

Means of Protection.—The use of proper respirators makes immune its users and indeed, to a large degree, those under attack; but the advantage lies always with the former who attack unexpectedly and only with a favoring wind and are fully and properly protected whereas the latter are always to a greater or less degree unprepared, even though supplied with the respirators, all too readily torn, mislaid or lost.

Early Effectiveness.—During the first days of its use this barbarous agent inflicted the most insufferable agonies upon the unprotected foe.

Morbid Changes Produced.—It produces an intense congestion of the mucous membrane of the trachea, larynx and larger and lesser bronchi, with a profuse flooding bronchorrheal discharge. In fatal cases the lungs are the seat of a congestion and edema so intense that they do not collapse when removed at autopsy "but appear like a solid cast of the thoracic cavity." On section the lesser bronchi are wholly unrecognizable and here and there local patches of emphysema bear testimony to *the "agonized violence" of the respiratory efforts* brought to bear upon the relatively few small parts of the lungs which escape the obliterative process and prevent the more merciful prompt strangulation.

Intense congestion and edema.

Heart.

General venous congestion is present in such cases and right heart dilatation is a prominent figure. Marked irritation of the gastric mucosa is described by various writers.

Agonizing.

Symptoms.—The symptoms vary in intensity with the severity of the individual case but are essentially those of *intense febrile suffocative bronchitis* associated with a peculiarly intense and persistent cyanosis, bronchorrhea, pain (epigastric, intrathoracic, and cephalic), stupor or mental confusion, conjunctivitis, salivation and vomiting.

In severe cases the painful gasping respiration, asphyxial facies of the

orthopneic patient racked by recurrent paroxysms of painful cough make a picture of suffering such as no pen may describe.

BRONCHIECTASIS

Definition.—*Bronchiectatic dilatation, single, multiple or universal, saccular or fusiform, regular or irregular, unilateral or bilateral,* is always secondary to other bronchial or pulmonary disease.* Usually multiple.

It ordinarily follows chronic inflammation of the bronchial wall attended by obstruction and accumulation of secretion and is oftentimes associated with pulmonary tuberculosis, chronic pleurisy with adhesions, chronic emphysema, aneurysmal pressure, or fibroid disease of the lung.

Though rarely caused by acute disease, it has been reported in pertussis, influenza† and broncho-pneumonia, and in its cylindrical form is probably a not uncommon sequence.

As usually encountered the sacculations are multiple, but cases of single cavity occur and in rare instances these are enormous.

In tuberculosis the apices are chiefly affected but in other forms the bases are oftenest involved.

Unilateral universal bronchiectasis is a rare condition, almost always congenital, which in the still rarer acquired form is a sequence of chronic interstitial pneumonia. A clinical curiosity.

Physical Signs.—*With respect to the bronchiectases themselves they are often wholly absent. When present, they are merely those of lung cavity, usually but by no means always, lacking the distinguishing qualities of a vomica occupying infiltrated tissue.* Often absent. Signs of cavity.

Hence the diagnosis of bronchiectasis usually rests upon the presence of causative factors, the peculiar paroxysm of cough, the characteristic fetid sputum and the X-ray findings, which are peculiarly satisfying in the saccular type. Typical sputum.

The more or less regular filling and emptying of the bronchiectatic cavity causes an instability of physical signs which is of diagnostic importance in rare instances.

In some cases daily fever occurs and the hectic physiognomy is present. Dyspnea is sometimes decided and cyanosis marked in the severe generalized forms. Clubbing of the terminal phalanges is not uncommon in such cases. Fever.

Cough and Sputum.—Once or twice daily, usually in the morning on arising, and bending forward or while turning over in bed, a violent paroxysm of coughing occurs attended by extraordinarily profuse expectoration not infrequently attended by vomiting.‡ Posture.

This is offensive in odor, gray or grayish brown and purulent, and upon being placed in a glass it will after a time separate into three layers. The Three layers.

* 60+ per cent. are bilateral and in some cases the lungs are literally honeycombed.

† Its occasional occurrence in the influenzal hemorrhagic pneumonitis of 1918 has been reported by several observers.

‡ Over a liter daily has been reported in the medical literature. The odor is sometimes less offensive than would be expected.

lowest is thick and granular; there is a thin mucoid intermediate layer, and the surface layer is brown and frothy (high air content).

The tiny yellowish white, bread-crumb like, foul-smelling Dittrich's plugs may or may not be present.

Posture and cough.

Changes of attitude may greatly influence the onset of cough, and to empty the bronchiectatic cavity some patients learn to assume an attitude that brings the head and upper thorax below the level of the rest of the body.

RATIONALE.—It is evident that a secreting bronchiectatic cavity must of necessity be at one time more or less completely full and at another empty.

Its stagnant contents must necessarily be continuously foul, full of micro-örganisms, capable of causing toxic absorption and hectic fever, of keeping up a persistent irritation or even ulceration, and of tending to further enlarge the cavity.

Varices may form and *hemoptysis* from this source occurs in about one-third of the cases.

Superficial vs. deep lesions.

If it be superficial in location, occurs as a complication or one of the end results of tuberculosis or fibroid phthisis, or is associated with marked reactive inflammation in the limiting tissues, one may elicit the physical signs of

Cavity with or without infiltration zone.

cavity with surrounding infiltration. Usually however, such sacs lack the boundary zone percussion dulness and peculiar auscultation signs of tuberculous cavities, and diffuse more widely such auscultatory signs as may be present.

As usually occurring, it will present *masked* cavity signs or none at all, unless near the surface, because of the absence of reënforcement of vibrations no less than the common presence of an obscurant emphysema.

Compression dulness.

If multiple large sacs exist, however, the lung tissue between them may be markedly infiltrated or compressed and highly conductive over a sharply limited field.

It follows that diagnosis must be based chiefly upon the character of the cough and sputum reënforced by the presence of potential etiologic factors

X-ray.

and more or less indefinite physical signs of cavity, often clearly demonstrated by the X-ray. (See under "Roentgenography.)

Prognosis.—The condition once established is permanent and slowly

Extremely chronic.

progressive, though usually extremely chronic, and is rarely relieved save by operative measures in selected cases.

Pleuritic adhesion, pleurisy with serous or purulent effusion, pulmonary gangrene or broncho-pneumonia may occur as complications and it is obvious that right heart decompensation may be induced.

Patients live long, usually for decades, and die of exhaustion or intercurrent disease.

In exceptional cases of superficial single cavity, surgery is effective but the surgical mortality in general is high and the results on the whole unsatisfactory.

Great variety.

FOREIGN BODIES IN THE BRONCHI.—Coins, whistles, nails, pins, tacks, buttons, jewelry, bullets, pebbles, false teeth, tooth crowns, bones,

Right side chiefly.

fruit stones, peas, beans and the like may be drawn into the glottis and usually fall promptly in the larger and more direct *right bronchus.*

Violent cough and air hunger or true stenotic dyspnea and cyanosis usually follow and are associated with widely varying degrees of pain. Immediate symptoms.

If the object is not expelled, death may result in a few minutes, but, more often, the attack subsides and secondary inflammation, often suppurative, ensues and results in localized septic broncho-pneumonia, abscess, or even gangrene. In some instances chronic pneumonia with or without bronchiectasis results and lasts for months or years, ending rarely in the extrusion of the foreign body. Sequelæ.

Diagnosis.—*This depends upon the anamnesis, violent paroxysmal cough, the roentgen-ray findings and the physical signs of localized inflammation often associated with airless areas representing atelectasis.* Usually simple.

EMPHYSEMA

VESICULAR EMPHYSEMA.—Definition.—*A condition which may be primary or secondary, general or local, temporary or permanent, acute or chronic, and is characterized by dilatation of the infundibular and alveolar walls of the lung with resulting atrophy of the latter in the chronic forms.* Dilated alveoli and infundibula. Alveolar atrophy.

The chronic type occurs chiefly in males over forty years of age, is frequently accompanied by a generalized arteriosclerosis and may be associated either with enlargement or shrinkage of the lungs, the former being the common form.

A localized emphysema may be temporary or permanent and is usually *vicarious and compensatory*, representing the effect of overaction associated with temporary or permanent loss of function on the part of the other lung or in portions of the same lung (cicatrices, tuberculous areas). Vicarious emphysema.

The best examples of the chronic compensatory form are afforded by the otherwise unaffected lung in cases of unilateral pulmonary tuberculosis or extensive pleural adhesion with its persisting unilateral impairment of pulmonary expansion and capacity.

The acute compensatory emphysema is beautifully shown in pneumonia, pneumothorax and unilateral pleural effusion and in either condition the vital respiratory balance is acquired with astonishing rapidity.*

Acute Bilateral Emphysema.—This occurs abruptly in asthmatic seizures and in certain cases of angina pectoris and rapidly subsides as the attack passes.

Interstitial Emphysema.—This condition is caused by the rupture of a superficial air vesicle which, during violent cough or straining, permits the escape of the air into the areolar tissues and produces (*a*) *interlobular or* (*b*) *subcutaneous emphysema*, or even (*c*) *pneumothorax* according to the site and degree of its extension. Ruptured septa.

Subcutaneous emphysema is especially common in connection with traumatism or ulceration involving the lung structure and as a clinical rarity in interstitial emphysema. *It is at once detected by the exquisite, crackling, palpable, crepitation produced by the pressure of the fingers upon the distended skin.* Subcutaneous "crackling."

* In one of the rarest of clinical accidents, recently observed by the author, namely, a sudden and complete spontaneous pneumothorax in a healthy adult, a few days sufficed to render him able to get about in comfort.

VESICULAR HYPERTROPHIC EMPHYSEMA

The common form.

Cause and Effects.—*In this, the common form of emphysema, the air cells are overdistended, their walls weakened, atrophied, and prone to rupture, with resulting coalescence of the dilated cells and obliteration of the lung capillaries. As a result, the lung is increased in size, its alveoli become fused in certain areas and its circulation and expansile power are greatly and progressively diminished. The impeded circulation causes ultimate hypertrophy and terminal failure of the right heart and greatly increases the tendency to chronic bronchitis so generally observed as a persistent or frequently recurring ailment in this condition.*

Bronchitis, dyspnea and cyanosis.

SYMPTOMS.—*In established cases of hypertrophic emphysema the symptoms are primarily those of a chronic bronchitis plus slight or intense cyanosis, dyspnea, either persistent or developed only by exertion, and certain well-defined and easily recognized physical signs definitely expressing the pathologic changes.*

Slight emphysema the common type.

*A considerable degree of emphysema may exist without evident dyspnea, marked cyanosis or persistent signs of bronchitis, and as a matter of fact, the great bulk of the cases observed are of this type, yet it occasionally exhibits the most profound cyanosis seen in adult walking patients.**

Diminuendo cough.

Cough.—This may be variable in degree as in persistence and may be dry, moderately productive, or accompanied by profuse expectoration. In severe cases it is somewhat characteristic, the recurring coughs of each individual paroxysm being greatly prolonged *diminuendo*.

Heart.—Symptoms of failing right heart may be long postponed but the strain to which its chambers are subjected must ever be remembered.

FIG. 134.—Emphysema. Attitude frequently assumed in marked cases, concealing barrel form of chest.

PHYSICAL SIGNS.—Inspection.—*The shape of the chest is that of forced held inspiration (barrel chest). The anteroposterior diameter is increased and the epigastric angle wide. True expansion is largely replaced by a vertical lifting movement, and constant overuse makes prominent the accessory muscles of respiration, particularly the sterno-mastoids. Inspiratory retraction of the lower interspaces with excessive supraclavicular bulging is common in advanced cases. The ribs are rigid, the costal cartilages prematurely ossified, the interspaces broadened and the expiratory descent of the thorax is prolonged. The apex beat of the heart is ordinarly invisible, the neck appears short though often thin, and a compensatory curve of the spinal column may cause the chest to appear flattened and the patient round-shouldered.*

Barrel-chest.

Wide epigastric angle.

Chest movement.

Expiratory lagging.

Apex beat obscured.

Spinal curve.

If such a patient lies upon the back on a table, the chest will at once assume its typical emphysematous outline. *Epigastric pulsation* is common

* The author has carried throughout his professional life the vivid clinical picture presented by two walking cases showing extreme cyanosis, which were shown simultaneously to students by the great London clinician in whose clinic he was working; one a toddling child with congenital heart disease, the other an aged man with emphysema.

and *Litten's sign* shows an abnormally slight respiratory range of diaphragmatic excursion confirmed by the fluoroscope and by careful comparative percussion, which procedures show also the abnormal lung rarefaction and *persisting low position of the diaphragm*, characteristic of this ailment. Litten's sign. Fluoroscopic appearance.

FIG. 135.—Emphysema (barrel chest). Same chest, patient in dorsal recumbent posture.

In severe cases of long standing, as in all cases of chronic right heart over-strain and insufficiency, one is likely to encounter more or less clubbing of the finger tips and incurvation of the nails. Drumstick fingers.

Palpation.—*Palpation confirms inspection and reveals an enfeebled vocal fremitus; a weak or wholly obscured apex beat, and some downward displacement of the liver.* Feeble vocal fremitus.

The author has seldom or never seen a well-marked case lacking some degree of arteriosclerosis.

Percussion.—*Percussion develops hyperresonance; low position of the lung border, impaired movement of the lung bases and apices; downward displacement of the liver dulness and diminution or entire absence of the superficial cardiac area.** Low lung borders.

Auscultation.—*The breath sounds are enfeebled; the expiration prolonged though usually low-pitched, and bronchial râles are often present at the base. The heart sounds are muffled, especially in the mitral area, and the pulmonary second sound, though enfeebled and distant may or may not show relative accentuation when compared with the aortic tone which is often exaggerated because of co-existing arteriosclerosis.* In old and severe cases tricuspid regurgitation may be present. Enfeebled sounds.

Secondary Symptoms.—The effect of long-standing severe emphysema and chronic or recurrent bronchitis is to overburden the pulmonary circulation and right heart; hence if incompensation follows, one may expect to find the symptoms of a cardiac lesion. As a matter of fact, such severe terminal symptoms are rarely encountered, because of the extreme chronicity of the primary ailment, and the interposition of fatal intercurrent diseases, though minor degrees of secondary congestion affecting the organs tributary to the heart and great veins are extremely frequent. Cardiac strain.

RATIONALE OF EMPHYSEMA

In hypertrophic vesicular emphysema we *deal with dilated air cells; an overvoluminous, inelastic, chronically overinflated lung; atrophied vesicular walls, an impaired intrinsic circulation and a more or less rigid thorax.*

The elasticity of the lungs is one of the chief factors in normal expiration and lack of post-inspiratory pulmonary retraction means a diminution of the range of expansion and hence of the inspiratory filling. Diminished expansion.

* In certain cases marked dulness may be present at an apex and in others the general percussion note is not clearly hyperresonant.

Lessened exchanges.

It is obvious, therefore, that the inelastic, voluminous lung and rigid non-expansile chest wall, so characteristic of emphysema, not only will embarrass the movements of respiration, but also diminish the air exchanges of the lung.

Capillary obliteration.

Overdistention of this organ is persistent in established chronic emphysema and oxidation is further diminished by the wiping out of a variable but considerable proportion of the capillary network of the pulmonary alveoli in consequence of the overstretching and atrophy of their walls.

This interference with the interchanges between air and blood in the lungs results in further diminished oxidation and a carbon dioxide retention which so overstimulates the respiratory center in the medulla as to impel deeper and perhaps more frequent respirations, a tendency increased by any co-existent bronchitis.

Dyspnea and cyanosis.

It is thus quite evident that both cyanosis and dyspnea in varying degree must inevitably accompany any established hypertrophic emphysema. It is also evident that a constant obstruction to the "lesser circulation" exists

Bronchitis.

Right heart.

which must of necessity invite bronchitis, greatly increase the work of the right ventricle and ultimately lead to hypertrophy and perhaps dilatation of that heart chamber.

We would expect primarily, therefore, an accentuation of the pulmonary second sound because of the emphatic closure of the valve as a result of the increased primary thrust and the sharp recoil of the right ventricular blood column against the resistance in the pulmonary artery resulting from the obstructed flow in capillary areas of the emphysematous lungs. To this is added sometimes a considerable sclerosis of the pulmonary artery itself.

Right heart decompensation.

If we do not hear such accentuation we, allowing, of course, for the obscuration of the sound by the overdistended lungs, assume (*a*) that the ventricle is greatly weakened or, (*b*) that the tricuspid valve has yielded to the strain and cast its own burden and that of the ventricle backward upon the great veins and systemic circulation.

If, as is usually the case, corroborative evidences of such extreme cardiac decompensation are absent, we remember that the voluminous, air-stretched, rarefied lungs push forward and so bury the heart and valvular areas as to muffle and obscure all heart sounds or murmurs, wholly obliterate superficial cardiac dulness and even depress the diaphragm and the sheltered viscera to the extent of several inches.

Any relative accentuation of the pulmonary second sound as compared with the aortic second is of decided importance, though as previously stated, arteriosclerosis may so intensify the latter as to cause misinterpretation unless this possibility is remembered. In such cases equality between the two sounds would mean decided accentuation of the pulmonary second. Epigastric pulsation is a common sign in this disease because of the right heart enlargement.

Wide epigastric angle.

The physical signs are equally obvious. The persistently overinflated bulky lung progressively demands more space and this can be met only by a widening of the circumference which results in a shortened vertical measure-

ment, a consequent broadening of the intercostal spaces and an "opening up" of the epigastric angle. The chronically overinflated lung must yield a *hyperresonant note* because of its excessive air content. Hyper-resonance.

Expiration is prolonged as is indicated by the "lagging" descent of the thorax and the known elasticity of the pulmonary tissue. Prolonged expiration.

The breath sounds are feeble and muffled unless severe dyspnea or existing bronchitis intensifies or harshens them for the same reason and because of the reduced volume of air passing to and fro. Muffled sounds.

Vocal fremitus is of course enfeebled by the poor conduction of bulky but rarefied lung and perhaps also damped to some degree by the excessive pressure sometimes exerted upon the chest wall. Enfeebled fremitus.

In no disease are the subjective symptoms and physical signs more absolutely the logical and inevitable result of the pathological conditions.

Chronic hypertrophic emphysema is almost unbelievably slow in its advance and the cases of the extreme typical form are few in comparison to those showing an imperfect syndrome. Most cases mild.

ATROPHIC OR SENILE EMPHYSEMA.—The small contracted chest, wasted muscles, increased costal obliquity, depressed sternum and clavicles, narrowed lower interspaces and bowed shoulders are striking features.

The expansion of the rigid chest is trifling and inspiratory retraction, both supraclavicular and costal, may be evident.

The lungs are small but present an extraordinary appearance post-mortem, being made up of large vesicles representing the result of degeneration and atrophy of the alveolar walls and their fusion into large air sacs.

SPASMODIC ASTHMA

Definition.—*This is an ailment of ill-defined causation characterized by more or less severe transitory paroxysms of dyspnea, dominantly expiratory, and acute emphysema; tending to recurrence and chronicity, and associated with a tendency to establish chronic bronchitis and emphysema.*

Etiology.—*Spasmodic asthma* may be an isolated, apparently causeless clinical syndrome, but is often closely associated with, or one of the manifestations of hay fever, a weakened right heart, polypi, adenoids, supersensitive or hypertrophied turbinate bodies, intestinal auto-intoxication or irritation, malnutrition, hysteria, psychasthenia or epilepsy. Associated or related conditions.

The question of anaphylaxis has assumed great importance of late in this connection and sensitization to certain proteins has been demonstrated repeatedly and may be tested easily by the intradermic injection of minute quantities of protein. The author has observed several striking examples. Anaphylaxis.

The onset of an attack may be precipitated by any sensation of limitation of respiratory movement or even by the remembrance of previous seizures. Such sensations may not proceed directly from the respiratory tract. Flatulent distention, straining at stool, or even a tight belt or corset, may be sufficient.

Eppinger and Hess have described as "vagotonia" a condition characterized by heightened response of the vagus to pilocarpin injections. Vagotonia.

Broncho-tetany.

A similar condition in spasmophilic children is termed "broncho-tetany" by Lederer.

Sex and age.

There is no doubt that in asthmatic adults and children alike, phenomena of the sort described by Eppinger and Hess may occur, but it is by no means true of all, nor does it serve to clear up the *fundamental* factors in etiology.

Males are chiefly affected, the ratio being as two to one, and 80 per cent. of all cases occur in persons under forty and one-third of these in children under the age of ten.

Heredity.—*In some instances it appears to be distinctly an hereditary alternative in the group of neuroses.*

No *one* of them causative.

Theories as to the Pathology of the Attack.—*Five of those which have been advanced in the past may be mentioned:*

1. **Bronchial spasm,** *i.e.*, spasmodic contraction of the muscles controlling bronchial caliber.

All operative in some degree.

2. **Hyperemia and swelling of the mucosa.**
3. **Spasm of the diaphragm.**
4. **Spasmodic contraction of other inspiratory muscles.**
5. **Exudative bronchiolitis.**

In part mere consequences.

Combining certain of these we would have bronchial spasm (1), *congestion and the attending exudation* (2 *and* 5), *as undoubtedly representing the most prominent* **events,** *while it is probable that all five are operative in varying degrees.*

At present no single etiologic factor can be held accountable for spasmodic asthma and it will be noticed that these so-called "causative factors" are after all, merely the phenomena attending an attack.

Probable cause.

It is more than probable that a more exact knowledge of allergic and anaphylactic processes and cryptogenic infections will yield the solution.

Exciting Factors.—Careful inquiry should be made concerning the exciting causes and the number and variety of the attacks. Such patients become close observers and often describe with exactness the clinical phenomena and apparent determining factors. Among these are *odors* of all sorts; those of drugs, perfumes, talcum powders, of certain flowers, of hay, of horses or dogs or cats, fish, game or coffee; *irritants* such as dust or fog and *heat or cold*, whether local or general. Old feather beds and pillows have in more than one instance proved causative factors and *emotions* such as pleasure, pain, joy and sorrow, sexual stimuli, grief and worry are alike potent in excitation. In certain instances *heavy meals at night* and certain articles of food or drink may cause a seizure and in some cases it is distinctly related to the menstrual epochs. Chronic poisoning, such as that from gout or lead, intestinal worms, and various occupations exposing their followers to dust, animal emanations, pollens and the like, or alternations of heat or cold may invite attacks. The sensation of hampered respiratory movement or mere remembrance of it as an exciting cause has already been mentioned.

Bizarre factors.

Food sensitization.

It must be remembered also that bacterial proteins absorbed from

points of focal activity and certain sera, such as diphtheria antitoxin, may be potent factors in excitation.

Inherited sensitiveness to certain food substances, pollens, and the like, is a factor of considerable importance.

Food sensitization is especially common in cases which have originated during childhood.

Method of Testing for Protein Sensitization (Walker).—"A number of small cuts, each about $\frac{1}{8}$ of an inch long, are made on the flexor surfaces of the forearm by means of a sharp scalpel, not deep enough to draw blood, although they do penetrate the skin. On each cut is placed a protein and to it is added a drop of $\frac{1}{10}$ normal hydroxide solution to dissolve the protein and to permit of its rapid absorption. At the end of a half hour, the proteins are washed off and the reactions are noted, always comparing the inoculated cuts with normal controls, on which no protein was placed. A positive reaction consists of a raised white elevation or urticarial wheal surrounding the cut. The smallest reaction to be called positive must measure 0.5 cm. in diameter and any smaller reactions are called doubtful. Negative skin tests with protein rule out those proteins as a cause of asthma and all proteins which give a positive skin test should be suspected as a cause of asthma. In the case of bacteria, however, the skin test has to do only with the protein element, so that even though bacteria give a negative test, they may still be a cause of asthma through their infectious nature, and the patient need not be sensitized to bacterial protein.

"*Proteins Recommended for Routine Tests.*—For practical purposes, however, such an outlay of proteins is not necessary, since the majority of patients are not exposed to such an extensive list of proteins and consequently they are not sensitive to such an unlimited number. The following kinds of proteins should be used as a routine: horsehair or dandruff; cat hair; feathers; the pollens of timothy, redtop and ragweed; staphylococcus pyogenes aureus and albus; streptococcus hemolysans and viridans; pneumococcus types I and IV and diphtheroid bacillus; the common foods such as egg, milk, cereals, meats, chicken, potato, and any other that the patient is accustomed to eat frequently. Occasionally the patient's history, occupation, or surroundings may lead one to suspect some other common or even unusual type of protein.

"An understanding of the seasons during which the different types of plants pollinate and the usual variety of plant pollen which causes asthma is most helpful. There are three definite pollen seasons in New England. The first occurs in April and May when the various trees pollinate; among the first to pollinate during this season are the willow, birch and maple, and the last one to pollinate is the pine, which occurs the last day or two in May. Although the pollen of any of these trees may cause asthma, we have had only one case caused by willow, one by birch, and one by pine pollen. Since the length of time which each tree requires to complete pollination is no longer than two weeks, and in the case of the pine tree only one day, it is quite unnecessary to treat these patients for such a short exposure to the pollen.

"The second pollen season, and a more important one, occurs during June and July. The principal plants which pollinate at this time are the rose, June grass, orchard grass, redtop, and timothy; the latter two are also grasses and are familiarly known as hay. We have not had a case of asthma caused by the rose, although there are such on record; neither have we found orchard grass to be a cause; these two plants surely are not frequently a cause of asthma. June grass is so similar botanically to redtop that it may be disregarded in tests. The important plants in this group are redtop, and the timothy grasses, and of these two timothy is the chief cause of bronchial asthma at this season. Practically every patient who was sensitive to timothy was also somewhat sensitive to redtop, but usually less so, and redtop never gave a stronger reaction than timothy.

"The third pollen season, and by far the most important, occurs during August and September, and the important pollens at this time are those of ragweed, goldenrod and daisy. The daisy really begins to pollinate in July and finishes in early September; goldenrod pollinates during August and September and ragweed usually begins to pollinate about the middle of August and ceases with the first heavy frost, which, in New England for example, usually occurs early in October. Ragweed is practically always the cause of pollen asthma at this season, and in only one case have we found the daisy the cause, and in one other was goldenrod the cause. These three plants belong to the same botanical family, although they are not intimately related to each other, and consequently every patient that reacts positively to one need not react to all.

"Stated briefly, 18 per cent. of the pollen asthmas were due to timothy and redtop, 72 per cent. were due to ragweed, 9 per cent. were due to both and had continuous asthma from June to October, and the remaining 1 per cent. was due to miscellaneous pollens. Frequently, hay fever patients have a very slight attack of asthma at some time during their hay fever, often hay fever symptoms end with a day or two of asthma, and still others are more or less choked up at times during their hay fever. Asthma, caused by pollens, may continue after the termination of the pollen season. The continuation of asthma in these cases is due to secondary bacterial infection, causing bronchitis in a patient whose resistance, either local or general, has been lowered because of prolonged severe pollen asthma."

Cardiac dyspnea vs. Cardiac asthma.

Spurious Spasmodic Asthma.—A Word of Warning.—*The utmost care should be observed to avoid the common and disastrous error of confounding "renal," "aneurysmal" and "cardiac" asthmas with those of the ordinary spasmodic type.*

Exact simulation.

Renal Asthma.—*The paroxysms of "renal asthma" are absolutely like those of true asthma* and the cause can be determined only by a careful examination and quantitative analysis of the twenty-four hours' urine, and an application of the sphygmomanometer and the tests of renal permeability, for the attacks are usually coincident with low urea excretion and *high arterial tension.*

Aneurysmal Asthma.—*In the absence of roentgen-ray findings, the pseudo-*

asthmatic paroxysms produced by aneurysm of the aortic arch may at times deceive the elect, but are fortunately rare save in connection with frank physical signs. — May fool the elect.

Cardiac Asthma.—The ordinary cardiac dyspnea is either persistent or distinctly dependent upon exertion and is inspiratory in type or a mere air hunger.

Nevertheless, typical asthmatic attacks occur in association with, and apparently as one of the results of, a weakened heart and especially in myocardial cases with unstable, delicately balanced compensation. They are associated often but not always with decided coronary sclerosis and may substitute or alternate with attacks of major angina pectoris in rare instances. — Frequent association with coronary sclerosis.

In established asthma the heart strength is an extremely important factor both in treatment and prognosis. — Watch the heart.

Time of Attack in Spasmodic Asthma.—Like croup, angina pectoris major and nocturnal epilepsy, attacks of true asthma occur usually at or after midnight and commonly, in the early morning hours. The same is true of the *spasmodic cardiac asthma* which seems so closely allied to angina pectoris of the severer type.

Auræ.—Like epilepsy, attacks of asthma may be preceded by premonitory symptoms recognized by the patient. Among these are mental depression or exaltation, flatulence, yawning, sneezing, itching, headache and polyuria. Ordinarily, however, the attacks occur without any such warning. — Usually absent.

SYMPTOMS.—The onset *may be gradual, but is usually abrupt. There is a* **sense of constriction** *of the throat or chest and pronounced* **dyspnea** *with labored, stridulous and prolonged respiration affecting chiefly the expiratory phase though marked in both. The agonized patient brings every muscle to bear but in spite of frenzied effort the rate of respiration is usually below normal.* — A pitiable picture.

A short, dry, feeble cough *increases the patient's discomfort, and an acute bilateral emphysema enlarges the girth of the lower chest so that the female patient, if attacked when dressed, is obliged to loosen the clothing.* — Acute emphysema.

The eyes are blood-shot and staring, the voice gasping and weak, the face pallid, cyanotic, drawn and agonized. — Facies.

The mind is absolutely clear, the patient usually apprehensive and fearful. — Mental state.

If in bed, orthopnea is extreme, the knees are drawn up, the shoulders advanced, the back rounded, and the head is on the hands, or, sometimes, one will be found leaning over a rigidly held chair or bed-post, or, thrown back, with raised arms grasping some support above that will give added purchase to the accessory muscles of inspiration. In many cases patients rush to an open window, regardless of extremes of temperature. — Attitudes assumed.

Cough.—This is ordinarily feeble and unproductive until the end of an attack, when there may be a thick, viscid sputum or, more rarely, free expectoration. In cases of pure asthma uncomplicated by bronchitis one may find the pearly globules of the size of a hemp seed which contain *Curschman's spirals* and Charcot-Leyden crystals. — Spirals and crystals.

Pulse.—The pulse may be markedly weak and accelerated. During any — Heart.

severe attack the right chambers of the heart are dilated, yet death during a paroxysm is most unusual *in true spasmodic asthma.*

Recurrence.

Duration of the Paroxysm.—Attacks usually end gradually, less often abruptly, after two or three hours, but may be shorter or much longer; usually recur with increasing severity on successive days, and are followed by a variable period of immunity.

EXAMINATION OF THE CHEST.—Inspection shows prolonged expiration, an increased basal girth, urgent dyspnea, a defective excursion, an inactive low diaphragm, and rigid, abdominal muscles with which the patient is seeking to reinforce the thoracic and cervical groups of muscles.

Palpation.—The râles are easily felt and vocal resonance and fremitus alike are enfeebled.

Percussion and Auscultation.—The signs are those described under "Emphysema" with bronchitis and stridor superadded.

RATIONALE OF SPASMODIC ASTHMA

Dominant Factors.—*One may reasonably assume:*

Apparent sensitization.

1. *An extreme susceptibility on the part of the patient to certain substances often specific for the individual.*

Nervous instability.

2. *A lability of the nervous system especially marked in the psychic sphere, and in many instances akin perhaps to that encountered in the spasmophilic diathesis of children.*

Swollen mucosa.

3. *Transient hyperemia and swelling of the mucosa, with a variable amount of exudation, perhaps involving even the bronchioles.*

Bronchial spasm.

4. *A decided spasmodic tonic contraction of the muscles controlling bronchial caliber and also of the thoracic, and, to a less degree, the accessory thoracic, respiratory muscles.*

Abrupt onset.

5. *That all these factors usually become operative very suddenly.*

Dyspnea.

Symptomatic Results.—Extreme dyspnea is inevitable and the breathing is both slow and noisy because of the mechanical difficulties at once established. The spasmodically stenosed bronchi obstruct inspiration, *trap* the air and yet more strikingly block expiration. The lungs progressively and quickly expand up to and beyond the normal forced inspiration boundaries thus still further diminishing the respiratory radius and air movement, and producing acute emphysema with excessive dyspnea predominatingly of the expiratory type.

Inspiratory dyspnea.

Predominance of expiratory dyspnea.

Cumulative factors.

The extreme overdistention of the lung, impotent diaphragm, loss of use of the spasmodically contracted respiratory musculature of the thoracic wall, the imperfect aëration of the blood, consequent unsatisfied oxygen demand, CO_2 overload and overstimulation of the respiratory center in the medulla, all combine to produce a condition of acute and pitiable suffering, and oftentimes a sensation of impending death.*

A pitiable condition.

Physical Signs.—A combination of emphysema and acute bronchitis. It is evident that the physical signs must be those of acute emphysema com-

* When this last symptom is excessively marked one must always consider the possibility of cardiac or cardio-renal asthma or a dangerously dilated right heart.

bined with acute bronchitis in the dry stage, viz., a hyperresonant note, *extreme* prolongation of expiration, diminished fremitus, low-lying, widely expanded and almost immobile lung borders, loud sibilant râles, a widened thoracic circumference, highly deficient expansion and a broadened epigastric angle.

Cyanosis must inevitably be marked and the right heart is under great strain. With the subsidence of the attack moist râles may appear and a thick viscid sputum or more rarely, free expectoration, be present.

Effective coughing or loud talking is manifestly impossible at the height of the attack.

Chronic but shortens life.

Prognosis.—*The attacks are recurrent, the condition itself chronic and long endured, but on the average there is, nevertheless, marked shortening of life expectancy as is proven by life insurance statistics.*

Age important.

Asthma occurring in childhood usually depends upon a removable cause and is often curable. Those cases occurring between the ages of ten and twenty are sometimes cured, those of the intermediate period are rarely cured, and those in patients above sixty years of age, it has been said, are never cured. The author's experience would indicate that at no age is the last statement absolutely true if all contributive factors are sought and the full resources of medicine and surgery drawn upon.

Rest and stimulation for the tired heart, proper attention to the accessory sinuses and naso-pharynx, removal of obscure septic foci, and improved nutrition for the individual, may accomplish much even in some apparently hopeless cases, *but the complete cure of secondary conditions of long standing* is never possible in the adult.

PLEURISY (*Pleuritis*)

Definition.—*An inflammation of the pleura, acute or chronic, associated with an exudate which may be fibrinous, serous, bloody or purulent.*

Important relations.

Applied Anatomy.—Each pleura completely invests the lung, dips between the lobes and is reflected upon the chest wall so as to form a closed potential cavity. Normally the parietal and visceral pleural layers are in close apposition and move freely over one another, lubricated by the normal lymph.

*The pleural cavity is actually a great lymph space structurally continuous with the diaphragm, pericardium, cervical fascia, chest wall and lung.**

Genesis of friction sound

When inflamed, the pleura becomes dry, exfoliated and harsh and the attrition of apposed surfaces gives rise to pain, cough, and the characteristic physical sign of dry pleurisy, viz., an audible, often palpable, friction sound.

Terminations

The course of the inflammation is variable and may terminate as follows, viz.:

Varieties.

(*a*) *A slight attack of pleurisy with a small fibrinous exudate may terminate promptly, without permanent damage to the pleura.*

(*b*) *The fibrinous exudate may become organized and form pleural adhesions of variable extent and density.*

* The pleural folds or sinuses and Traube's space are discussed elsewhere.

Exudates.

(*c*) *A serous exudate may result, relieve the pain by separating the pleural layers and in its turn be reabsorbed or remain and become converted into pus* (*empyema*).

The character of the exudate varies greatly with the cause of the pleurisy and the nature of the associated microörganisms.

Hemothorax.—*Bloody fluid in most instances indicates malignant growths of the lung and pleura, though the same appearance is presented in certain frankly tuberculous cases and in some rare instances of purpura, hemophilia and scurvy.*

Other causes of hemothorax are rupture of an aortic aneurysm, pulmonary abscess, and, of course, direct injury or penetrating wounds.

Secondary or primary.

Purulent exudate is usually the result of neglected effusion, but is often primary, particularly in cases associated with the lobar pneumonia of children and in streptococcus and staphylococcus infections.*

Pressure.—Bearing in mind the important structures contained in the mediastinum, one readily understands the importance of the pressure symptoms of large unilateral pleural effusions. The lung of the affected side is compressed from below upward and toward its root *pari passu* with the increase of exudate and in long-continued cases may remain permanently crippled and bound down by adhesions after resorption of the effusion. The heart is pushed toward the unaffected side unless the mediastinum is firmly anchored by adhesions.

Effect upon lung and heart.

Empyema.

ETIOLOGY.—The pneumococcus, staphylococcus, or streptococcus, may be found alone or in combination in cases of purulent pleurisy, and apparently sterile purulent exudates (10 to 15 per cent. of the total) are for the most part tuberculous in origin. To these may be added, as rarer causative agents or associated organisms, the influenza, typhoid, and colon bacillus.

Serous exudates.

Bacteria other than the tubercle bacillus are found in from 15 to 20 per cent. of the serous exudates, pneumococci and staphylococci predominating.

A complication of many diseases.

Disease Associations.—Pleurisy may appear as a complication of various diseases. First in importance are tuberculosis, lobar pneumonia and pericarditis, but peritonitis, influenza, acute rheumatism and chronic nephritis are frequent. It may occur in the exanthemata, in typhoid fever and the acute infectious diseases generally, as well as in scorbutus, gout, chronic diseases of the liver and gallbladder, and in cancer. Traumatism of the chest with or without perforation may cause it.

Complicating Pneumonia.—Pleurisy is almost invariably present in lobar pneumonia, but in adults is seldom then associated with large or even demon-

Note.—**Transudate vs. Exudate.**—Fluid in the pleural cavity may be non-inflammatory in origin.

Rivalta's Test.—Add 2 drops of glacial acetic acid to 200 c.c. of distilled water. A drop of the suspected punctate is then dropped upon the surface of the acetic acid solution.

If it is exudate a gray nimbus indicates the course of the drop as it falls to the bottom. If it be a transudate, only a laggard haziness appears. The nature of the protein precipitate is unknown.

* Pneumococcus empyema is said to offer the best prognosis.

strable, serous effusions. The greater number of so-called simple pleurisies doubtless represent a localized, but as regards the pleura, usually self-limited, tuberculous infection, and the statistics of foreign observers show a percentage of tuberculosis pleurisies ranging from 25 to 90 per cent.* in the different reports. Few autopsies are made which do not reveal evidence of past pleurisies whatever the actual cause of death.

High percentage of tuberculous pleurisies.

SYMPTOMS OF ACUTE FIBRINOUS PLEURISY.—(1) *Fever*, usually moderate in degree and lasting but a few days.

Fever, pain, cough and friction.

(2) *Pain*, most commonly felt in the axillary or inframammary region, yet sometimes referred to the terminations of the intercostals over the abdomen or even to the sound side. It may be fixed, stabbing or shooting in character, and is increased by deep breathing and cough.

Referred pain.

The fact that abdominal pain may be pleuritic in origin should never be forgotten for many important surgical conditions may be accurately simulated even to an apparent defensive rigidity and localized superficial tenderness.

3. *Cough;* this is harsh, unproductive, painful and suppressed.

4. *Friction:* Auscultation yields a shuffling, creaking, squeaking, or even a crackling sound, simulating at times true crepitation or even rhonchi, but persisting after cough, superficial in character and usually occurring early in the inspiratory phase of a respiration. Friction is often palpable as well as audible.

Characteristics.

Physical Signs.—*Inspection reveals shallow catchy breathing, and comparative immobility of the affected side due solely to pain which causes both voluntary and reflex inhibition of chest movement, and suppressed cough.*

"Catchy" breathing.

The patient's decubitus is variable, oftentimes he lies upon the affected side as if to limit its movement during the stage of congestion and primary inflammation or give greater latitude to the expansion of the sound side if effusion follows.

Decubitus.

PLEURISY WITH EFFUSION

The first effect of effusion is to separate progressively the inflamed surfaces, relieve pain and diminish the area of friction sounds.

Sudden large effusions are unusual but may constitute a source of considerable danger owing to the resultant abrupt heart dislocation and lung compression.

Precipitate effusions.

Borders and Mobility.—The fluid gravitates primarily to the most dependent portions in about one-fourth of all cases and in most instances fills ultimately the inferior thoracic cavity. If not encapsulated or limited by adhesions it may slightly or, more rarely, markedly, change its level in accord with the position of the patient and the amount of effusion. The small effusions are sometimes freely movable but large ones of inflammatory origin seldom show any gross change in the line of percussion dulness on change of posture.

Variability of shifting dulness.

Small vs. large effusions.

* These are based upon the injection of large amounts of exudate into susceptible animals. Of public service cases nearly one-half develop a definite pulmonary tuberculosis within five years. In private, well-to-do patients the percentage is much smaller.

The Ellis curve.

Owing to the conformation of the chest and the peculiarities of internal pressure, the upper line of pleural effusion often describes a parabolic curve when the patient is sitting or erect. This reaches its maximum height in the mid-axillary or posterior axillary line. If the fluid reaches the middle of the second rib, the curve disappears. The lateral posture usually assumed by the patient when attacked and while the effusion is forming doubtless is

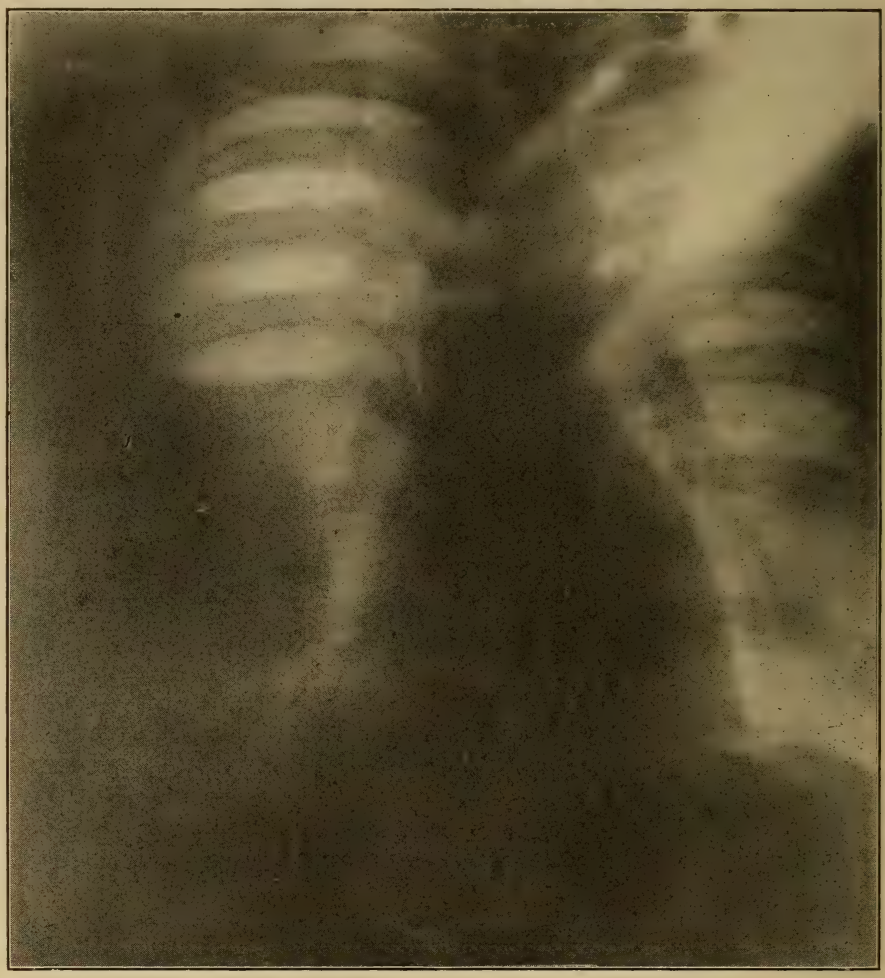


FIG. 136.—Encapsulated serous pleural effusion. Right pleura.

a factor in determining the line of the upper level of flatness. When he is in dorsal recumbency the level is highest near the spine.

In moderate effusions this line may resemble somewhat an attenuated letter "S" lying on its side ("Ellis' line") and, near the spine, in the triangular niche formed by the upward and outward ascent of the fluid level and the vertical line of the spinal column sufficient, resonance may be elicited by percussion after cough and deep inspiration, to contrast with the flatness

below the level of the line of dulness. Both the curved upper border and the angle of resonance ("Garland's angle"), *when present*, aid in differentiating pleural effusion from massive pneumonia. Garland's angle.

PHYSICAL SIGNS.—Inspection.—*There is relative immobility and loss of expansion of the affected side, shallow or flush interspaces,* increased dyspnea,*

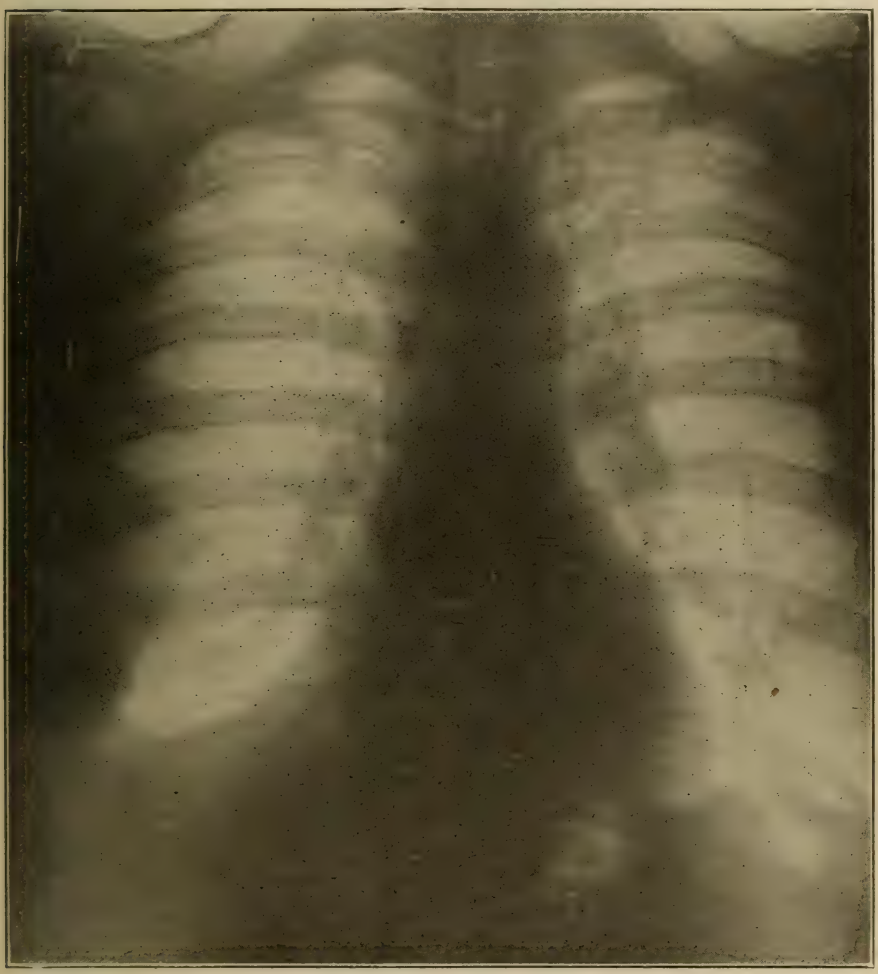


FIG. 137.—Encapsulated serous pleural effusion. Right pleura after removal of 800 c.c of serum (see Fig. 135).

with cyanosis or even in extreme cases lividity, and a displacement of the heart to the opposite side which may carry the *visible cardiac impulse* to the axilla on the left side or, on the right, nearly to the mid-clavicular line, the beat being due in the latter instance to the right ventricle. Occasionally in moderate left-sided effusion no cardiac impulse is visible. Displaced heart.

* The "bulging interspaces" are a clinical curiosity in simple acute exudative pleurisy.

Lessened fremitus.

Palpation.—*Palpation confirms inspection and reveals absent or diminished vocal fremitus unless a fibrous adhesion band crossing through the fluid conducts sound from the compressed lung and bronchi, or a dislocated bronchus lies in contact with the sternum and conveys the vibration.*

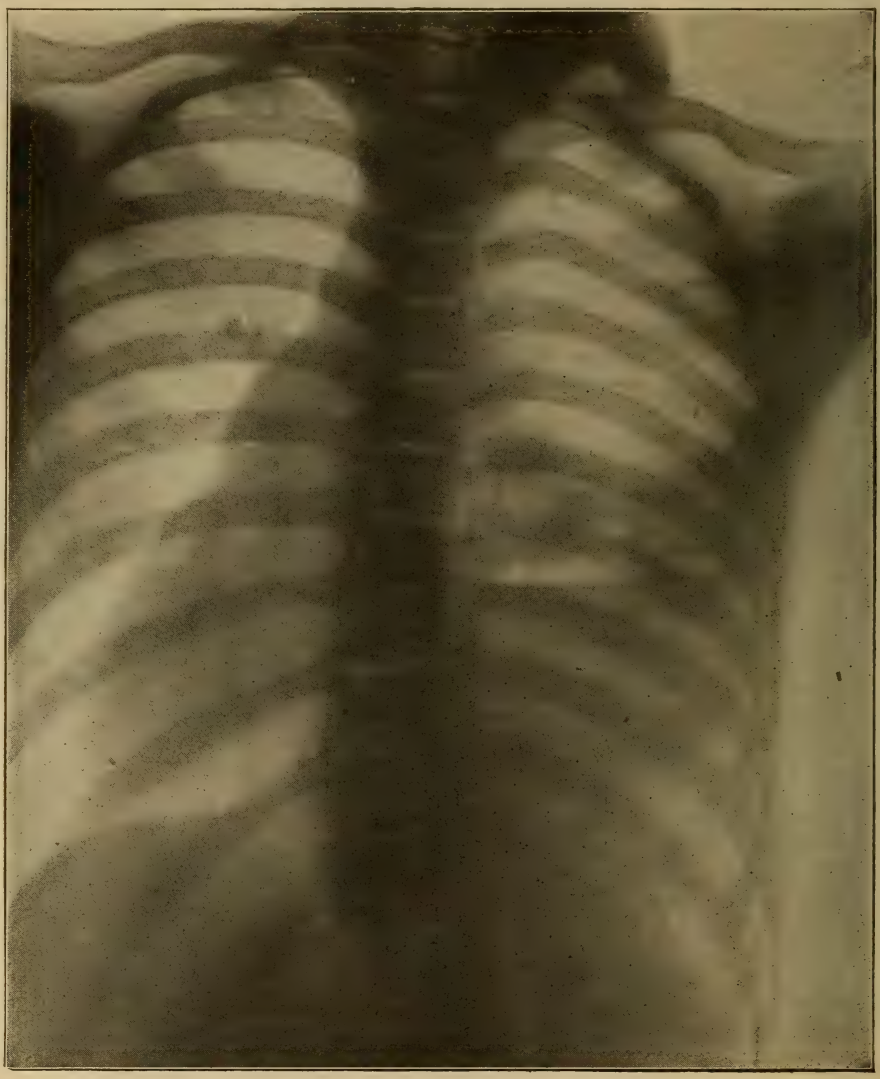


Fig. 138.—Pleurisy with effusion (same figure as shown under Roentgenography). Note displacement of heart to left. Right lung is undergoing gradual compression, *pari passu* with accummulation of the exudate. (*Dr. Frank S. Bissell.*)

Resistance and flatness.

Percussion.—*Palpatory percussion best reveals the absolute resistance over the exudate and simple percussion elicits a flat note, decidedly differing from that of consolidation and simulated only by pleural growths and dense pleural adhesions. Just above the level of the fluid anteriorly, the note is often hyperresonant or tympanitic in quality* (skodaic resonance).

Skodaic note.

Little difficulty is ordinarily encountered in delimiting the upper border of the effusion or in following any slow alterations of its level with changes of posture when these are present.*

Lung compression.

Posteriorly, dulness due to a compressed lung may be encountered and, well above the fluid level in small or moderate established effusions, the unaffected tissue may yield the hyperresonance of vicarious emphysema.

Traube's space.

Movable dulness in Traube's semilunar space often reveals very small free exudates in the left pleural space if light percussion is used.

Characteristic change of note.

The note is not typically flat in such cases, but passes sharply from slight dulness on light percussion to pure stomach tympany as the patient changes from the erect to the recumbent posture.

Boundaries.

Grocco's triangle is an area of dulness lying outside the spinous processes of the unaffected side. Its vertical median boundary represents the vertebral spines and slightly exceeds the height of the effusion; its horizontal inferior border may extend from 2 to 7 cm. outward from the spine and thus the hypothenuse is formed by a line extending from the median line at the upper level of flatness to the outer extremity of the line lying at the lowest level of the same area.

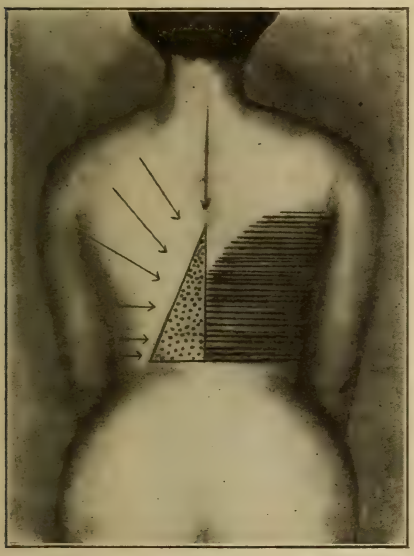


Fig. 139.—The Grocco-Korányi Paravertebral triangle. Pleural effusion on right side. Stippled area represents dulness—arrows indicate the lines of percussion. (*After Da Costa.*)

Its presence is readily explained by the pressure of the dislocated mediastinal contents upon the sound lung and the effect of the liquid exudate in damping vibrations of the adjoining vertebral column. The patient should be sitting or standing, moderate or strong percussion being required and the area best determined by percussing rapidly from the sound lung toward the spine in oblique lines until decided dulness is encountered.

Rationale.

In such cases Grocco's triangle usually shows a marked shrinkage when the patient lies upon the affected side and an increase if he lies on the unaffected side (Ewart's "crucial test"). An area similar to Grocco's triangle may be observed in massive ascites, though it presents a broader base and less height.

Area in ascites.

Auscultation.—Over the compressed lung posteriorly and near the spine the breathing is tubular, but below the level of the fluid the voice and breath sounds are markedly and abruptly obscured, *though seldom entirely lost.*

Compressed lung.

* Marked shifting of the level of dulness is far more constant in transudates than in inflammatory exudates.

Over the elastic chests of children, and even adults, conducted breath sounds may prove misleading though these are usually *of a suggestively distant quality.**

Symptom constant.

The greatly diminished *fremitus* as determined by palpation is a valuable and striking symptom in most of such instances.

Pleuritic friction is sometimes heard above the level of the fluid, particularly if this is receding, and may persist for long periods either as friction or crepitation.

Mensuration.—In large effusions the measurement of the affected side is increased.

Fluoroscopy.—The diaphragm of the affected side is lowered and shows a greatly lessened respiratory excursion and the fluid is clearly indicated by the corresponding shadow.

Rhythmic Lateral Displacement of the Heart.—In 1902, the author reported† observations based upon a number of cases of unilateral pleuritic effusion. He found that in the presence of a considerable unilateral effusion, the heart, in addition to its primary displacement, showed a distinct lateral movement of respiratory rhythm varying with the depth of respiration and the amount of effusion. In some cases, the superficial *heart impulse* could be seen with the naked eye to move outward and inward; in nearly all, auscultatory percussion during full inspiration and full expiration verified the observation, and in every case the fluoroscopic findings were positive.

The phenomenon seems to depend chiefly upon the piston-like action of the diaphragm initiated by the thrust of the abdominal muscles. In full inspiration, pressure is relieved by the descent of the diaphragm and the expansion of the chest, and the readily movable heart tends to approach the median line. In forced expiration, the rising fluid column‡ and the narrowing chest force the organ away from the median line, sometimes to the extent of 3 inches.

The only importance of the observation lies in the fact that it does not seem to occur under conditions such as subdiaphragmatic abscess or in other conditions likely to be confounded with pleurisy, either serous or purulent.

The maximum range of movement occurs in medium-sized effusions. In very small effusions there is practically no movement. In massive effusion both the chest and diaphragm are almost passive. In no such case so far observed has the author found an entire absence of upward thrust and lateral cardiac displacement. Hence, he has not been able to confirm the statement made by certain writers to the effect that in massive pleurisies the diaphragm is *completely* immobile and may even bulge into the abdominal cavity. In every case so far observed the diaphragm on the affected side has shown movement of some degree and *must* if the abdominal muscles act strongly. *Forced*

* Recently the author removed over 1,500 c.c. of serous fluid from an elderly patient in whom pure tubular breathing was marked throughout the area of flatness.

† New York Medical Journal, 1902. Transactions of the Association of American Physicians, 1905.

‡ In every case so far examined the upper border of the effusion was seen to rise in expiration.

respiration is necessary and might demand the exhibition of morphia if pain were severe despite the effusion. In no instance so far observed has this been necessary.

RATIONALE OF PLEURISY

The Normal.—In health the smooth, glistening, lubricated, closely approximated serous surfaces of the pulmonary and costal pleura constituting the internal and external walls of the great potential lymph space move smoothly upon each other without exciting sound or sensation.

Pleuræ normally silent.

Checking of Secretion.—Given an active congestion or inflammation of these surfaces, whatever the exciting cause, they primarily suffer endothelial exfoliation and become dry and harsh: hence pain, limitation of movement and friction sounds, audible and sometimes palpable, appear, to which frank manifestations of toxemia in the form of fever and malaise may or may not be added.

Congestion results in friction.

Toxemia.

Reactionary Changes.—As in the case of all serous or mucous surfaces, primary checking of secretion is followed by reactive oversecretion to a degree dependent upon the intensity and nature of the inflammatory excitation and the constitutional peculiarities of the individual attacked.

Tendency to reactive oversecretion.

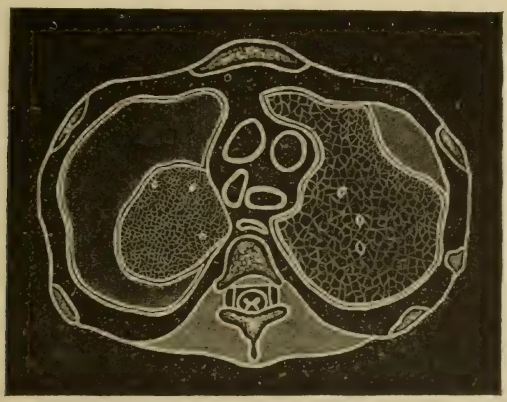


FIG. 140.—Pneumothorax (left), encapsulated pleurisy (right). Lung compression may reach a much higher degree in pneumothorax. Inevitable displacement of heart to right, not shown in diagram. (Repetition of plate.)

Friction Variants.—Manifestly, the friction sounds must be superficially produced and convey to the trained ear the sensation of proximity. Unlike liquid râles due to exudate which are more or less modified by cough or deep breathing, the friction sounds tend to be fixed in location and persistent during any given *seance*. On the other hand, they may be modified even by a reactive fibrinous exudate and the movement of the glutinous surfaces may produce softer friction sounds or even a resemblance to liquid or crepitant râles, which, however, as to persistence and proximity, follow the general rules outlined above.

Qualities of friction sounds.

Misleading modifications.

Lung tissue silent.

Lung Substance Uninvolved.—Aside from the softer breath sounds due to pain limitation of movement there are, of course, at this juncture, no pulmonary signs except such as may represent the antecedent lung lesions in the case of secondary pleurisies.

Complete.

Recovery.—If the inflammatory process now subsides the signs disappear and the lung function may be restored despite any small permanent areas of adhesion between the parietal and pulmonary pleural surfaces, costal or

Incomplete.

diaphragmatic. On the other hand, most extensive adhesions may remain and seriously impair lung function, chest movement and thoracic symmetry.

Separation of pleural surfaces.

Serous Effusion.—If serous effusion appears the pleural surfaces are separated primarily to a degree and over an area dependent upon the amount and location of the exudate and the location and firmness of previously formed adhesions.

Loss of friction.

As a result friction sounds are lost over the area underlain by the effusion.

Filling inferior pleural sinuses.

The liquid naturally gravitates to the lowest free spaces, filling the pleural sinuses if these be not already firmly adherent, slowly but steadily encroaching upon the cardiac area and pushing aside the mediastinal space and its contents.

Displacement of adjoining viscera.

Thus it dislocates the readily movable heart, depresses the liver, spleen, and stomach and obstructs the movements of the diaphragm.

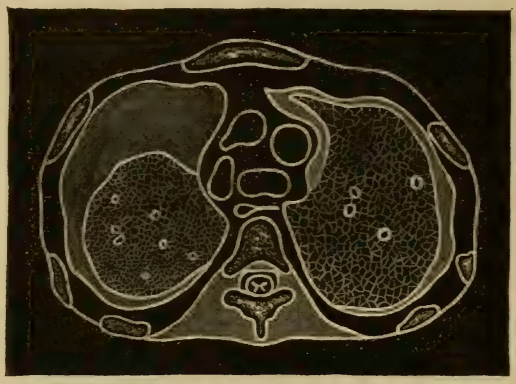


FIG. 141.—Pleurisy with effusion (bilateral). Note pulmonary compression on left. Such exudates as shown upon the right may exist in pneumonia and obscure symptoms. They accompany also malignant growths involving primarily or secondarily the pleura or may occur in any type of pleuritic inflammation.

Lung compression is shown on left side. On the right the upward compression chiefly would be operative at this stage. Displacement of heart not shown in diagram. (Repetition of plate.)

Compression of lung.

As an exudate increases its field of action through gradual accumulation, the lung, so to speak, floats upon it, and its involved portion is gradually and gently compressed toward its hilus and rendered increasingly airless and bloodless. This process does not demand that the exudate shall be under great pressure nor is such the case in most instances.

Deceptive hilus region.

In extreme cases the compression is carried to such an extent as to produce consolidation signs anteriorly, and, even more intensely, posteriorly, over and near the hilus region, provided that the large bronchi remain patent.

These signs contrast sharply with the muffled or absent breath sounds below the level of the exudate itself.

The effect upon air exchanges and the pulmonary circulation is manifestly great and explains the cyanosis and dyspnea, no less than the real danger involved in the rarer cases of abrupt onset of profuse, massive exudates.

Skodaic resonance and egophony.

In that portion of the lung just above the fluid level the normal tonus is markedly relaxed, from which may result the almost constant hyperresonant percussion note and the less dependable, curious bleating voice sound, known as egophony.

Paradoxical signs.

Even below the level of effusion the voice sounds and whispered voice sometimes may be clearly heard or even carry the *timbre*, pitch and intensity of consolidation.

Vagaries of conduction.

Such may be the case if an area of compressed lung connected with a large bronchus be dislocated against the chest wall, or, if tense conducting bands or cords traverse the fluid and are attached at such points as to permit them to carry vibrations from the bronchi.

Fremitus.

The chest, itself, may be so resilient as to convey vibrations from the hilus. Vocal fremitus is, however, rarely increased, save in the elastic chests of children, and still less often is it undiminished even though still palpable.

Decisive Character of Percussion Note.—*In these exceptional cases the differential value of the characteristically flat percussion note elicited by light percussion* and the sense of absolute resistance, is extremely great.*

As stated previously, decided dulness on *light percussion* may be present over Traube's space even in very small *free* exudates if the patient lean forward while erect or in the sitting posture, and such dulness may disappear and be replaced by normal gastric tympany in recumbency. In like manner the area of hepatic relative dulness may be replaced by a flat note with change of posture, very early in the onset of right-sided effusions.

In each case it is the low-lying and roomy complemental pleural space, lying below the lung level and extending almost to the costal border, which receives the flooding exudate.

It is evident that, save for their diagnostic significance as to etiologic factors, the hemorrhagic and purulent exudates, as affecting physical signs, differ in no essential respect from those of serous effusions.

Auscultation signs over the back, in the region of the hilus or in front over and about the compressed lung are manifestly unreliable in large effusions.

Encysted effusions.

It should never be forgotten that encapsulation of effusion may occur through limiting adhesion bands and that this is especially common in empyema, when one or more separate or communicating cavities may exist, each containing pus.

Marked Postural Changes Unusual.—The author ventures to repeat that even serous effusions, if inflammatory, unless associated with pneumothorax, are not necessarily freely movable with changes of posture;

* In the child, especially, heavy percussion may produce a fictitious resonance which obscures the true character of the note.

probably because of the constant resisting pressure offered by the lung and mediastinum and also the tendency to marginal agglutination or adhesions too strong to be overcome by mere gravity, though unable to resist tidal fluid accumulation.

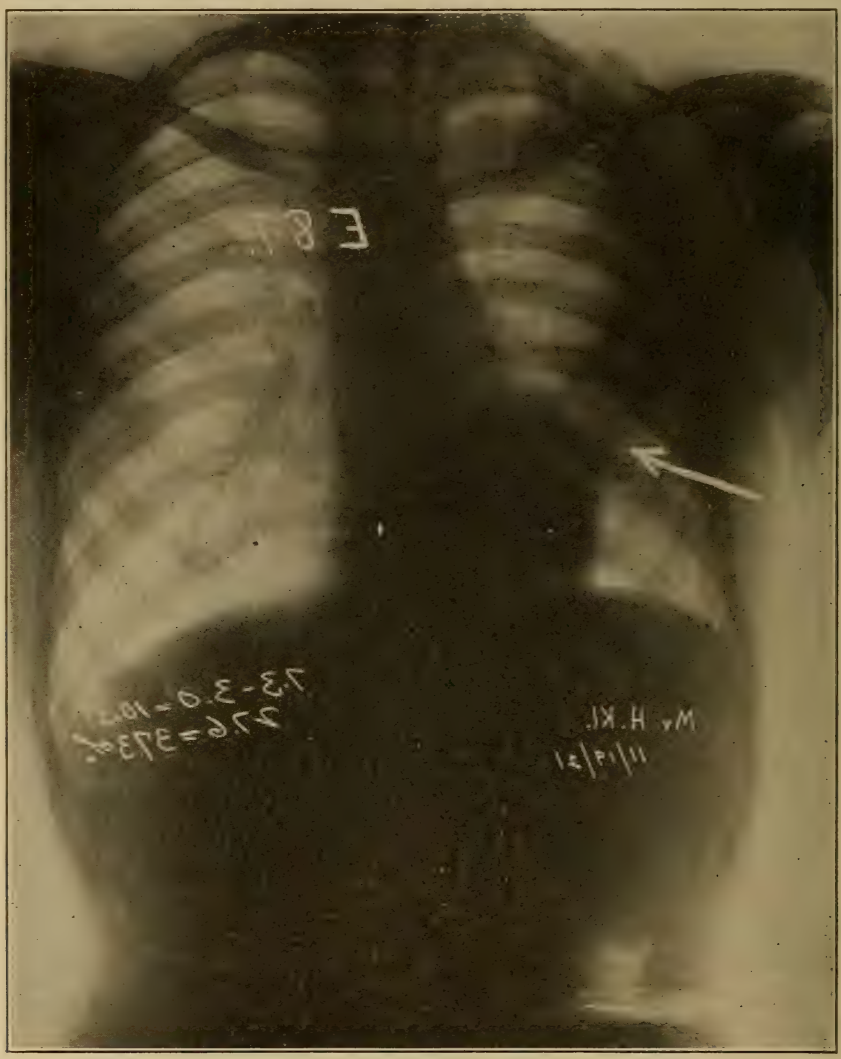


FIG. 142.—Adhesion following double lobar pneumonia. Point of adhesion indicated by arrow. Displacement of heart to left clearly shown. (See Fig. 142 for comparison.)

Many effusions are overlooked because this postural displacement of the upper line of flatness is deemed an essential factor in the diagnosis.

Course and Termination of Pleurisy.—*Simple plastic pleurisy* runs its course in a few days, leaving behind, ordinarily, a few adhesions often discoverable only *post-mortem*. *In cases with serous effusion* aspiration

is usually required, but spontaneous reabsorption is not uncommon especially in cases with a rheumatic etiology.

Long persisting, slow receding exudates leave behind extensive adhesions and, all too often, a lung incapable of more than partial expansion. *Empyema* almost invariably demands surgical interference and leaves behind a

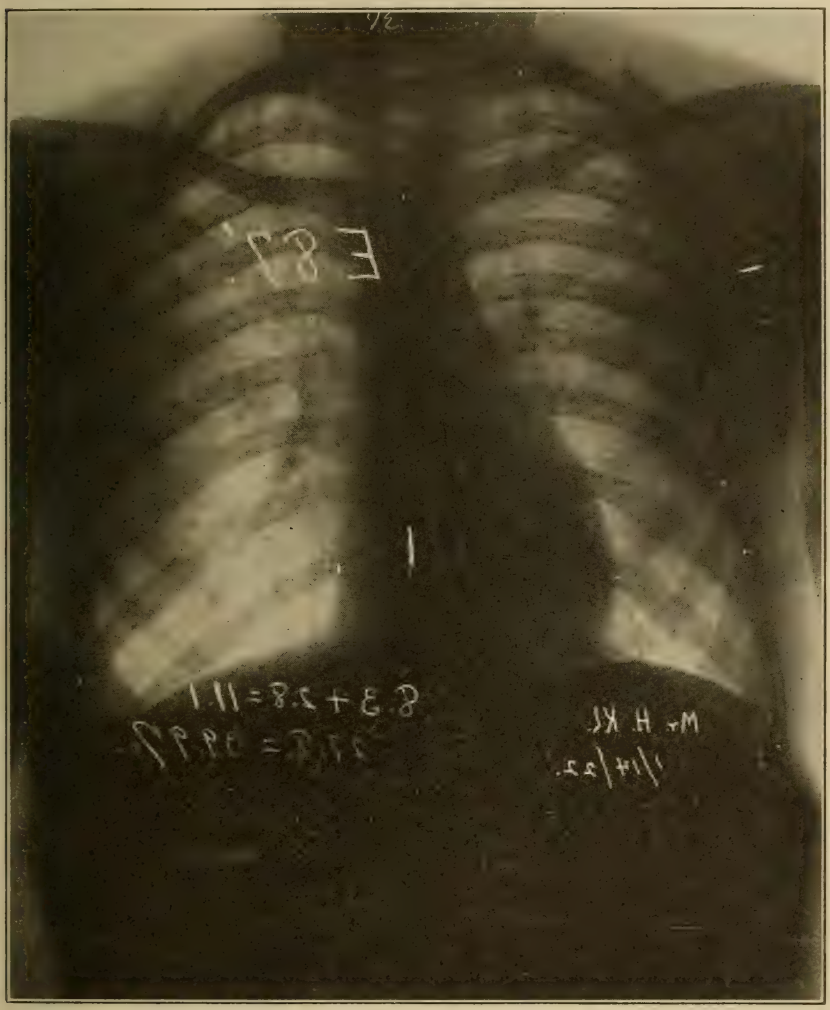


FIG. 143.—(E 87′). Note disappearance of adhesion shown in Fig. 141. and return of heart to approximately normal position. These exposures were made three weeks apart.

variable area of thickened adherent pleura, and an imperfectly expanded, more or less crippled lung.

DIAPHRAGMATIC PLEURISY.—Such a primary pleurisy may be acute or chronic, dry or wet, serous or purulent, and may not extend beyond the diaphragmatic surface, though usually other portions are involved.

Marginal thoracic pain.

Tenderness.

Signs and Symptoms of Diaphragmatic Pleurisy.—*The physical signs may be absent or be those of simple acute pleurisy, with fever, rigid abdominal muscles, costal breathing, an extraordinary degree of marginal thoracic pain on breathing, swallowing, talking or laughing, and oftentimes tenderness over the region of the tenth rib. Referred abdominal pain is especially common and pain in the neck or shoulder is not an unusual finding.*

EMPYEMA

Wet pleurisy plus sepsis.

Symptoms.—*This form of pleurisy is usually distinctly a secondary process, and to the symptoms of serous effusion are added: intermittent (septic) temperature, with or without chills and sweating, sometimes a localized edema, and in rare instances, actual bulging of the interspaces or the ultimate absorption or escape of pus from the pleural cavity into and through the lung to the bronchi.*

"Empyema necessitas."

In such neglected cases it may "point" at an intercostal space, where it forms a tumor beneath the skin and may cause an intractable fistula through spontaneous rupture. Such wall perforations usually occur in the anterolateral portion of the chest.

Blood findings.

The urine may show albumose, and a marked leucocytosis is the rule; the average count being about 18,000, few falling below 12,000, and the blood may show iodophilia. One would naturally expect the highest counts in pneumococcus cases.*

Pyogenic Organisms.—The pneumococcus and the streptococcus pyogenes occur with about equal frequency (40+ per cent.) save in children, in whom the former is the dominant or sole organism in two-thirds of the cases.

While no absolute rule can be formulated, it is usually true of complicating empyemas that streptococcus cases are encountered usually in bronchopneumonia cases and are likely to develop early in contrast to those due to the pneumococcus which are more sluggish in their development, tending to become manifest after crisis and during the convalescent period.

Of these the pneumococcus, while usually producing the more decided symptoms, offers the best prognosis if promptly and efficiently dealt with.

Staphylococcus cases are unusual (3 to 4 per cent.), the balance being tuberculous with rare exceptions.

In the epidemics of influenza, measles, and streptococcus pneumonias experienced during the Great War, the streptococcus empyemas greatly predominated. During certain epidemics the virulence of this complication varied widely and in the fulminant streptococcus type drainage seemed futile, if not harmful.

Act promptly.

Neglected Cases.—Obviously cases of rupture and internal or external discharge are almost invariably the result of neglect or carelessness on the part of the physician and one of the readiest methods of avoiding such disaster and of resolving doubt in difficult cases, is the early *and fearless use of the ex-*

* In simple serous pleurisy a few cases may show moderate leucocytosis during the febrile period, but in fifty-two cases examined by R. C. Cabot, the average count was 8,820, and in only nine did it exceed 12,000.

ploring needle in all cases of pleurisy with effusion and its repeated use if any suggestive symptoms arise,

Obscure cases.

In elderly and debilitated individuals especially, but by no means exclusively, empyema may develop most insidiously. In children with lobar pneumonia it is a frequent and too often, unsuspected complication. Most of the cases of suppositious "delayed resolution" prove to be empyemas.

Common error.

Undue persistence of leucocytosis and fever in lobar pneumonia always suggests strongly the presence of empyema or abscess of the lung.

The Use of the Exploring Needle.—Aspiration may sometimes be performed with an ordinary hypodermic syringe, but inasmuch as a negative result is without significance, owing to the fact that thick pus cannot enter the tiny needle, and further, that the needle itself is much too short even to penetrate the greatly thickened pleura of most empyemas, one usually needs one of the larger syringes of the same type with a long needle of at least threefold caliber.

Choice of needle.

The resistance felt and the fixation or holding of the exploratory needle in cases of dense adhesion is decided and constitutes one of the oldest of the grosser symptoms of this condition. It may be encountered also in long-standing cases of empyema or the persisting serous effusions of polyserositis.

Adhesions.

Such an examination, performed under proper antiseptic precautions, is almost absolutely free from danger. The physician's fingers laid upon the ribs should mark the limits of the interspace and the needle should be thrust quickly and deeply between them without touching the rib and keeping well to the lower part of the interspace to avoid the intercostal artery. If fluid be present and the point entered has been rendered anesthetic by the ethyl chloride spray, or other means, the procedure is painless.

Danger almost nil.

Use needle boldly.

Painless usually.

The site of the puncture is ordinarily the fifth interspace in the axillary line, but it is often necessary to enter the chest at other points representing maximal dulness, and if care be taken to avoid the heart, spleen, liver, great vessels and diaphragm the operator has a wide latitude.

Site.

The X-ray.—It is unnecessary perhaps to emphasize the great value of X-ray examinations in these cases where the proper facilities exist for its use.

PLEURITIC ADHESIONS.—*A clinical diagnosis of slight adhesions is often impossible, even fluoroscopically, but in general, the symptoms are as follows:*

Inspection shows impaired movement of the affected side or retraction and mediastinal displacement if extensive adhesions are present, and Litten's sign may show marked impairment of the diaphragmatic excursion on the affected side.

Lagging and retraction.

Palpation shows diminished vocal fremitus and lessened expansion.

Percussion shows a change of note varying from slight dulness to absolute flatness according to the extent and density of the adhesion.

Auscultation shows diminished breath sounds and voice conduction, and oftentimes, in certain areas, pleuritic râles.

Fluoroscopy.—A distinct shadow may indicate extensive adhesions and the diaphragmatic movement is markedly impaired.

INTERLOBAR EMPYEMA.—This ordinarily takes the form and presents the symptoms of an interlobar abscess situated between the upper and middle lobes near the root of the lung. It presents great difficulties to the diagnostician unless the roentgen-ray is available, and is ordinarily mistaken for true pulmonary abscess on account of its peculiar location. The purulent accumulation may vary from an ounce or two to a pint or more. It ordinarily follows a general pleurisy. (See "Roentgenography.")

An obscure disease.

Chronic Pleurisies.—These may be dry or wet, serous or purulent, and for the most part present the same physical signs as are found in acute disease. Chronic pleurisies are usually tuberculous and the same may be said of chronic empyema, but they often represent neglected non-specific cases. Malignant disease of the pleura, and active tuberculosis, are the commonest conditions attended by hemorrhagic effusion and chronic inflammation.

Usually tuberculous.

Chylous Exudate.—In rare instances rupture or blocking of the thoracic duct may result from trauma, carcinoma or other tumors, and find an outlet in the pleural cavity. The nature and differentiation of such exudates is described elsewhere.

Character of the Purulent Exudate.—The macroscopic appearance of the exudate is an uncertain guide to the identity of the dominant organisms.

In general terms one may say that the streptococcic *type* is that represented by the relatively thin purulent exudate, separating into a double layer, thin above, thick below and containing little fibrin.

Streptococcic vs. Pneumococcic.

Pneumococcic purulent accumulation, on the contrary, is usually creamy, fibrinous, viscid and homogeneous. Nevertheless, the author has encountered most virulent and deadly exudates, showing pneumococci in pure culture yet outwardly resembling the streptococcic exudate.

Treacherous guide.

Encapsulated Empyema.—The tendency of purulent exudates, especially, to become encapsulated must never be forgotten. Quite frequently the main cavity is emptied by operation and larger or smaller, single or multiple pockets left behind to continue the drain upon the vitality of the patient.

Relatively common finding.

In this connection the X-ray is most helpful, though usually unnecessary if a thorough physical examination is made before the patient leaves the operating table.

Necessary precaution.

PNEUMOTHORAX

Definition.—*An effusion of air or accumulation of gas within the pleural cavity. A liquid exudate is almost invariably present and the true descriptive terms are pyopneumothorax, hydropneumothorax, and hemopneumothorax, the first being the commonest form as the tendency in almost all cases is toward ultimate purulent transformation.*

Etiology.—By far the greater number (90 per cent.) are associated with advanced tuberculosis of the lung and the formation of a fistulous communication, though in neglected empyema a fistulous opening may originate in the pleura. Among other causes may be mentioned penetrating wounds of the chest wall, traumatic rupture of the lung, rupture of emphysematous

Usually tuberculous.

Multitude of possible causes.

vesicles, pulmonary abscess, gangrene, infarct, cancer, growth of the B. *aerogenes capsulatus* in a liquid exudate, and even hydatids.

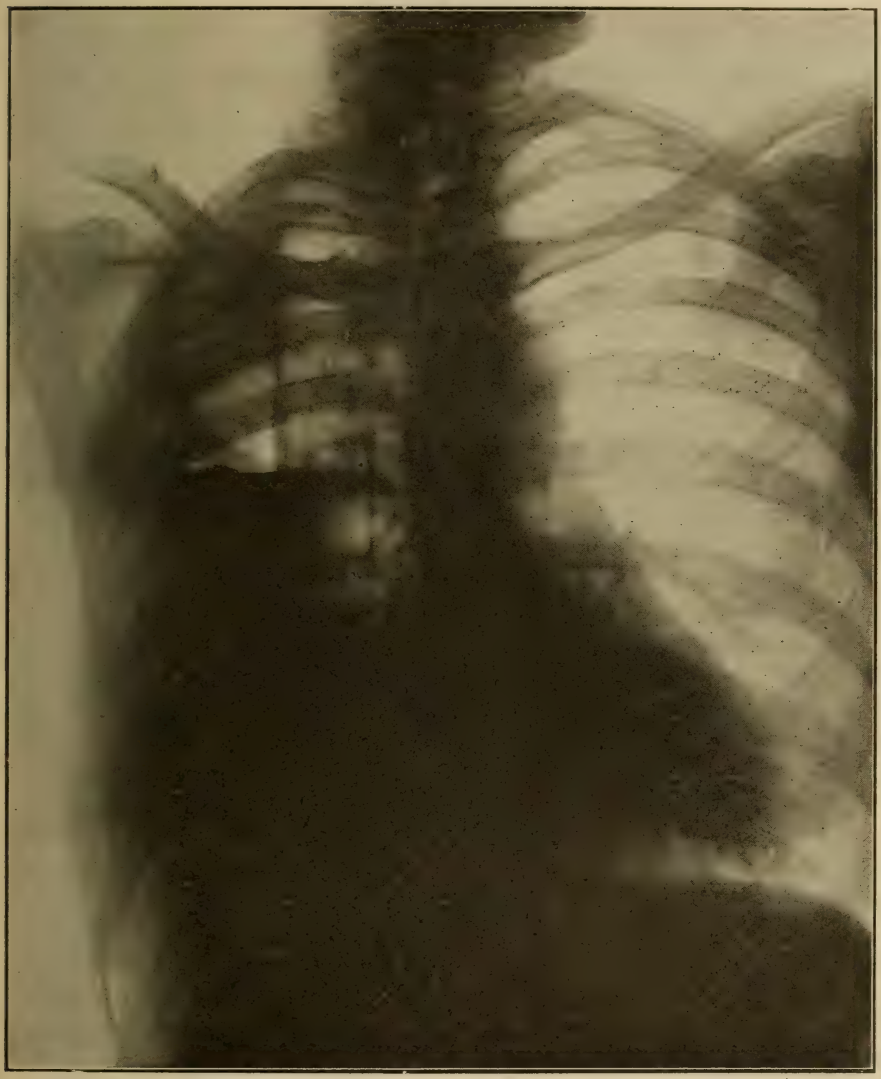


FIG. 144.—Pyopneumothorax. Note knob indicating collapsed lung. Also dense shadows produced by bismuth paste, introduced through fistula under false diagnosis. (*Dr. Frank S. Bissell.*)

Males chiefly affected.

Nearly all of the cases occur in males and the disease is invariably unilateral. Sudden and complete bilateral pneumothorax should cause almost instant death.*

*A case of left hydropneumothorax recently observed by the author occurred suddenly, without warning, when the patient, a healthy young adult, was engaged in office work and entirely free from any cough or other symptoms. After the primary collapse induced by the abrupt onset, which occurred in the absence of any muscular effort, sneezing or coughing,

Massini and Schönberg report a case, verified by the roentgen ray and by the autopsy, which lay alive in the Basel clinic for eight days after the seizure, though both lungs were compressed to an incredibly small volume.

SYMPTOMS AND RATIONALE OF PNEUMOTHORAX

In pneumothorax as ordinarily encountered, the first inrush of air is followed by a period during which it is rhythmically pumped into the pleural space, through a single valve-like opening, by the respiratory movement and recurrently "trapped," thus creating an intrapleural pressure such as is almost unknown in the case of large liquid exudates, as may readily be demonstrated by the X-ray plate. The lung is *abruptly and excessively* compressed in the direction of its root, the mediastinal tissues, if not anchored by old adhesions, being displaced maximally toward the unaffected side, and diaphragmatic movement unilaterally almost wholly abolished.

Lung compression.

Usually pyopneumothorax.

The pleura becomes inflamed and in most cases a *small amount* of purulent, rarely a serous, effusion is present. In open pneumothorax the mean intrapleural pressure is, of course, that of the atmosphere. With the closure of a pneumothorax of any type, gradual absorption of the *gaseous* content and relief of that pressure may be anticipated.

The onset *is almost invariably sudden and usually follows directly upon injury, physical strain, vomiting, or, more frequently, a paroxysm of cough.*

Shock.

Symptoms Urgent.—*Symptoms of severe shock result from the sudden compression of the lung and the mediastinal displacement, there is urgent dyspnea, cyanosis and lividity, as the result of the strain thrown upon the right heart and the sudden reduction of aëration areas, and the patient ordinarily feels that there has been some "internal rupture."*

Dyspnea and cyanosis.

Silent Onset Rare.—A genuinely insidious and subjectively symptomless onset is exceedingly rare and the author has observed but three such cases. In these the opening is tiny and so obstructed that only the smallest quantities of air *intermittently* enter the pleural cavity.

PHYSICAL SIGNS.—Inspection must necessarily show immobility and bulging of the affected side and shallow or flush interspaces, as the result of the substitution of high positive for the normal negative pressure, together with evidence of a displaced heart, unilaterally impaired diaphragmatic movement, the physiognomy of shock, cyanosis, and marked dyspnea. The patient usually lies upon the affected side in order to facilitate the movements of the sound side.

Striking symptoms.

Important peculiarities.

Palpation.—This confirms inspection and as there is usually no underlying lung save that anchored by adhesions or compressed at the hilus, vocal fremitus must obviously be greatly diminished or entirely lost over the air-filled

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and was so severe as to be most alarming, the case progressed slowly but steadily and uneventfully to complete recovery. Personal and family history were alike wholly negative, the X-ray then, and after rapid, complete recovery, revealed absolutely nothing of etiologic importance, and the patient is now in perfect health. Such cases are among the curiosities of medicine.

area, exactly as in the case of liquid pleural effusion and with the same rare exceptions, but extending over a wider area and without the upper limitation or definite demarcation of a liquid effusion.

Percussion.— *The percussion note* is ordinarily distinctly tympanitic, though temporarily, and under conditions of extreme pressure, quite a marked degree of dulness may be encountered.

Dulness possible but rare.

The entire pleural cavity is air filled, if limiting adhesions are lacking.

A somewhat neglected sign *is the resulting extreme initial extension of hyperresonance, which in the absence of adhesions, follows the boundaries of the pleura rather than that of a lung and, therefore, involves the entire half of the thorax, sometimes extending across the median line to the opposite sternal border.*

Entire space air-filled.

Fluid is, of course, almost invariably present but is at first, and usually indeed throughout, not only small in amount but *freely movable* and hence easily displaceable by postural shifts made for purposes of examination.

Free fluid.

Moreover it always presents a *horizontal upper boundary.*

Displacement of Sheltered Viscera.—It is evident that in such cases hepatic, cardiac, splenic and renal, *thoracic* areas of dulness appertaining to the affected side are markedly diminished or entirely lacking and that the contrast between the boundaries of thoracic resonance of the two sides is extremely marked.

Auscultation.—Breath and voice sounds are necessarily feeble and distant or entirely absent except directly over the region of the hilus near the spinal column or sternum.

Hippocratic Succussion.—If the patient be raised cautiously to a sitting posture and *gently* rocked to and fro, *Hippocratic* succussion is obtained. This is merely a splashing sound caused by the waves of the free fluid in the great air space, readily detected if the ear is applied to the chest during the maneuver, and often heard at some little distance.

A "swashing" sound.

Free mobility of the fluid may be readily demonstrable by fluoroscopy.

An exquisite "*metallic tinkling*" is present in many cases, as is the "*coin sound*," obtained by placing the ear upon the chest wall anteriorly and having an assistant place a good-sized silver or gold coin on the back and tap it with another, the conducted sound having a musical, bell-like or clear metallic ring.

Metallic tinkles.

The bell sound.

The "water whistle sound," resembling that produced in drawing air through water by suction, as in smoking the *narghili* of the Turk, may be present when an open lung fistula lies below the surface of the exudate.

Cardinal Signs.—*The four cardinal signs are* (1) *metallic tinkling* (due probably to the dropping of fluid from the lung above into the exudate below in the smooth, tense-walled, air-filled space); (2) *Hippocratic succussion;* (3) the *coin sound* which is due to selective reënforcement of vibrations and overtones; (4) the X-ray findings, usually clear and absolute.*

* In certain cases the fluoroscope reveals the paradoxic diaphragmatic response to inspiration; that of the affected side ascending, by reason of the relief of intraabdominal pressure coincidently with the descent of its fellow on the sound side.

All of these signs together with the widely diffused hyperresonance and cardiac dislocation, are ordinarily strikingly clear. An error in diagnosis can seldom occur save in the cases in which an encapsulated pneumothorax exists or in the extremely rare instances of huge pulmonary cavitation. Such may simulate pneumothorax but rarely or never yield its pressure signs. In cases where*

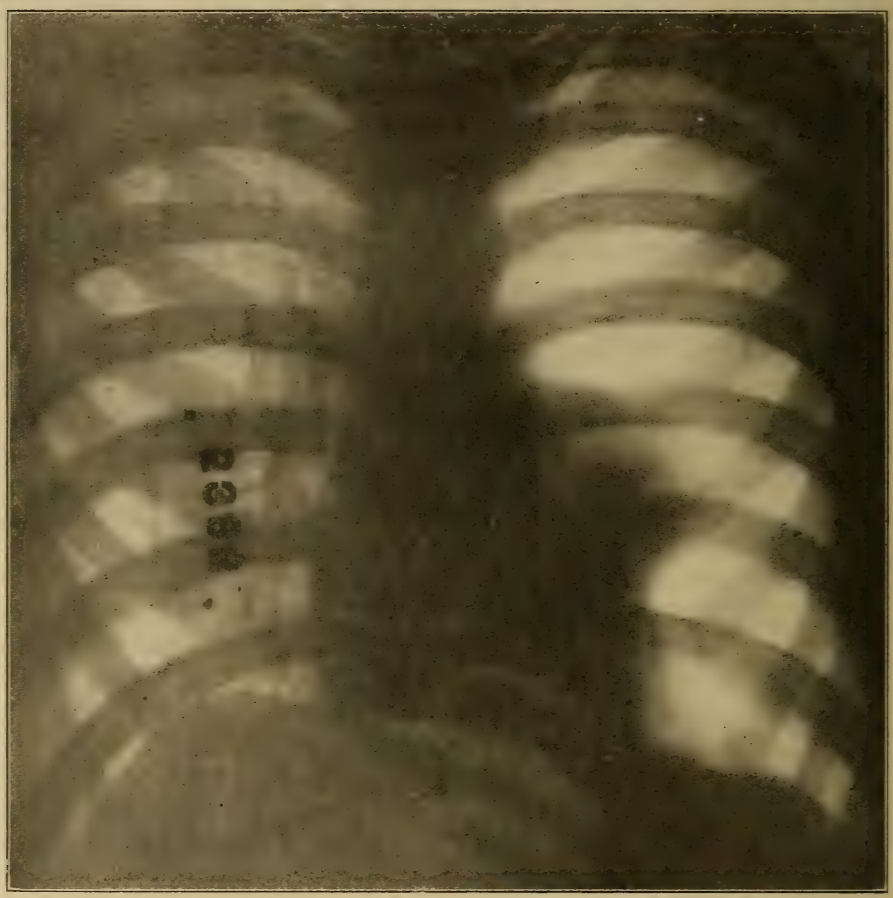


FIG. 145.—Pneumothorax. Case quoted in text as showing sudden onset in patient enjoying then and now perfect health. Note knob representing compressed left lung. This picture was taken one week after onset. It will be noted that the heart (modified "drop" type, showing congenital tendencies largely overcome) is *apparently* normal in size (13 cm.) and position, though markedly displaced originally. Patient slender and small boned, but otherwise of exceptionally good physique. (Compare with Fig. 145.)

a large communicating opening exists, the signs may be merely those of an enormous open pulmonary cavity with firm walls.

* In the most extreme cases of this kind ever encountered by the author, the entire right lung, save for a few shreds, had been destroyed and very imperfect physical signs of pneumothorax were present. The opposite lung, also the seat of multiple tuberculous foci, had so hypertrophied as to encroach upon its empty air-filled fellow, and thrust the heart so far into that cavity as to give the appearance of a *situs inversus*.

Metallic gurgling and bubbling may result, if a patent fistulous opening enters below the level of the fluid. (See above.)

In diaphragmatic hernia the history, the X-ray, borborygmi (rumbling, Diaphragmatic hernia.

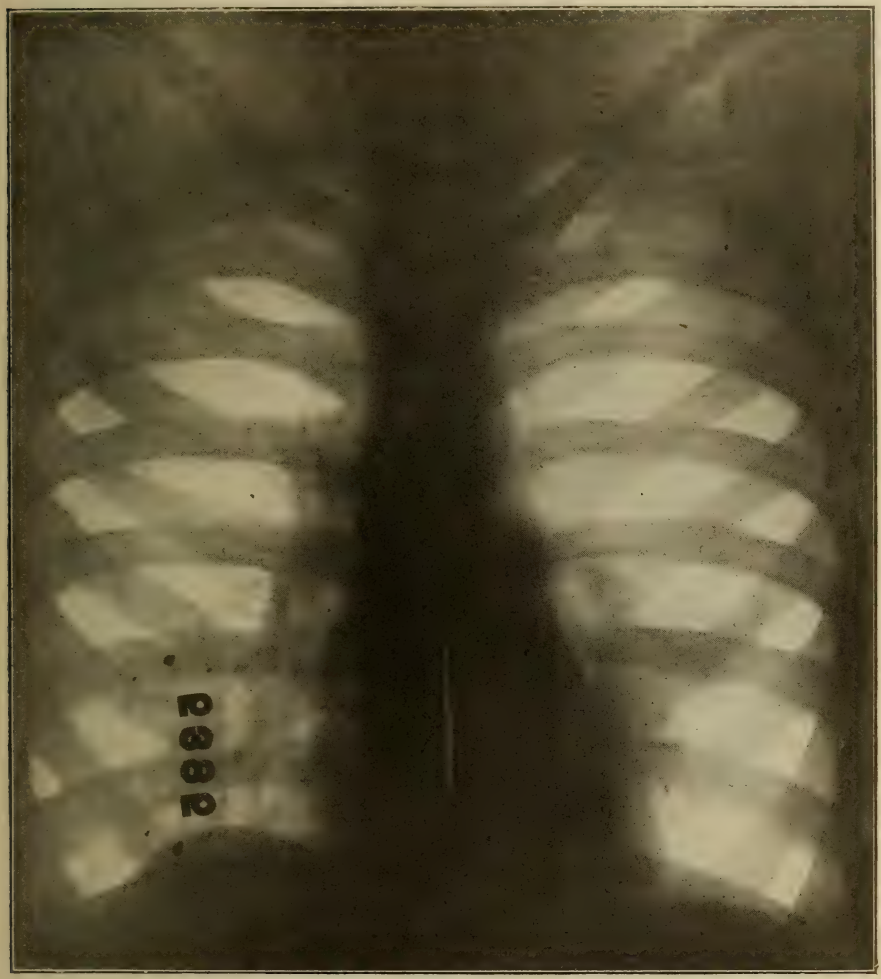


FIG. 146.—Apparently causeless pneumothorax. Case quoted in text occurring abruptly in man of perfect health, robust physique, and extraordinarily clean family and personal history. Absolutely well up to this time (ten years). This picture was taken two weeks after onset. Note expanding left lung. Heart showed a decided reduction in size a few weeks later (total transverse measurement 11 cm.). Modified "drop." Such hearts are apparently almost invariably associated with a symptomless gastroptosis, an associated asthenic stigma less readily remedied structurally than the cardiac stigma. Initial small exudate promptly absorbed in this instance. (See Fig. 144.)

cooing,) and the change of note on taking large quantities of water into the stomach or colon may assist in differentiation.

Such hernias are almost invariably left sided, which fact goes far to rule them out when the right half of the thorax is found air-filled. Roentgeno-

graphy after the ingestion of a bismuth meal is clarifying. The symptoms are predominatingly though not wholly abdominal, the stomach and a part of the colon forming the hernial mass in most instances. Furthermore, the lung structure may be visible on the X-ray plate despite the presence of a gas-filled stomach or colon. "Idiopathic unilateral ascent of the diaphragm" is so rare as hardly to need consideration in this volume.

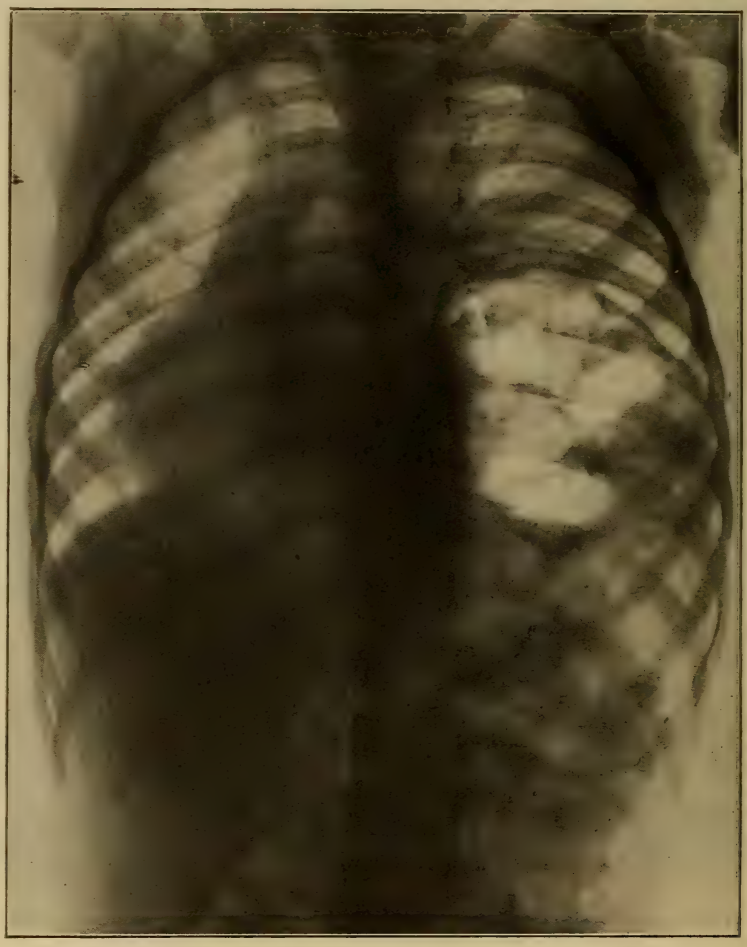


FIG. 147.—Diaphragmatic hernia. Note gas and colonic markings high in *right* thoracic cavity. An unusual location, most cases being left-sided. (*Dr. Frank S. Bissell.*)

Course and Termination.—The course of pneumothorax depends chiefly upon its cause and as those associated with chronic tuberculosis (90 per cent.) are almost invariably fatal, the mortality is enormous.

HYDROTHORAX.—This is a serous pleuritic *transudate* without pleuritis, and is a secondary complicating ailment, associated with cardiac and renal disease or terminal anemias and cachexias. Unlike the effusions of pleurisy,

Suggestive likeness.

it is bilateral though unilateral predominance of transudation is not uncommon and the difference may be postural in causation.

It is usually one of the later events in an anasarca and the physical signs are those attributable to liquid pleural effusions from whatever cause, to which may be added striking postural change in level.

Such transudates may occur insidiously at any time, in Bright's disease especially, and the condition is often overlooked.

LOBAR PNEUMONIA

(*Pneumonitis, Croupous Pneumonia, Lung Fever.*)

Definition.—*An acute infection characterized in its typical and usual form by primary fibrinous inflammation of the lobar type, polymorphonuclear leucocytosis, profound toxemia, the frequent presence of pathogenic pneumococci in the circulating blood in the more virulent infections and an abrupt onset and termination.*

Agents of Infection.—Though found in normal buccal secretion and diseases other than pneumonia, *Fränkel's diplococcus pneumoniæ or micrococcus lanceolatus* is the most constant causal factor. It is present constantly in the saliva of some persons (25 to 30 per cent.), and is found also in the secondary processes of the disease. It is elliptical, the lanceolates occurring in pairs, and, in sputum, is ordinarily encapsulated.*

FIG. 148.—Diplococcus Pneumoniæ. (Frankel-Weichselbaum) and the pneumobacillus of Friedlander. Upper segment shows former, lower segment shows latter.

Other organisms such as the Pfeiffer bacillus, the streptococcus or staphylococcus are found not infrequently in association with pneumococci in cases of lobar pneumonia, but the last is well established as the causative factor in an overwhelming majority of the cases.

The recently established fact that wide differences with respect to virulence and the conferring of immunity exist in members of the pneumococcus group has served to explain much that was obscure and apparently contradictory in bacteriologic, clinical and statistical studies of this disease.

We now recognize the existence of various strains possessing certain cultural characteristics in common yet varying sufficiently in their reactions to the sera of immunized animals to permit accurate classification and differentiation.

Limitations of space forbid any extended discussion of this great advance in our knowledge of lobar pneumonia which makes possible more accurate

* It is readily stained as follows: (*a*) Make thin smear, dry in air and fix by heat. (*b*) Cover surface with the His stain (gentian violet, saturated alcoholic solution 5 c.c. plus 95 c.c. distilled water. (*c*) Heat till steam rises. (*d*) Wash with copper sulphate solution (20 per cent.). (*e*) Dry and mount. The germ is Gram-positive.

diagnosis, a better understanding of transmissibility, epidemiology, virulence, immunity, treatment and mortality.

The table appended shows the classification at present in use at the Rockefeller Institute. Adequate laboratory service is essential.

DETERMINATION OF PNEUMOCOCCUS TYPES BY AGGLUTINATION

Pneumococcus suspension 0.5 c.c.	Serum I (1:20) 0.5 c.c.	Serum II undiluted 0.5 c.c.	Serum II (1:20) 0.5 c.c.	Serum III (1:5) 0.5 c.c.
Type I	++	−	−	−
Type II	−	++	++	−
Subgroups II				
a, b, x	−	+	−	−
Type III	−	−	−	++
Type IV	−	−	−	−

Incubation for 1 hour at 37°C.

The incidence and mortality in 100 cases at the same institution are shown in the following table:

INCIDENCE OF VARIOUS TYPES OF PNEUMOCOCCI AND RESULTING MORTALITY

Type of pneumococcus	Incidence, per cent.	Mortality, per cent.
I	33	25
II	31	32
III	12	45
IV	24	16

Three stages.

MORBID ANATOMY.—Three stages of the disease are recognized for purposes of clinical convenience, but are often blended at autopsy.

1. **Congestion.**—The lung is enlarged, dark red, less elastic and less crepitant than normal and exudes frothy red liquid on section, but portions will float in water. The microscope shows marked capillary turgescence and erythrocytes, leucocytes and desquamated epithelium.

2. **Red Hepatization** (**Consolidation**).—The lung does not crepitate, is friable, and sections sink in water. The cut surface is finely granular, the exuded serum is bloody (not frothy) and minute fibrinous plugs may be scraped from the surface.

The *microscope* shows a coagulated fibrinous exudate and fibrinous threads holding in mesh great numbers of erythrocytes, leucocytes, and desquamated alveolar cells. There is marked round-cell infiltration of the interalveolar septa.

3. **Gray Hepatization** (*Resolution*).—In this stage the gray color displaces the dark red of congestion and consolidation, the lung rapidly softens, becomes more crepitant, but is still friable, and a purulent exudate follows

the knife. As a result of the subsidence of inflammation and hyperemia, liquefaction of the corpuscles and fibrin takes place through a digestive ferment possessed by the leucocytes, the cellular elements undergo fatty and granular degeneration, and, within from two to four weeks, absorption taking place through the lymphatics, together with free expectoration, remove the inflammatory products.

The duration of the stages of pneumonia is extremely variable. Consolidation may be complete in a few hours, but usually takes from twelve to twenty-four. The hepatization period varies from three days to two weeks or more, from three to nine days being the usual period. The process of resolution is wonderfully rapid, and with its coming the whole complexion of the case changes in a few hours.

Rapid changes.

Points of Importance.—*The morbid anatomy makes clear the physical signs of the disease, but one should remember that areas contiguous to consolidation are hyperemic and edematous and that bronchitis and pleurisy of some degree are invariable accompaniments of fully developed lobar pneumonia. Pleuritic effusion is comparatively slight and usually undiscoverable by physical examination, yet pneumonia constitutes one of the chief causes of empyema, especially in children.*

Associated conditions.

Predisposing Factors.—60 or 70 per cent. of all cases occur in males and in the United States the colored people suffer more than the whites. Children between the ages of two and six are especially susceptible, and after an interval of relative immunity its incidence and mortality steadily increase from puberty to old age. Hence, in incidence and mortality it is a disease of the extremes of life. It is most prevalent in the late winter and early spring (75 per cent.), is present in all civilized countries whatever their latitude or climatic conditions, and is peculiarly fatal in some high altitude regions, as in Butte, Mont., where the elevation and smelter fumes, in the past made it exceptionally deadly. Alcoholism, acute or chronic, is a marked predisposing factor unfavorably affecting prognosis and many acute "drunks" are brought to the police station suffering from an unrecognized pneumonia.

Males chiefly.

Age.

Season and altitude.

Habits.

Fatigue and exposure to cold and wet if coincident are *predisposing* factors. Individual predisposition is marked; one attack invites others and an astonishing number of recurrences have been reported in the same individual.

Fatigue and exposure.

It must be noted, however, that recurrent attacks in most instances represent the activity of a type of pneumococcus other than that producing the original attack.

The contagiousness of pneumonia is a proven fact and a reasonable degree of isolation should be established in all cases. On the other hand individual immunity is common and the degree of contagiousness varies with the type of organism present in the given case.

Cold in itself must be regarded merely as a means of depressing vitality and lowering resistance and is more potent when combined with dampness.

Cold merely contributory.

City dwellers are much more subject to the disease than are those living in the open country.

Antecedent Disease.—Coryza and bronchitis often precede an attack,

Coryza and bronchitis.

and no doubt furnish a favorable soil for the further development, increased virulence and wider dissemination of pneumococci.

Chronic ailments.

Terminal Pneumonias.—Chronic disease may play a prominent part, and pneumonia is a frequent terminal event in acute or arrested pulmonary tuberculosis, chronic nephritis, diabetes, carcinoma, arteriosclerosis, prostatic hypertrophy and chronic heart disease.

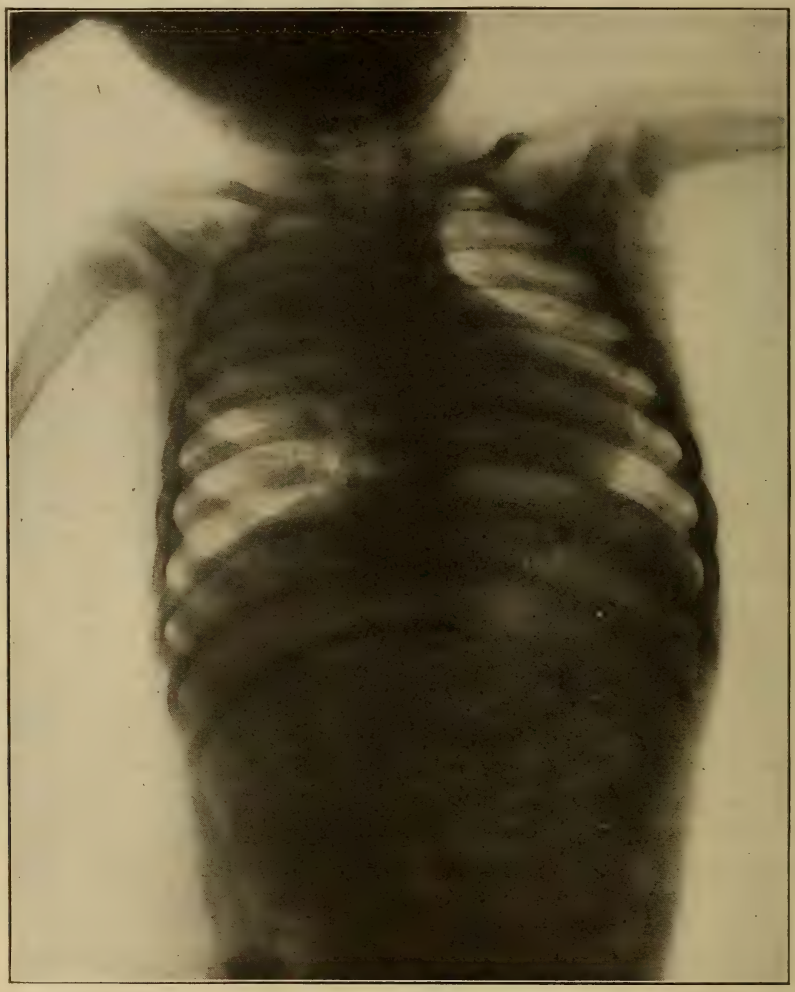


FIG. 149.—Lobar pneumonia in child five years old. Upper right lobe involved. No clear physical signs. Patient roentgenized to determine cause of high temperature. Resolution by crisis on ninth day. Apparent cardiac displacement represents distortion of outline due to faulty position of child at the moment of exposure. Probably a "massive" pneumonia, *i. e.*, one with blocked bronchi. (*Dr. Frank S. Bissell.*)

Complicating.

VARIETIES OF PNEUMONIA.—*Pneumonia may complicate any acute infectious disease and is especially frequent in influenza.*

Hypertoxic cases.

The term "typhoid pneumonia" should be confined to pneumonia with an overwhelming toxemia which produces a typhoid state, not to pneumonia complicating typhoid fever.

Traumatic, Migratory, and Massive Forms.—*Traumatic pneumonia is rare and migratory pneumonias are both unusual and interesting.*

Massive pneumonias are sometimes encountered in which the large bronchi are plugged and the physical signs greatly obscured by suppression of auscultatory phenomena.

Puzzling cases lack displacement signs.

They most strongly resemble pleurisy with effusion but lack marked visceral displacement signs and the low level of flatness of that condition. Furthermore, in the full-developed case one finds the dulness more or less definitely related to lobar divisions and the true *flatness* of liquid effusion is usually absent though dulness may be so extreme as to require the use of the exploring needle for differentiation.

Often obscure lobar boundaries.

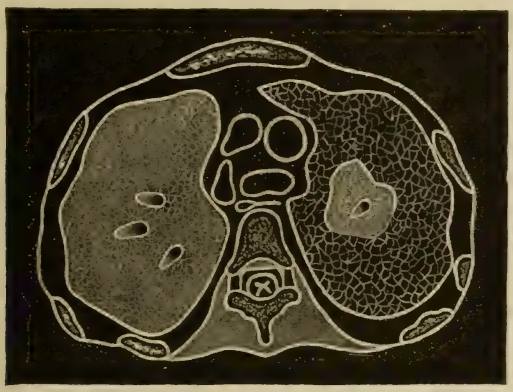


FIG. 150.—Lobar pneumonia (left); central pneumonia (right). The homogeneity and density of tissue resulting from the complete consolidation of tissue shown on the left makes clear the reason for the exquisite transmission of the peculiarly modified breath and voice sounds, no less than the dull percussion note. Compare this diagram with that illustrating pleural effusion. If the large bronchi were blocked the signs of pleurisy might be exactly simulated. (Repetition of figure.)

Afebrile and Senile Pneumonia.—*Afebrile pneumonia occurs especially in the aged, and it should be remembered that senile pneumonias may lack chill, high fever, cough, crisis, expectoration and typical physical signs.*

Often overlooked.

Central pneumonias *are very misleading, and it may be days before the process reaches the surface and yields physical signs of a positive nature.*

Distant tubular breathing.

One should carefully note any distant breathing of a tubular quality, however faint, especially if associated with increased transmission of the voice (fremitus), whether to the finger or to the ear.

Significant areas.

No less significance should be attached to the circumscribed areas of hyperresonance which frequently precede the consolidation and are often sharply and suggestively limited by the interlobar division lines. The sputum is, moreover, often typical and diagnostic.

Note.—*In all varieties of lobar pneumonias save the senile group, the abrupt onset and decided symptoms of febrile toxemia suggest the disease which is further attested by the relatively high leucocytosis so generally present. It should be remembered that pleural effusion may accompany it.*

Delayed Resolution—In the author's experience *long-delayed* resolution is an extremely rare event, usually proving to be an empyema; but slow

Rare.

resolution is relatively common. The oft-asserted frequency of delayed resolution in the lobar pneumonias of children is open to doubt, in the opinion of the author.

A common error.

In an astonishing number of instances in his consulting and public practice a supposedly delayed resolution in the child has proven to be a pleuritic exudate and this, but too often, purulent in character.

Doubtless pleural exudations complicate the lobar pneumonias of children with especial frequency and in the child, even the empyemas of the pneumococcus group may and occasionally do undergo spontaneous cure. These facts would lead to exaggeration with respect to the occurrence of "delayed resolution."

Selective Points.—The right lung is affected in about 60 per cent. of the lobar pneumonias, the left in 30 per cent., and both in from 10 to 15 per cent. of all cases. As regards the lobes affected, apex pneumonia occurs in about one-fifth of the cases, especially in influenzal infections, and the whole lung is affected about as often as is the lower lobe alone, in which the inflammation usually commences.

Terrible mortality.

General Comment.—In these United States from 70,000 to 80,000 of our people die each year of lobar pneumonia, and the disease is apparently increasing. As Osler says, it is the real "Captain of the Men of Death,"

Increasing.

and the true "friend of the aged." Its apparent increase may be due to more careful methods of diagnosis, but it is probable that other factors are operative.

Striking usually.

SYMPTOMS.—The ordinary symptoms of lobar pneumonia are: (*a*) **Chilliness** or repeated rigors, for which convulsions may be substituted in children.

(*b*) **Fever.**—Rising abruptly from normal to 103 to 105°F. and tending to assume a continuous form. It lasts throughout the period of congestion and hepatization (ordinarily from three to fourteen days) and terminates by crisis.

Dramatic contrast.

(*c*) **Crisis.**—A critical drop in temperature and the dramatic contrast between intense suffering and relative comfort is an astonishing phenomenon. The fall usually reaches subnormal and is attended by sweating or perhaps diarrhea. Immediately before it there may be a marked rise in temperature (pre-critical); following it there may be secondary elevation rapidly subsiding

Pseudo-crisis.

to normal (post-critical). *A pseudo-crisis* may occur and raise false hopes, and finally, *an ante-mortem rise or fall* may occur. The latter is most misleading, but is ordinarily associated with other signs of impending death that should prevent misconstruction.

Pleuritic in type.

(*d*) **Pain** is almost invariably present and *is distinctly pleuritic in type.*

(*e*) **Cough and Expectoration.**—In the early stages the cough is dry, painful and unproductive, the sputum is at first scant and mucoid, then muco-

Rusty sputum.

purulent and blood-streaked, later, during hepatization, it becomes rusty

"Prune juice" sputum.

and during resolution creamy, in appearance. The aptly named "prune juice" sputum is of serious portent and *the sputum of the first two stages is so extraordinarily tenacious as to cling to the lips and to the bottom of the inverted sputum cup.*

(*f*) **Respiration.**—Dyspnea is always present, the respiration varying from 30 to 70 per minute, according to the extent of involvement and still more to the severity of the toxemia, and pleuritic pain may cause a grunting respiration and shallow breathing. Grunting.

(*g*) **Heart and Pulse.**—*The pulse is full and bounding, but, as in typhoid, relatively slow, and the pulse-temperature ratio and condition of the heart sounds are important factors in prognosis.* Prognostic signs.

If the heart is acting properly, the pulmonary second sound is strongly accentuated, and its marked diminution or absence, in the presence of persistent or advancing consolidation, points to right-sided cardiac dilatation and impending failure. Acute dilatation.

The Blood.—In lobar pneumonia, pneumococci may be recovered from the blood in considerable proportion of cases (30–50 per cent.) and *leucocytosis* roughly measures the reciprocal action of toxemia and resistance. Its absence is ominous or may be suggestive of coincident infections other than that due to the pneumococcus, *e.g.*, influenza or typhoid fever. The average number of leucocytes is about 25,000 per cu. mm., and counts between 30,000 and 50,000 are not uncommon. The significance of a failure of leucocytosis is somewhat diminished in *complicating pneumonias* such as those associated with typhoid or smallpox, diseases in which the vitality of the patient may be greatly lowered.* Leucocytosis the rule.

The leucocyte count ordinarily reaches normal within two or three days after crisis and its undue persistence suggests septic complications. Persistent leucocytosis.

The Urine.—*Albuminuria is common but is usually of the febrile type, subsiding promptly with the termination of the active process. The urinary chlorides are diminished or absent during the active stage of the disease and the urine is scant, dark, and of high specific gravity.* Albumin and chlorides.

Gastrointestinal Tract.—The tongue is ordinarily furred, vomiting or diarrhea may be present, and meteorism may greatly embarrass circulation and respiration. Meteorism.

Headache and Delirium.—Severe headache may be present at the onset or more rarely throughout the patient's illness. Delirium is unusual, the patient being ordinarily rational and acutely sensible of his situation. Either low or active delirium is ominous. Maniacal delirium occasionally occurs, and delirium tremens frequently develops in alcoholic cases.†

Physiognomy of Pneumonia.—If the patient be conscious, the expression is anxious, eye bright, the attitude active, and the decubitus usually lateral, the affected side being most often undermost during the early stages. Herpes appears on the lips in a majority of cases‡ and *dyspnea* is evident both in the chest movement and the working nostrils. Both cheeks may be flushed, or Herpes. Flush.

* From clinical experience the author would advise special care in the lobar pneumonia of arrested or apparently cured tuberculous cases. Many of these experience sudden collapse after a few days' illness and often at the time of apparent crisis.

† Too frequently the pneumonia is overlooked in such cases.

‡ Herpes occurs frequently not only in pneumonia, but in all other infections in which the pneumococcus plays a chief part.

that of the affected side alone and more or less cyanosis is always present.

Cyanosis.

Deepening cyanosis should be carefully watched for and met by appropriate treatment directed to the circulation. If the nails become deeply cyanotic the end is usually near.

PHYSICAL SIGNS OF FRANK LOBAR PNEUMONIA.—Inspection.—Aside from the physiognomy and decubitus one notes *lagging* or *immobility* of the *affected side* as a whole or chiefly over the lower zone if the base only be involved, together with an increased rate of respiration and overaction of the accessory muscles of respiration.

Lagging.

Palpation.—Confirms inspection and in established consolidation *if fremitus is tested after cough* (to remove obstructive secretion) *it will be increased* unless the larger bronchi are filled with fibrinous exudate (massive pneumonia).

Increased fremitus.

If only the engorgement of the early stage is present, fremitus may be normal or actually decreased. Pleural friction may be felt in some cases and palpatory percussion reveals marked resistance over the affected area.

Percussion.—*Dulness with well-marked resistance* is the sign of *consolidation; hyperresonance or tympany*, that of *primary engorgement*, and in early or doubtful cases percussion may be made while the patient is lying prone, or, *if deemed safe*, sitting up and leaning forward to avoid confusing differences due to the unequal thoracic pressure involved in the lateral position.*

Central pneumonia.

Percussion may be wholly negative in central pneumonia for several days, but the note is so frankly dull, ordinarily (when hepatization is fully established), as to require no decided shifts of posture and leave no doubt in the mind of the examiner. Equally frank is the hyperresonance above the dull area (advancing infiltration—vicarious emphysema—impaired tonus) which *may even simulate the cavity percussion note* in quality and *variations*, especially in cases of apex pneumonia. In a case of lobar pneumonia in its early stages, *i.e.*, within twelve to twenty-four hours after the initial symptoms, *the affected area itself sometimes shows a most misleading hyperresonance.*

Hyperresonance of advancing consolidation

Furthermore, it may show resonance anteriorly long after dulness is demonstrable posteriorly.

Auscultation.—*In the first stage, or in massive pneumonia, the breath sounds may be diminished or even suppressed and, in the former, one should seek to hear the true crepitant râle at the end of inspiration.*

Value of crepitation.

As the consolidation advances and nears the surface the intensity and bronchial character of the sounds increase pari passu until exquisite, dry, tubular breathing is audible over the area of consolidation however varied may be the râles over adjacent areas or in the opposite lung.

Voice sounds best guide.

The voice sounds may be normal or diminished early in the case, but, as a rule, together with fremitus, give better and prompter news of an advancing process than do the breath sounds and ultimately yield the pure bronchophony of

*It is seldom necessary to subject a highly toxic heart muscle to the strain involved in the forced assumption of the sitting posture. As stated previously, the patient who is too ill *to sit up* is too ill *to be set up*.

superficial consolidation. As in palpation, if the signs are obscure, the patient should cough to clear the bronchi before auscultation is completed.

In the stage of resolution there is a rapid recession of the pure tubular breathing and voice conduction, associated with the reappearance of fine crepitation (often missed by the physician), shortly succeeded by coarser moist râles, together with diminishing percussion dulness and fremitus.

Diagnosis.—*The diagnosis of lobar pneumonia, usually simple,* depends upon the *frank, febrile* onset and high fever,* pulse-temperature ratio, rusty sputum and physical signs, and the sputum is often characteristic when physical signs are lacking (central cases). Blood cultures may be positive.

Value of sputum.

The chief confusing conditions are: (*a*) **afebrile senile pneumonias** in which the physical signs and toxemic symptoms must govern.

Misleading conditions.

(*b*) **Cases of pleural effusion with transmitted tubular breathing,** which are differentiated by the flatter percussion note, displacement of the heart, more distant quality of the tubular breathing, relatively slight fremitus, *and, positively,* by the results of exploratory puncture.

Exploratory puncture.

(*c*) **Cases of acute pneumonic tuberculosis,** in which an existing or pre-existent lesion or a tainted family history may be suggestive, but which can often be differentiated only by the subsequent course, evidences of a destructive process and the recovery of the tubercle bacillus from the sputum.

(*d*) **Hypostatic pneumonia occurring in a febrile disease,** in which case the primary condition itself usually suggests the probability of hypostasis which is often incomplete, usually bilateral, markedly and persistently dorsal, and is in correspondence with and dependent upon the decubitus of the patient and the heart strength.

Lobar Pneumonias Associated with Mixed Infections.—It is well known that lobar pneumonia may be associated with a variety of pathogenic organisms but the student should remember that mixed forms almost always lack both the typical general symptoms and the frank physical signs of the pure or dominant pneumococcus form. They tend, on the other hand, to a suggestive irregularity of course and development.

Broncho-pneumonia is wholly different as to course, sputa, and physical signs, save in the exceptional cases of extensive fusion of broncho-pneumonic areas, and in these the course, and the associated and *antecedent* pulmonary signs are usually sufficiently distinctive.

Usually unlike.

Infarct.—*When, as rarely happens, an infarct is of sufficient size to simulate lobar pneumonia and is accommodatingly typical, it is usually suggested by the nature of the primary disease with which it is associated, by its instantaneous onset, its sharply limited area of involvement and a bright bloody sputum or actual hemoptysis.*

Central Pneumonia.—*This may not be positively diagnosed until rusty sputum appears or the signs become evident from superficial consolidation,* but as a matter of fact, is to be recognized usually, even in its larval forms, before these evidences appear. Its *onset,* the more or less decided leucocytosis, and

Usually detected.

* The continuous type of the fever is usually emphasized but many cases occur in which it is markedly remittent.

the *suggestive* though not clean-cut *localized departures from normal physical signs are usually sufficiently suggestive.*

Important diagnostic data.

As previously stated one must lay much stress upon *localized areas of hyperresonance* in such cases, especially if these stop abruptly at an interlobar division and, furthermore, increased fremitus and voice sounds together with faint distant tubular breathing are frequently to be made out.

RATIONALE OF LOBAR PNEUMONIA

Signs and Symptoms.—*The physical signs and the symptoms of frank lobar pneumonia are in precise accord with the fundamental factors underlying their production in any disease of the lung.*

Fever and leucocytosis.

High-grade Toxemia.—From the known nature of the infectious agent (pneumococcus) one anticipates a profound toxemia, a high and relatively stable fever and, in persons of average resisting power, a well-defined reactive and defensive leucocytosis.

Color, viscidity and tenacity.

Sputum.—The extreme congestion and inflammation, and the fibrinous character of the exudate, readily explain the color and peculiar viscidity and tenacity of the sputum.

Disturbed tonus.

Stage of Congestion.—The obscuration of physical signs in the early stage of congestion and edema is manifestly due to the diminution of normal pulmonary tonus combined with some disturbance of tissue homogeneity; obviously this would apply to any point of inflammatory, central pulmonary consolidation or the line advance of an extending superficial consolidation.

Increased conduction.

Bronchial and Tubular Breathing.—The bronchial and tubular breathing, increased fremitus, and bronchophonic phenomena, of the stage of complete or decided consolidation, reflect the extraordinary conducting quality of dense, airless lung in direct communication with patent bronchi and through these with the trachea.

Ideal conditions present.

The Crepitant Râle.—The crepitant râles must, of course, result from the presence of the very conditions which we know to be ideally adapted to their production, viz.—a viscid glutinous secretion present in the alveoli and infundibula at the period of marked congestion and beginning consolidation, and to a less degree, at the period of recession represented by the commencement of resolution (crepitus redux).

At a certain stage in each of these periods the forcible indraught of air can separate the agglutinated walls at the height of pressure, *i.e.*, the end of inspiration.

Massive pneumonia.

Blocked Bronchi.—If the bronchi of any area are completely blocked by exudate, as rarely happens, the conduction is abolished or greatly diminished; hence areas of massive pneumonia are silent, though yielding the percussion signs and constitutional symptoms of the disease and lacking, of course, the displacement symptoms and usually the absolutely flat note of liquid exudate.

Toxemia the Dominant Factor—The dominant importance of the associated toxemia in prognosis and treatment rather than the degree of lung

involvement, and the effect of the toxin and of the impeded pulmonary circulation upon the right heart must ever be in mind.

Furthermore, the value, in prognosis and treatment, of accurate observation of the changes in force and accentuation of the pulmonary second sound, a careful delineation of the accessible heart borders, and a critical study of the other heart tones cannot be overestimated.

Cyanosis and Dyspnea.—The phenomena of cyanosis and dyspnea are self-evidently the results of a process which suddenly wipes out a considerable capillary area in the lesser circulation, diminishes the air exchange poisons the heart muscle, and overstimulates the respiratory center.

Crisis.—The changes wrought in the sputum and physical signs by crisis and resolution are so directly in line with the pathology as to need no extended comment.

Complication.—Acute arteritis is an extremely rare complication.*

PROGNOSIS.—Pneumonia is, as stated previously, an extremely fatal disease and kills from 10 to 70 or even 80 per cent. of the cases affected, according to the virulence of the type and the age and vitality of the individual.

Public vs. private patients.

In a high-class private practice at least 10 per cent. of all cases die. In general hospital practice the mortality will vary from 20 to 40 per cent. according to the type and age of the patients. It is said that in patients above sixty years of age two-thirds will die.†

Important factors.

The outlook is bad at the extremes of life, in the obese, in those of bad habits and in such as are debilitated by overwork, unfortunate sanitary environment or chronic disease.

The tuberculous experience a high mortality, and recurrent cases are somewhat more dangerous than primary attacks.

Influenzal pneumonia of the lobar type is especially fatal, though sometimes peculiarly transient and most atypical in its localization and duration.

Ominous signs.

Excessive rapidity and arrhythmia, both as affecting pulse and respiration, are ominous signs, as also are extreme cyanosis, absent or markedly diminished pulmonary second sound, extending heart borders, profound albuminuria, low leucocyte counts, and marked delirium.

Vital element.

The essential element in prognosis and treatment consists in the appreciation of the fact that one is dealing with a toxemia, to which all local manifestations are secondary, and that hope lies in assisting the patient to resist its virulence, and in guarding the poisoned, overburdened, myocardium.

Intensely septic in course.

SEPTIC PNEUMONIA.—This *extremely fatal* type is characterized by the intensity and septic type of its manifestations, by a purulent sputum, often most profuse, and a tendency to destruction of the lung through the production of multiple abscesses or gangrene. *Each form may be either broncho-pneumonic, the commoner type, or more or less atypically lobar.*

* George Douglas Head; Amer. Jour. Med. Sci., August, 1921, vol. clxii, p. 157.

† So many variations enter into both private and public practice that arbitrary figures become ridiculous and our judgment must depend upon a knowledge of the favorable or unfavorable factors in the individual case. In general, public patients enter the hospital late and in bad condition.

Three forms.

Three types are encountered which differ little save in causation:

(1) *Aspiration pneumonia;* (2) *sepsis by extension;* (3) *metastatic or embolic.*

Aspiration pneumonia is that caused by the entrance of foreign bodies or food particles.

The septic form due to extension is encountered in certain cases of hepatic and subphrenic abscess or empyema.

The metastatic or embolic form may, as its name implies, arise from almost any septic infection, is distinctly hematogenic and usually begins abruptly with more or less well-defined symptoms of infarction. It is quite unlike lobular pneumonia and lacks primarily the rusty sputum and *immediate* temperature rise of the true lobar form.

BRONCHO-PNEUMONIA

(*Catarrhal pneumonia, lobular pneumonia, capillary bronchitis.*)

Definition.—*An acute infection associated with profound toxemia and an inflammation of the terminal bronchioles and lobular tissue of the lung.*

Mixed infection.

Etiology.—No special germ has been identified, but the diplococcus pneumoniæ is most often found, usually in connection with other germs, such as the pyogenic streptococci, Friedlander's bacillus, and, more rarely, Klebs-Löffler and influenza bacilli.*

Mixed infections are the rule though pure cultures of the pneumococcus may be found in the primary and pseudo-lobar forms.

The general predisposing factors are essentially the same as in lobar pneumonia. Seventy-five per cent. of all the pneumonias in children are of this type and of these about two-thirds follow bronchitis. It is very common in whooping cough, influenza, diphtheria, in the aged and, as a complication, in various acute and chronic diseases.

Inhalation Pneumonias.—*Any irritant, as irritating fumes or dust or the vapor of anesthetics may excite it, in the presence of pathogenic germs.*

Morbid Anatomy of Lobular Pneumonia.—The first stage is one of intense congestion and the capillary bronchiolitis and alveolar congestion is followed by a serofibrinous or hemorrrhagic exudation associated with pronounced desquamation of epithelium. Ordinarily an antecedent bronchitis extends to these structures, the bronchioles become plugged, the alveoli gradually lose their contained air and collapse. *The exudate, ordinarily termed catarrhal, is sometimes distinctly fibrinous,* but its cellular constituents show a predominance of epithelium over leucocytes. The process is bilateral, affecting chiefly the lower lobes posteriorly; the lung appears mottled with bluish-brown airless depressions indicating collapsed areas, interspersed with bright red emphysematous and crepitant fields. Fusion of areas may bring about extensive consolidation or the affected portions may remain isolated and are sometimes so minute as to constitute an *acute miliary broncho-pneumonia.* Collapsed areas may be distended by the blowpipe, the lungs are larger than

Bronchiolitis.

Exudate.

Bilateral.

Fusion of areas.

Miliary form.

* The peculiar type of pneumonia proving so fatal a complication of influenza during the epidemic of 1918 was associated with the presence of the influenza bacillus in most instances.

normal, have a nodular feel, and drip on section and the *process* of resolution is much the same as in lobar pneumonia, but, save in the acute cases, is unfortunately more irregular in its points of development so that the disease ordinarily terminates in protracted lysis.

Two Chief Divisions of Broncho-pneumonia.—The disease may be divided into primary and secondary cases. The former come on acutely, attacking primarily the terminal bronchioles and terminate, as a rule, quite abruptly.

Primary vs. secondary.

The latter are invariably subsequent to a bronchitis or complicate some existing disease.

Primary Broncho-pneumonia.—*The primary type is infrequent in patients over four years of age, but constitutes about one-third of the cases in children and is almost always a distinct pneumococcus infection, as might be inferred from the abrupt character of its onset and termination. The primary congestive form may kill in from twelve to twenty-four hours or may terminate favorably in a few days.*

Affects especially children under four.

Primary congestive form.

Secondary Broncho-pneumonia.—Most broncho-pneumonias are of the long-persisting secondary type and it is a common sequel of the bronchitis of children and certain of the acute exanthemata, especially measles, scarlet fever and diphtheria,* and as a terminal event in cachexias and senile conditions. In the latter, its course may be insidious and lacking in the franker clinical manifestations. *In measles and scarlet fever* it is severe and protracted, in *diphtheria* extremely fatal. In influenza one meets with two types, one extremely brief, the other protracted and severe. The latter form is peculiarly liable to leave behind it a persistent spasmodic cough.

A serious complication.

Persistent cough.

Next to gastro-enteric diseases, broncho-pneumonia is the most common and certainly the most fatal of the more serious and common diseases of infancy.

SUBDIVISIONS.—Aside from the general division into primary and secondary cases, we recognize three forms of the disease itself:

1. **An acute congestive broncho-pneumonia.**
2. **Disseminated broncho-pneumonia,** often described as capillary bronchitis, and
3. **The common form**† **of the disease.**

Ninety per cent. of the acute "congestive" and "common" types are bilateral, and the disseminated form invariably so.

4. **Acute hemorrhagic pneumonitis.** (See under Influenza.)

Selective Points.—*In the acute congestive and the common form, the signs are always most marked posteriorly and nearly always in the interscapular space.*

Interscapular space.

Pulmonary at Onset.—Save in the acute congestive pneumonias, the symptoms and physical signs are *strikingly pulmonary* from the outset, though sometimes obscure, atypical, and silent as regards actual physical signs, save for significant multiple areas of hyperresonance, confusing and

Sometimes silent.

* Councilman states that broncho-pneumonia is seldom absent in the severer types of this disease.

† Following Holt's classification.

wholly out of accord with the wide involvement shown by autopsy in fatal cases.

Explained by pathology.

The nature of the pathologic process explains this fact by showing that even where consolidation exists the condition of the surrounding tissues may be such as to produce a hyperresonance which masks the percussion signs of consolidation.

Often malignant.

ACUTE CONGESTIVE BRONCHO-PNEUMONIA.—Symptoms.—*This may be rapidly fatal, killing in twenty-four to forty-eight hours, and the physical signs may be limited to harsh breath sounds and possibly a slightly dulled percussion note.*

The signs are usually bilateral, but not necessarily of equal intensity over the two sides.

The onset is sudden, often with vomiting and occasionally with convulsions in children or chill in the adult.

The fever runs high and may steadily increase to a fatal termination (103° to 107°).

Cough may be entirely absent or violent and harassing.

Toxemia profound.

Pulse and respiration are extremely rapid, cyanosis marked, mental dulness and apathy early and extreme and the attack may merge into the common form of the disease or terminate sharply by crisis.

Miscalled capillary bronchitis.

ACUTE DISSEMINATED BRONCHO-PNEUMONIA.—This form which some of the foreign clinicians still term "capillary bronchitis" is primarily an inflammation of the capillary bronchi; but, post-mortem, is invariably associated with areas of true broncho-pneumonia.

Abrupt.

Symptoms.—*Like the other forms, it comes on acutely, pulse and respiration are rapid, prostration is marked but not immediate and both dyspnea and cyanosis*

Low fever.

are prominent. The cough is severe and harassing though not sharply painful, but the temperature usually runs low.

Basic signs.

The physical signs *consist chiefly of the subcrepitant and coarser râles, generally distributed, always bilateral, and best marked in the interscapular space. The percussion note is hyperresonant or even tympanitic.*

Recovery is the rule and the disease ordinarily runs a short course of a few days or a week or ten days, terminates by lysis, or merges into the common form of the disease.

THE COMMON FORM OF BRONCHO-PNEUMONIA.—Onset.—

Abrupt onset usual.

Remittent fever.

Although often preceded by a bronchitis, the actual onset is often abrupt; the fever high at its maxima but, with rare exceptions, strikingly remittent (often showing a variation of three or four degrees).

Vomiting *sometimes marks the onset and very often complicates the established disease. Convulsions are infrequent as an initial event.*

Cough *is early, intractable and persistent, harassing, but usually painless.*

Early rapid respiration.

Dyspnea.—*The respiration is rapid* (60 to 80 *or even* 120 *in severe cases*) *and may be noted as a premonitory symptom even before the frank onset.*

Dyspnea.

The signs of actual dyspnea are pronounced; the alæ nasi active and inspiratory recession of the lower interspaces marked.

Prostration *is marked and soon becomes extreme and expectoration may be wholly absent in young children.*

Cyanosis *becomes marked in all severe cases and convulsions may occur as one of the terminal events.*

In rare instances infants or badly nourished children may show a subnormal temperature.

The pulse may reach 200 and *is always rapid* and sometimes irregular. Rapid pulse.

Gastrointestinal disturbances greatly complicate the treatment in many instances, flatulence and exhausting diarrhea or persistent vomiting aggravated by cough being the most troublesome factors.

The urine is of the febrile type, scant, high colored, and containing an excess of urates and a trace of albumin.

The ordinary course is from two to three weeks and even in favorable cases it usually terminates in a tedious lysis. Cases may, however, be prolonged for two or three months or a chronic broncho-pneumonia may succeed the acute form and often proves to be a tuberculous process. Termination.

PHYSICAL SIGNS OF THE CHIEF TYPES OF BRONCHO-PNEUMONIA.—*These are primarily the signs of an acute bronchiolitis, the most characteristic sign being the subcrepitant râles heard chiefly and predominantly over the lower lobes in the interscapular spaces.* Subcrepitant râles.

Unlike lobar pneumonia, the left lung is usually chiefly affected, though both are involved. Left lobe chiefly.

Bronchial râles, dry and moist, of a larger and coarser character, may mask the finer crackles, and after a variable period evidences of consolidation may appear over scattered areas or, more rarely, produce extensive consolidation.

Inspection.—*Inspection reveals the facies of profound illness, and its pulmonary nature is indicated by the evident dyspnea, harassing cough, slight or marked cyanosis and inspiratory recession of the lower interspaces.* Facies.

Palpation.—*Fremitus is negative or impaired in the congestive type and diminished in the disseminated form, but may be increased over certain areas in the "common form" if consolidation be sufficiently extensive and superficial.* Fremitus.

Percussion.—*Percussion is negative in the congestive form or shows only slight dulness and there is hyperresonance or tympany in the disseminated variety. In the common form scattered areas of impaired resonance or decided dulness may alternate with hyperresonance and, furthermore, certain areas of dulness may disappear and reappear from day to day or hour to hour.* Vague signs. Shifting signs.

Light percussion should invariably be practised and it should be remembered that consolidation areas are first and chiefly evident in the interscapular spaces save in those instances, *usually influenzal or tubercular*, in which the localization is anterior and apical. Light percussion.

Auscultation.—*Auscultation is by far the most valuable procedure. The characteristic subcrepitant râles are usually overshadowed by their coarser congeners both dry and moist, and in their turn tend to obscure the crepitant râles often intermingled with them.* Most valuable procedure.

Areas of increased voice conduction and broncho-vesicular or even tubular breathing may make any existing consolidation manifest to the ear even though it is not revealed by the percussing finger. Importance of voice sounds.

Friction sounds are rare and it should never be forgotten that all signs may Friction rare.

Crying assists.

be lacking save in forced deep inspiration, in children often obtainable only during the act of crying.

Remember that in all cases of broncho-pneumonia the patient is manifestly too sick for the simple bronchitis which the disease may simulate in its early stage or directly follow. (See also discussion of the peculiar complicating pneumonia of Influenza, p. 386.)

Complications.—Ileo-colitis is dangerous and troublesome when it occurs. Meningitis and nephritis are rare, tuberculosis is not uncommon and is undoubtedly often primary, especially in the complicating broncho-pneumonias of measles and pertussis. Endocarditis is fortunately a clinical curiosity. Suppuration and abscess formation are rarely seen save in aspiration and deglutition cases.

Character of exudate.

RATIONALE OF BRONCHO-PNEUMONIA.—The chief distinctions between lobar and broncho-pneumonia lie manifestly in the character of the exudate, which is catarrhal rather than fibrinous, and in the fact that the bronchioles and peribronchial tissue bear the brunt of the attack.

These facts greatly assist one in understanding the peculiarities of its physical signs.

Dominant rale.

Under such conditions of catarrhal bronchiolitis the subcrepitant râle must be the dominant one in auscultation, and with blocking of the lumen of the bronchioles comes atelectasis of certain lobular areas through absorption of the trapped air content and consequent collapse of the vesicles.

With localized peribronchial and perivesicular inflammation, relaxation and edema in the intermediate areas must occur and affect those fields in which vicarious overdistention or emphysema exists.

Atelectasis.

Fusion of areas.

Furthermore, the blocking of any lobular inlet by the relatively thin *catarrhal* exudate may at any time be relieved and the tributary territory again become aërated or, *per contra*, by aggregation of involved areas, nearly or quite all of the lobe itself may become airless and a true lobar pneumonia be simulated.

Hyper-resonance.

Shifting signs.

"Patchy" distribution.

Hence the indeterminate, shifting medley of physical signs—hyperresonance over relaxed or emphysematous areas; adjoining areas of dulness of varying degree, situation, and persistence; hyperresonance masking and perhaps modified by underlying dulness, and the somewhat characteristic limitation of signs to *patches* of territory.

Diagnosis seldom difficult.

Despite the seeming confusion and vagrancy of signs the diagnosis of the common form is not often really difficult.

ATELECTASIS AND THE PULMONARY CONGESTIONS

Trivial or "massive."

Definition.—A condition due to blocking of air passages, deficient respiratory action or lung compression and characterized by localized or *extensive* loss of the pulmonary air content and complete collapse of the vesicles in the area affected, whether temporary or persistent.

Physiologic type.

Etiology.—*Usually Secondary.*—Aside from the atelectasis so frequently present at the lung bases in shallow breathers, the weak, and the bedridden,

which is ordinarily abolished by a few long breaths, or that encountered sometimes in the insufficiently inflated lungs of the new-born child, it results from extreme respiratory weakness or actual obstruction of the bronchi, large or small.

Fundamental causes.

As examples one may cite broncho-pneumonia, compression of the lung by tumors, foreign bodies, or the pressure of growths or exudates, *e.g.*, mediastinal or pleural growths, pleural effusion, pneumothorax, greatly enlarged heart and pericardial effusion.

Obstruction and compression.

Slight grades result from mere deficient expansion in delicate women or children and higher grades from chronic obstruction in the upper respiratory tract, as from adenoids or enlarged tonsils. "Pigeon breast" and "Harrison's groove" represent chest deformities due in part to such a condition. In extreme cases the lung is carnified, and persistent and extensive atelectasis leads to atrophy or fibrosis.

Adenoids, and the like.

Carnification. and fibrosis.

Examples of this last change occur frequently in cases of prolonged pleural exudation especially of the purulent type, in which the lung may fail to reinflate.

Massive collapse of the lung following chest injuries with or without perforation of chest wall is rare in civil practice but occurred quite frequently during the Great War.

It may affect the side opposite to that receiving the direct injury and produces symptoms easily mistaken for pleurisy or pneumonia if the following points are not borne in mind.

(*a*) The chest is *retracted* over the collapsed lung.

(*b*) The heart is *displaced toward* the affected lung.

Radiography or fluoroscopy would show high position of the diaphragm on the affected side.

Diagnosis.—*The presence of an adequate cause, inspiratory recession of interspaces or actual retraction, absent or distant tubular, breath sounds and corresponding fremitus variations may be present.*

Atelectatic inspiratory retraction.

These are for the most part silent or hushed areas, though in cases of broncho-pneumonia deep inspiration or cough may clear the bronchi and convert them into noisy ones.

Hushed areas.

Variable areas.

In atelectasis of this silent type the condition is very different from that which results from continuous obliterative or stenosing pressure upon a bronchus, as in aneurysm or malignant growths, or from such extreme and long-enduring compression as may occur in pneumothorax or pleural effusions. In the latter instances the whole lung may be airless and carnified, yet directly in connection with the primary bronchus, and may transmit sound and yield dulness like any other consolidated lung.

Important form.

Deceptive cases.

Fine crepitations heard over the lung bases at the end of inspiration and persisting after one full inspiration are of significance as indicating the onset, increase or persistence of myocardial insufficiency.

CONGESTION OF THE LUNGS.—Active congestion and the three forms of passive congestion, in which is included so-called pulmonary edema, will be discussed under this head.

Irritation.

ACTIVE CONGESTION.—The inhalation of irritating substances, chiefly vapors and gases, and the primary stage of the pneumonias, are the chief factors in the production of an extensive, active congestion and it is almost invariably but a stage in some dominant disease process.

Doubtful distinctions.

PASSIVE CONGESTIONS, HYPOSTASIS AND EDEMA Dubious Distinctions.—Sharply drawn lines between obstructive and hypostatic congestion and between these and pulmonary edema are unfortunately largely confined to the autopsy room.

Venous stasis.

Toxemia.

Identical Fundamental Etiology.—*Between the former two* there is little need of distinction, for both arise from an obstructed venous circulation whether from valvular disease, a weakened myocardium, toxemia, the pressure of tumors, a pleuritic or pericardial exudate, abdominal distention, or cerebral lesions.

No dividing line.

Decubitus a localizing factor.

Clinically, the hypostatic form as distinguished from the simple obstructive cardiovascular congestion is encountered in conditions of profound exhaustion, especially such as are characterized by the typhoid state. It represents oftentimes a secondary circulatory weakness and extreme asthenia often associated with a more or less profound toxemia. Both forms are markedly affected by the decubitus of the patient.

Pulmonary Edema.—*Edema accompanying acute pulmonary congestion or inflammation is termed "infectious" or "inflammatory" as opposed to the ordinary edema of the lungs associated with passive congestion.*

This latter in its fulminant form is a terminal event, or paroxysmally recurrent, in many diseases and may both come and kill suddenly and without warning or supervene upon prolonged congestion. In certain cases it seems to be purely toxemic but fundamentally its etiology is that of the associated congestion.

Morbid Anatomy of Passive Congestion.—Long-continued passive congestion may produce brown induration, the organ being dense, bulky and russet brown; its capillaries are distended, the bronchioles and alveoli filled with epithelium, blood cells and pigment, and there is connective tissue increase. This represents especially the obstructive congestion typified in incompensated mitral lesions.

In the hypostatic form the engorgement is intense and portions of the lung sometimes airless. If these areas are extensive the term *splenization* is applicable.

In pulmonary edema the bulky lung is sodden and the foamy yellow or reddish serum infiltrates the interstitial tissue and fills the air passages.

Any of the forms of passive congestion *may* exhibit unilateral predominance of involvement, usually postural, *but they are usually bilateral.*

Slight symptoms.

Symptoms.—*The milder types of all varieties of pulmonary congestion may produce but slight subjective and objective symptoms.*

The condition invariably demands cardiac stimulation.

Hypostasis often overlooked.

In cardiac disease, for example, the condition may not attract notice until here is marked dyspnea and a blood-streaked sputum containing the peculiar pigment-bearing cells.

A careless practitioner often entirely overlooks chronic *hypostasis* until it has reached a pneumonic stage (splenization). Less frequently mild forms of edema occur which may be fugitive and transitory. Fugitive edemas.

In the severer forms of passive congestion, dyspnea and cyanosis are present and in marked edema are excessive.

In all there is restriction of chest movement pari passu with the area involved.

Fremitus may be diminished or increased, according to the degree of relaxation on the one hand or actual consolidation on the other.

The percussion note may be hyperresonant from mere congestive relaxation or dull from consolidation.

Auscultation may reveal harsh, diminished, absent or tubular breathing according to the stage of congestion, the presence of atelectasis or the establishment of true consolidation. Variable physical signs.

Râles, both dry and moist are present, their pitch and quality varying with the pathologic conditions.

In established, severe edema the objective and subjective symptoms are pronounced, the sputa being profuse, frothy, serous or blood-stained and in inflammatory edema presenting the "prune-juice" appearance. In established edema, moreover, the râles are peculiarly and frankly liquid, the breath sounds and voice conduction being usually suppressed or feeble. Marked subjective and objective signs. Liquid râles.

Important Comment.—*Slight* **persisting** *crepitation at a base in a nephritic subject, or in any patient suffering from the diseases associated with passive congestion, demands serious attention though it may persist over long periods without critical symptoms.*

Astonishingly sharp congestions may accompany obscure cerebral apoplexies unassociated with persisting paralysis or loss of consciousness. Congestions of apoplexy.

In a case of this type recently observed two cerebral hemorrhages of the silent type occurred without loss of consciousness or persistent loss of power, the only symptoms being sudden pallor, mental confusion, transient motor weakness, *acute cardiac dilatation*, and, in the second attack, transient motor aphasia. In each there was marked pulmonary congestion lasting for about twelve hours. A third hemorrhage ten days later produced almost instant death.

Many similar cases have been observed, one just at the time these words are written. The recovery of the pre-existing heart outline is usually prompt, even though direct cardiac stimulation is not employed. This is true, however, only in the event that absolute rest is enforced.

Cardiac stimulation, or, in hypostasis, mere attention to the necessary shifting of a patient's position may rapidly and markedly diminish the involved areas, the latter condition being often due to a weakened circulation plus a fixed posture, in senile, obese, cachectic or profoundly toxemic patients.

TUBERCULOSIS OF THE LUNGS

(*Pulmonary Phthisis, Consumption*)

Definition.—*An infectious disease caused by the tubercle bacillus of Koch, usually chronic, rarely acute, characterized by the formation of tubercles or diffuse*

infiltration of tuberculous tissue and tending to acute general dissemination, ulceration, fibrosis or calcification.

Distribution and Cost.—It is a universal disease visiting practically all races and every latitude. In the United States alone it kills at least 110,000 persons each year, and the annual loss in potential wealth has been estimated at $500,000,000. It costs the life insurance companies of America at least $6,000,000 annually, and as the deaths occur chiefly at early ages when few premiums have been paid, it constitutes the source of their greatest early loss.

It ranks second only to heart disease and pneumonia as a cause of death and has dropped from the first place only during the past decade.

Susceptibility.—Domestic animals and pets, such as cattle, pigs, guinea-pigs, rabbits and monkeys are extremely susceptible. Horses, dogs, goats and cats are much less vulnerable.

Climate.—Cold, damp, densely populated districts suffer most, and dry and high altitude regions least.

"Koch's Law" fulfilled.

MORBID ANATOMY.—If a pure culture of tubercle bacilli be injected into the tissues of a susceptible animal it produces tuberculosis. From the tuberculous tissues the germ can be recovered, grown in pure culture, again used for inoculation and so on indefinitely. This absolute proof of its infectious nature was established promptly and made the position of those fighting the "great white plague" unassailable.

Changes Subsequent to Inoculation.—(*a*) *Grouping of germs in tissue by multiplication, and their distribution by the lymph, or more rarely, blood-current.*

(*b*) The formation of epithelioid cells, about five days after inoculation, by multiplication and by metamorphosis of the cells of the capillary endothelium and connective tissues.

(*c*) Outwandering of leucocytes to focus of infection and multiplication of mononuclear forms.

(*d*) The formation of a marginal reticulum of connective tissue.

(*e*) The production of large epithelioid cells containing from four to twenty nuclei grouped at the poles or periphery.

These giant cells are most common in lupus and in glandular and bone tuberculosis and the more chronic and slowly developed the process, the greater is their number. In acute lesions they are scant and absent, nor are they peculiar to tuberculosis, being found in other of the granulomata such as syphilis.

Caseation and Sclerosis.—The changes described tend inevitably to devitalize the affected tissue and produce areas of central coagulation necrosis converting all structures into a cheesy homogeneous substance. This may be extruded, leaving cavities of greater or lesser size, or, more rarely, calcification may accompany encapsulation. Many cases terminate spontaneously without reaching caseation and often without yielding any recognizable symptoms.

Slow vs. rapid.

The Spread of Infection.—It will be readily understood that lymph distribution of the usual type means a slow process; infection through the blood

stream or the *thoracic duct*, a rapidly developing and widely disseminated process.

ETIOLOGY.—Race.—The Hebrews are to a certain extent immune, but in this country the Irish and the Scandinavian, the Indian and the negro show a heavy mortality.

Sex.—Its incidence is about equal in the two sexes.

Age.—Children are peculiarly prone to affections of the glands and bones, but after the age of childhood the lungs are chiefly affected and susceptibility diminishes considerably at the age of 30, although the mortality remains heavy even in the advanced age periods.

During the period between the fifth and fifteenth years relatively few deaths occur. From the age of six months to twenty-four months the heaviest death rate occurs.

Occupation.—Occupations involving exposure to extremes in temperature, dust and dampness are distinctly unfavorable. Workers grinding glass or steel or handling furs show a high mortality.

Sanitary Conditions.—Lack of sunshine, fresh air, overcrowding, filth and physical exhaustion are all predisposing factors. Among the tenement classes all conditions are favorable to its development and transmission. Common factors.

Preëxisting Diseases.—Tuberculosis frequently follows chronic disease of the tonsils, adenoids, neglected colds, broncho-pneumonia, lobar pneumonia, pleurisy, influenza, measles, whooping cough and enterocolitis, and may complicate diabetes, aneurysm, locomotor ataxia, sclerosis of the liver or chronic Bright's disease, any condition in short reducing vitality and resistance or tending to prevent free lung expansion. Predisposing factors. Diminished resistance.

Injuries.—Tuberculosis of the bones and joints and especially of the vertebra and hip-joints in children is frequently ascribed to injury, though due no doubt to a preëxisting latent infection.

Heredity.—Direct transmission *in utero* (placental tuberculosis) is probably extremely rare, and heredity is but another name for post-partum implantation of the germ upon a fallow soil which is usually that constitutional defectiveness of structure and tendency to unstable and inadequate function known as "congenital asthenia". The tuberculous mother readily transmits germs to her offspring and a neglect of sanitary precautions in any infected house is peculiarly dangerous to the children. The germs may remain latent in the glandular system for an indefinite period, a fact undoubtedly explaining the increased adult mortality from tuberculosis in families showing the taint. Post-partum infection. Often latent.

Individual Predisposition.—The "*habitus phthisicus*" has long been recognized, certain individuals being from birth less resistant to the germ than are others. Small bones, a delicate complexion, and a contracted chest, long sweeping lashes, large lustrous eyes and silky hair are found in one type; a muddy complexion with marked tendency to glandular swelling, acne and weak eyes mark the other type. Extraordinary beauty, unusual intelligence and a quick wit were recognized even by the ancients as qualities often associated with tuberculous predisposition. The children of either type fre- Well-known type.

quently represent undoubtedly cases of latent infection and need unusual care along hygienic lines. Having this they may grow up to manhood strong and well and die of old age.

May escape disease.

Chronic Congenital Asthenia.—This condition is closely allied to if not identical with the so-called "habitus phthisicus" and embraces that great host of ailments associated with congenital delicacy of structure, ligamentous relaxation and unstable function.

"*Habitus phthisicus.*"

Physical Characteristics of Congenital Asthenia.—*Slender bones, a weak illy-developed thorax, nephroptosis, gastroptosis, constipation, the "drop" heart with its peculiar attenuation and weak and easily overstrained musculature are some of the conditions which characterize the asthenic state.*

Delicacy of structure.

Nutritional Instability in Asthenics.—*According to the observation of the author, diminished resistance to acute disease, relatively slow or imperfect recovery from acute ailments, a tendency to chronic subnutrition and rapid weight reduction are no less striking than the coming and going of symptoms of the most diverse sort in close accord with weight loss and gain.*

Dependence upon hypernutrition.

Gastric neuroses, psychasthenia and temporary, recurrent, or persistent, minor cardiac inadequacy are the chief manifestations *aside from tuberculosis, to which they are peculiarly susceptible* and by which many are *actively* infected. They represent in a considerable measure that great group of individuals who carry active or latent or larval, rather than wholly obsolete foci.

The great army of invalids, actual or potential, recruited from the asthenic multitude with their manifold symptomatic possibilities still offer an apparently tempting, but actually most barren field, for the overzealous and unwise surgeon.

A temptation to the unwary.

Such readily present their nephroptoses, gastroptoses, uterine displacements, aberrant intestinal loopings and the like to the surgical enthusiast but those who survive operation for the most part relapse and become a plague to their would-be benefactor.

Visceroptoses.

The extreme type is represented by the dreaded patients who carry the notebook containing daily memoranda of symptoms and queries. Such are always seeking new theories and fresh advisers when, as a rule, their need is met and their cure effected by the simplest of measures, viz., helpful suggestion, rest and hypernutrition.* In times of peace these peripatetic and gregarious possessors of rebellious stomachs and "tittery" nerves fill the "spas" and the more *bizarre* of the sanatoria of Europe.

The nervous dyspeptic and hypochondriac.

Haunters of the "spas."

Preferential Site of Tuberculous Lesions.—Almost any portion of the body may be involved, the lungs most frequently in adults, the bones, lymph glands and intestines in children, while the peritoneum, kidneys or brain may be involved at any age. Secondary involvement of the intestines is very common in advanced pulmonary tuberculosis.

Modes of Onset.—*It should never be forgotten that nearly every case of tuberculosis, whatever its form or apparent suddenness of onset, gives a history of previous impairment of health.*

Previous history.

* Unfortunately, however, an expensive matter, as it often involves prolonged hospital treatment.

As regards actual onset, it may be acute miliary and widespread, or astonishingly insidious, slow and chronic, wholly glandular or osteal, or peritoneal, pleuritic or pneumonic.

Onset variable.

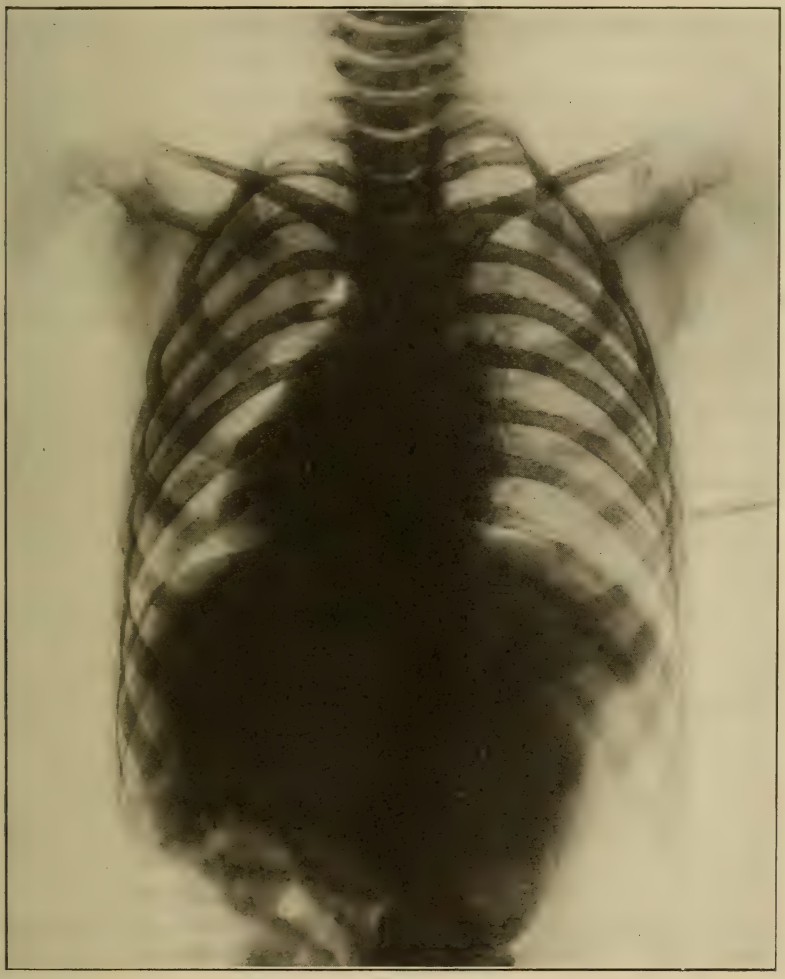


FIG. 151.—Acute miliary tuberculosis in a child. Note diffuse studding of lung fields with miliary tubercles. (*Dr. Frank S. Bissell.*)

ACUTE MILIARY TUBERCULOSIS

(*Acute Tuberculosis. General Diffuse Tuberculosis. Acute Disseminated Tuberculosis*)

Usual Cause.—The introduction of an adequate "dose" of virulent tubercle bacilli, from a latent or active glandular, osseous, or pulmonary focus, into the *blood stream*.

Wide dissemination of lesions.

Morbid Anatomy.—The viscera show general changes of an acute febrile infection and a general distribution of miliary tubercles; the lesions in the lung, pleura or brain predominating and the peritoneum or more often its diaphragmatic surface, being frequently much affected.

A deceptive type.

Symptoms.—*Typhoidal Form.*—The onset may be gradual and exactly simulate typhoid. The fever rises gradually or more rarely, sharply, and to a variable degree, 103° to 105°F. being common and afebrile cases rare. The symptoms may be those of the "typhoid state," but the following are prominent and should be carefully noted:

Important signs.

(*a*) *Markedly accelerated breathing.* (*b*) *Subjective and objective dyspnea.* (*c*) *A rapid weak pulse.* (*d*) *Cyanosis.* (*e*) *Sweats.* (*f*) *A variable, often intermittent or inverse temperature.* A *continuous* temperature may be present for some days prior to the development of *intermittency.* Usually but not invariably one observes (*g*) *cough,* unproductive or attended by purulent or

Bacilli may be absent.

mucopurulent sputum. (*h*) *Tubercle bacilli, which may be present* early, more often late, or, in rare instances, be absent throughout. (*i*) *Herpes labialis.* (*j*) *Rapid emaciation* is constant.

Usually scant.

Physical Signs.—At first they are absent or only bronchitic in character and for days or weeks may be scant or misleading. A careful examination may demonstrate the presence of *antecedent tuberculous lesions. The author*

Hyperresonance.

believes that diffuse and almost universally uniform hyperresonance is the most constant and significant early sign. In rare instances patches of consolidation are clearly defined.

The enlargement of the spleen occurs much later than in typhoid, and the same may be said of Ehrlich's diazo-reaction. Widal's agglutination reaction is absent, the stools quite unlike typhoid and blood cultures are negative. Furthermore, the pulmonary symptoms are strikingly predominant even in that acute general form which presents abdominal symptoms.*

Course.—Six to twelve weeks.

Prognosis.—Almost invariably fatal, though rarely the acute symptoms subside leaving a progressive but limited lesion.

Comment.—The general symptomatology of the disease is that of an acute intoxication with disseminated miliary foci and these facts explain the absence of marked physical signs. Cases may be wholly pulmonary or predominantly abdominal, but a division into pulmonary, typhoidal and peritoneal is useless.

Usually bronchopneumonic.

ACUTE PNEUMONIC TUBERCULOSIS (*Phthisis florida, Galloping Consumption, Acute Tuberculo-pneumonic Phthisis*).—This is a *relatively* rare disease representing not over 2 per cent. of the cases of pulmonary tuberculosis. It is usually bronchopneumonic in form; rarely lobar.

The Lobar Form.—*This may exactly simulate an ordinary pneumonia, but one often finds a history of previous illness or signs of old lesions.*

In these cases the destructive process is often rapid and extreme and

* As a matter of fact the confusion of typhoid fever and miliary tuberculosis need but rarely occur even if the diagnosis lacks the assistance of the Widal test and blood culture.

the author recalls one in which at autopsy the right lung was found to be entirely destroyed, leaving a single huge cavity, the left lung being infiltrated and hypertrophied to such an extent as to force the heart to the right and produce a concentric dexiocardia.

Instead of a true crisis, or following a deceptive pseudo-crisis, septic temperature develops with sweats and a purulent sputum containing tubercle bacilli, but in some instances the process subsides, becomes chronic and may even become arrested.

Pseudo-crisis or none.

Favorable outcome possible.

Early positive diagnosis is usually impossible.

Early diagnosis difficult.

The Broncho-pneumonic Form.—*The broncho-pneumonic form is characterized pathologically by broncho-pneumonic tuberculous lesions and shows a tendency to fusion, caseation and cavity formation.*

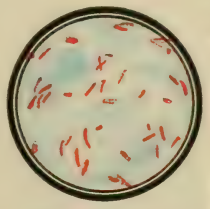


FIG. 152.—Tubercle bacilli. (Exaggerated. See plate under "Sputum" for better exposition.)

It presents the symptoms of an acute broncho-pneumonia of the common type followed by the signs of septic absorption, pulmonary infiltration and cavity formation.

Is evident later.

Tubercle bacilli may be and usually are present early, but their appearance may be delayed. Hemoptysis may occur and is sometimes the first event.

Bacilli often present early.

Comment.—In both the lobar and broncho-pneumonic forms of acute tuberculosis a delayed diagnosis is almost always necessary. The most careful questioning as to previous health and family history may yield no information, and the appearance of tubercle bacilli and the signs of destructive infiltration associated with hectic fever may be the only means of diagnosis.

Previous health important.

CHRONIC ULCERATIVE TUBERCULOSIS

Definition.—*A tuberculous disease of the lung characterized by chronicity, variability in severity and frequent intermissions. Aside from individual resistance, its termination in recovery or death depends upon the promptness with which a diagnosis is made and rational measures instituted.*

The common form.

The *sine qua non*.

Pathologic Anatomy.—The lesions are ordinarily those of a slowly progressive chronic tuberculous broncho-pneumonia; the terminal bronchioles and the alveoli being the seat of a sluggish inflammation forming areas of peribronchial pneumonia which may advance rapidly, remain almost unchanged over long periods or undergo complete arrest. The tendency in bad cases is toward fusion of areas, ulceration and cavity formation.

Chronic localized broncho-pneumonia.

Fusion of areas.

Favorable cases result in fibrosis, capsular limitation and arrest more often than is ordinarily supposed. Spontaneous recovery from *unrecognized tuberculosis* is extremely common, as shown not only by routine X-ray examinations and the results of the various tuberculin tests, but also by the post-mortem records at home and abroad; contracted apices, peribronchial nodules and adhesions being frequently found.

Fibrosis.

Frequency of arrest.

Inflammation of lung tissue in this disease leads to reflex defensive limitation of lung movement. The areas involved present more or less imperfectly, the signs of broncho-pneumonia either sharply localized or disseminated and often over extremely small areas; but large tracts may

Limitation of lung movement.

become involved with a corresponding increase in the frankness of the pulmonary symptoms.

Cavity formation.

By necrosis of the bronchial walls through inflammation aided by the pressure of retained secretion and a destructive ulceration, cavities are formed, the physical signs of which are fully discussed elsewhere.

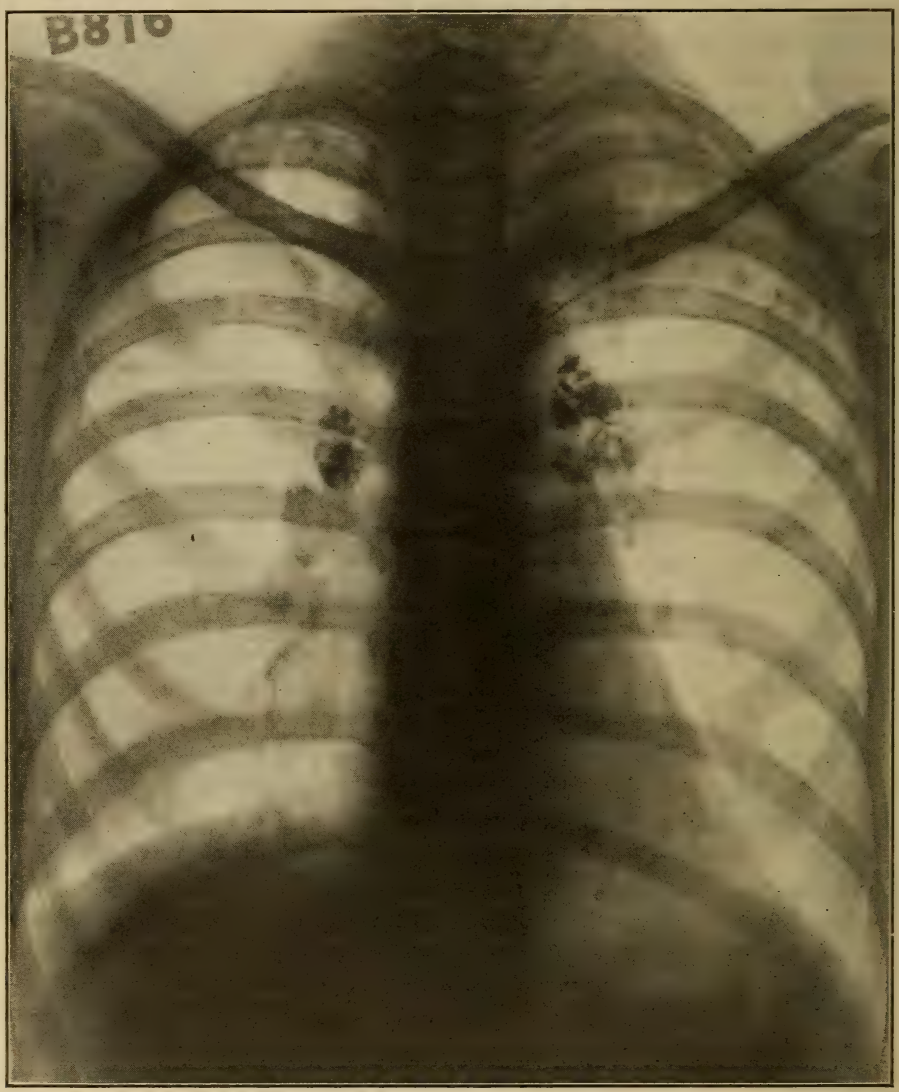


FIG. 153.—Calcified hilus and lung foci. Probably due to fungus infection in old pulmonary tuberculosis. (*Dr. Frank S. Bissell.*)

Cases of dubious signs.

New cavities without a firm limiting membrane may yield doubtful signs, particularly if they occur in the center of a caseous area lacking bronchial communication. These vomicæ vary in size, sometimes representing almost

the whole right or left chest as in the case already mentioned.* Small cavities are obliterated by a process of absorption, fibrosis and contraction; large cavities may be partially obliterated by the same processes and become inactive.

Variable size.

Processes of obliteration.

Hemorrhage may occur at any stage of a chronic ulcerative tuberculosis and though the arteries are usually resistant, *may* be the first symptom. The amount varies from a mere streak of blood to the rarer flooding that kills almost instantly.

Hemoptysis.

Bronchiectases are common in advanced cases, the intestines are often involved and this complication, *like ulceration of the larynx*, is painful, exhausting, and tends to greatly shorten life.

Laryngeal cases.

A Word of Warning.—*A large proportion of persons who have reached full maturity have at some time acquired a tuberculosis focus at an earlier period, in most instances trivial, latent or obsolete, at the time of examination, but sufficient to yield at any subsequent time a reaction to tuberculin and, in most instances, to show suggestive abnormalities in the X-ray photograph.*

Almost universal prevalence.

It is the duty of the physician to strive to detect even the latent foci by all safe means, but costly and burdensome therapeutic measures can properly be imposed only upon reasonable proof of the existence of active processes.

Question of activity of foci.

There can be little question that too great a dependence upon mere signs of past infection has often resulted in much unnecessary hardship to perfectly healthy individuals. On the other hand, one must be prompt and decided in the presence of any definite evidence of an *active process*, however slight.

Unnecessary and unjustified hardships.

Phthisiophobia.—The necessary and proper dissemination of knowledge concerning phthisis has developed to a considerable degree a morbid fear of infection and as a result one encounters many sound *phthisiophobes* who need a careful and thorough examination and positive reassurances based upon impeccable and defensible negative findings.

SYMPTOMS.—Early Symptoms.—*The modern theory of the curability of tuberculosis depends upon early diagnosis.*

Every physician who uses the X-ray knows that cases referred to him as latent or incipient are likely to be far advanced, *i.e.*, in the second or even third stage of the disease. They should be recognized before the stage of extensive infiltration and certainly before softening has begun if the best results are to be obtained. When so recognized at least 80 per cent. can be restored to apparent good health, and the larger number of these should remain well under proper conditions.

Early diagnosis.

Curability

Aside from tubercle bacilli and cavity signs, no one symptom is conclusive, but those following should be considered.

(*a*) **Loss of Weight.**—*The present weight, the best weight, and the weight prior to any noticeable impairment of health should be ascertained, as few cases develop activity without progressive weight loss.*†

Progressive loss.

* Case of Dr. B. J. Merrill, of Stillwater, Minn.

† Robust-appearing heavy-weights may have lost 30 or 40 pounds and such patients often receive scant sympathy and no proper examination.

Common.

(*b*) **Indigestion.**—Dyspeptic symptoms are common in the early stages of tuberculosis, and loss of weight is often attributed to that cause when it should be traced farther back.

Often denied when obvious.

(*c*) **Cough.**—*This may be entirely absent and is frequently unnoticed and denied by the patient, even though obtrusive during examination.* It is often

Usual character.

harassing and troublesome, particularly at night and, even in the advanced stage of the disease, may be entirely unproductive, hardly more than a clearing of the throat, or, associated with profuse cavity expectoration, bronchorrhea or even hemoptysis. It is oftentimes bronchiectatic in inveterate cases.

Excessive secretion.

Often absent.

(*d*) **Sputum.**—The sputum may be absent, mucoid, mucopurulent or purulent, according to the stage of the disease. Cases may show marked infiltration even before sputa are obtainable. Tubercle bacilli are present in all advanced cases, but a diagnosis must often be made in their absence. When found by proper staining methods the evidence is positive and final.

Open vs. closed cases.

Variable.

(*e*) **Pain** may or may not be present, and is often represented by a feeling of mere oppression, at other times being distinctly pleuritic in type or dull and ill-defined.

Seldom extreme.

(*f*) **Anemia** is frequently present in the early stages of the disease, and always in advanced cases. In the former it is sometimes of the chlorotic type, but usually one finds a *secondary* anemia.

(*g*) **Dyspnea.**—Dyspnea on exertion is not uncommon early and is an invariable *late* symptom.

Often confusing.

(*h*) **Bronchitis.**—A diffuse obscurant bronchitis or even spasmodic asthma is sometimes present and may mask the true lesion. In other instances it is a *localized and suggestive bronchitis.*

Suggestive types.

(*i*) **Pleuro-pericardial Friction.**—*The author has learned to regard with great suspicion frictional murmurs, pleuro-pericardial in type and site, not associated with a definite attack of pericarditis or pleurisy, but with impairment of the general health. In many instances these have proven the first demonstrable physical signs of a rapidly destructive tuberculous process.*

May be early.

(*j*) **Night Sweats.**—This troublesome symptom is ordinarily associated with the final stage, but may be encountered at any time in the course of the disease. Such sweats are both chilling and exhausting.

Labile pulse.

(*k*) **Pulse.**—*In connection with other symptoms* the excitability and undue rapidity of the pulse is extremely important.

Very important.

(*l*) **Temperature.**—In advanced tuberculosis, the temperature is simply that of a septic process, being distinctly intermittent or hectic in type and associated with night sweats. When new areas of the lung tissue are invaded, the temperature is likely to be continuous or only remittent. One is chiefly interested in the variations in *incipient cases* and here one must emphasize the importance of any persistent rise above the normal, however slight.

In febrile tuberculosis cases, the maximum rise is usually in the evening, but we may have an inverse temperature with the evening reading normal.

Afebrile cases

Some cases have no temperature, and one under the author's observation

a few years ago, went through the various stages of the disease to death without a rise above 99°F.*

Exertion temperature.

In many cases a temperature will be found only after exertion, and this is a matter of the greatest importance in relation to both diagnosis and treatment. Furthermore, in such cases a wide variation between a subnormal morning reading and the evening temperature, though the latter be within normal limits, may be as serious an indication as the abnormal rise.

Important clinical fact.

(*m*) **Hemoptysis.**—Although the fact is well known that hemoptysis without detectable cause is almost invariably due to tuberculosis, one finds many instances of most unfortunate time-loss due to a failure to put this practical knowledge into the form of early diagnosis.

Tuberculosis, pulmonary infarct, and syphilis are the three causes chiefly to be considered. Of these the two former are overwhelmingly the more frequent.

The term "hemoptysis" should not be applied to blood-streaked or even bloody sputum such as occurs in severe bronchitis, the pneumonias, or passive congestion of the lung. Its use should be limited to the actual hemorrhages of bronchial or pulmonary origin.

Aside from the three leading causes mentioned above, one must bear in mind bronchiectasis, pulmonary abscess and gangrene, aneurysm, malignant growths, actinomycosis, aspergillosis, streptothricosis, echinococcus, distomiasis, hemophilia, scurvy and the purpuras.

Fluoroscopy.—The skilful use of the fluoroscope and X-ray plate throws much light upon the condition of the lungs in incipient cases and by the former method one often finds a marked limitation of lung movement ("Francis Williams' Sign") as measured by the descent of the diaphragm, quite disproportionate to the amount of lung involved. Areas of extensive infiltration show as shadows, and cavities of considerable size as bright spots.

Extremely valuable.

Litten's sign may be used to test the diaphragmatic movement in the absence of the fluoroscope.

Tuberculin.—The subcutaneous use of tuberculin as a diagnostic agent has its many advocates and its few bitter opponents.

Varying views

It is argued that inasmuch as the physical signs produced are due to the production of congestion in tuberculous areas, every dose subjects the patient to unnecessary risks.

On the other hand, its advocates claim that the importance of "absolute" diagnosis and the rarity of bad results following its use serve as its justification. This might readily be admitted if the results obtained were positive *for active lesions only*, for bad results from diagnostic doses are extremely rare, but "absolute" information means usually nothing more than the fact that at some time, somewhere, the patient has been infected and, in the adult

Extreme frequency of reaction.

* The case was not observed in its incipiency though the infiltration was only slightly advanced, but as no temperature was shown during the stage of softening and when the sputum was filled with tubercle bacilli, it is to be presumed that it was absent in the earlier stages. The patient was a congenitally asthenic girl of 17.

Fallible.

one might "guess" correctly seven times out of ten without even seeing the patient. *It does not prove the activity of an infection.**

Routine use unwise.

The author believes that the routine use of the subcutaneous method is to be condemned and that the methods of Moro and Von Pirquet are adequate.

The fact that it is capable of producing a rise of temperature in so many apparently healthy controls† and in certain cases of syphilis, and the additional fact that from 60 to 70 per cent. of apparently normal individuals above the age of thirty react, certainly makes its findings less positive than might be wished.

Differential tests.

Furthermore, the recent attempts to differentiate the reactions quantitatively and read into them special diagnostic meaning are not convincing, despite the brilliant work of Wolff-Eisner, Teichman, Ellerman and Erlandsen, and others.‡

Some of the most striking reactions ever observed by the author have occurred in vigorous individuals wholly unconscious of any health impairment present or past and now enjoying good health after the lapse of years.

Preparation of Tuberculin for Subcutaneous Use.—"*Alt-tuberculin*" may be had in 1-c.c. (1000-mg.) ampules and one uses this content as follows:

Using sterile normal salt solution make a dilution of 1 in 10; with 1 c.c. of the 1 in 10 make another in the same manner and so on until four have been prepared.

Then 1 c.c. of dilution No. 1 = 0.1 = 100 mg.
1 c.c. of dilution No. 2 = 0.01 = 10 mg.
1 c.c. of dilution No. 3 = 0.001 = 1 mg.
1 c.c. of dilution No. 4 = 0.0001 = $\frac{1}{10}$ mg.

Thus by successive dilutions one may obtain any required lesser fraction of the milligram of old tuberculin.

The addition of 0.2 c.c. trikresol for each $\frac{1}{10}$ c.c. of solution will maintain its sterility or one may use a 0.5 per cent. solution of carbolic acid.

The Subcutaneous Tuberculin Test.—In *adults* $\frac{1}{10}$ to 0.5 mg. of *alt*-tuberculin in normal salt solution is first injected according to the condition of the patient. If no reaction occurs, 1 to 2 mg. are injected forty-eight hours later, and still later if the reaction fails 5 or even 10 mg.

Reaction Signs.—(*a*) *Fever.* (100°F. represents the "mild reaction," and 102°+F., the "severe form.")

* Chas. E. Simon gives the following table as illustrating the extraordinary frequency of the reaction:

Known pulmonary tuberculosis	90 to 100 per cent., positive.
Suspected cases	92.1 per cent., positive.
Supposedly normal cases	56.1 per cent., positive.

† Robert Koch himself developed a severe general reaction following the injection of 0.25 c.c. of tuberculin in the course of his experiments.

‡ Only the proof of marked superiority of this test, a proof not yet forthcoming, can justify its use in preference to the simple, safe and equally accurate test of von Pirquet. The author gives the test, nevertheless, because of the existing difference of opinion and its use and attempted justification by some of our ablest medical leaders.

(*b*) *General malaise*, aching and depression, prostration, sometimes nausea and vomiting.

(*c*) *Swelling of regional glands* in many instances.

(*d*) *Allergic reaction, i.e.*, redness and swelling at the site of injection.

(*e*) *Physical signs* may appear, or increase if preëxistent, indicating a temporary glowing or lighting up of old foci. Such are harsh breathing, crepitations, or crackles, increase or marked diminution of sputa, etc. Focal.

Precautions.—Injections should be made only in the forenoon lest an early temperature rise occurring in the night may be overlooked.

If fever is present one must await its subsidence over a period of several days.

If small doses are used, from ten to sixteen hours may be required before reaction occurs.

If the patient has any persistent fever the test is of little value.

Pregnancy, heart disease, diabetes, nephritis and laryngeal tuberculosis contraindicate its employment.

Modifications of the Tuberculin Test.—Von Pirquet's vaccination test depends upon the fact that hypersensitization of the tissues of an individual affected by an infectious disease (allergie) induces a specific reaction to the toxic substance even in the skin. This is merely an application of the doctrine of anaphylaxis. Simplicity.

The Von Pirquet Test.—Any good tuberculin, preferably the *alt-tuberculin* of Koch, is diluted by adding 1 part of 0.5 per cent. carbolic solution and 2 parts of normal saline solution; the patient's arm is prepared as for ordinary vaccination and three areas are lightly scarified, two through a drop each of the test solution *in situ*, the other similarly treated with the diluting fluid alone. The test and control punctures may be but slightly apart so as to be covered by the one vaccination shield or they may be on opposite arms. Both should be carefully protected.

The test as usually outlined at the present time demands the use of the undiluted old tuberculin. The special "*impfbohrer*" of von Pirquet is not essential.

Usually redness and *more or less infiltration* will appear at the site of the test abrasion within twenty-four to forty-eight hours, but even a vigorous reaction may be delayed for seventy-two hours or even more. No constitutional or localizing physical signs appear, and, though occasionally the reacting area appears angry and inflamed, all untoward signs soon subside. Absolutely harmless.

In all cases the author tests the degree of infiltration by pinching both the vaccination and control areas between the thumb and forefinger. This enables one to detect reactions in cases yielding little redness.

Wolff-Eisner and later Calmette attained the same result by instilling a 0.5 per cent. solution into the conjunctival sac where it was retained for a moment or two and in from twelve to forty-eight hours produced decided congestive or inflammatory signs in affected individuals. Conjunctival test.

Moro prepares an ointment of 50 per cent. strength, rubbing up *alt-tuberculin* with dehydrated lanolin. A pea-sized portion of this unguent is Moro's test.

gently rubbed into the skin of the chest or abdomen over an area of 5 cm., and this surface is left uncovered for from ten to fifteen minutes.

Papules in varying number with or without erythema appear within a period varying from a few hours to three days. Moro divides reactions into three grades, viz.: (1) *mild;* (2) *moderately strong;* (3) *severe.*

Relative value of tests.

From personal experience with all three tests the author believes the first (von Pirquet) and the last (Moro) to be quite as valuable and far safer than the tuberculin injections hitherto and even now extensively practiced, but as in the case of the older method, they fail to prove in adults much more than the fact that *at some time the individual has been infected.**

Conclusions.

The following conclusions represent the author's views as based upon the results of a series of tests undertaken with the three modified methods and a somewhat extensive later experience.

Results essentially those of old test.

(*a*) *The von Pirquet reaction is comparable and probably runs nearly parallel to the old injection test,† inasmuch as the percentage of reactions closely approximates that obtained by Beck and others using the older method.*

(*b*) *No fever or physical sign increase is to be observed.*

The "von Pirquet" the most reliable.

(*c*) *The three tests are almost equally valuable, though, in the author's hands, the von Pirquet proved the most reliable of them and should supplement any negative "Moro." The conjunctival test is the least certain and the only one of these three possessing elements of risk.*

"Arrest" reactions.

(*d*) *Active-incipient or moderately advanced cases in apparently resistant individuals and those carrying arrested or obsolete lesions react decisively, the last most vigorously and somewhat later in many instances.*

Prognosis.

(*e*) *In known active, progressive, but early cases, the more vigorous the reaction the better would appear to be the prognosis.*

Advanced cases.

(*f*) *Advanced cases with low resisting power afford a slight transient reaction or none at all, often to be detected only by careful comparisons of the infiltration produced.*

True value of tests.

(*g*) *As in the case of the subcutaneous test, the positive reactions are so numerous and decided in vigorous non-suspects as to be merely suggestive or reminiscent.*

Valuable in exclusion.

(*h*) *The value of the positive test in infants and very young children is considerable, and that of the negative test in suspects of all ages can harldy be overestimated, in that it practically excludes tuberculosis past and present.‡*

Known tuberculous subjects who do not react, almost without exception

* The value of negative results of these two tests in infants and very young children is obviously great and the younger the child the greater is the diagnostic significance of the positive test. Infection in children reporting to public clinics is said to reach 35 per cent. between the ages of six and ten.

† The author has made demonstrable preponderance of infiltration at the site of vaccination as compared with the control, the test of reaction rather than reddening of the skin alone, which sometimes fails especially in cachectic subjects.

‡ Franz has reported 61 per cent. positives in apparently healthy young recruits. Results obtained in school children as reported by Hamburger and Sluka, Feer, Hillenberg and others show positive reactions in from 40 to 60 per cent. Cohn has reported 100 per cent. positives in fourteen-year-old children of tuberculous parents.

present advanced, unmistakable and usually terminal symptoms with marked cachexia.

(*i*) *The methods of von Pirquet and Moro are wholly free from danger and cause little discomfort.*

The extraordinary prevalence of tuberculous infection in man, again may be emphasized by the following tables.

Staehelin has admirably summarized the evidence which supports the oft-quoted dictum, "*Jedermann hat am ende ein bischen tuberculose*" (every one has a little tuberculosis some time).*

Naegeli reports as follows on 500 autopsies.

Age	Fatal T.B.	Latent but active	Latent inactive	Total infected, per cent.
Under 1 yr.	0	0	0	0
1–5 yr.	17	0	0	17
5–9 yr.	25	8	0	33
9–17 yr.	15	15	8	38
18–30 yr.	35	36	24	95
30–40 yr.	47	28	39	94
40–50 yr.	22	23	55	100
50–60 yr.	20	18	62	100
60–70 yr.	9	25	66	100
Over 70 yr.	0	23	77	100

It appears that practically all in this series above the age of seventeen showed previous *infection* by the tubercle bacillus.

There is, of course, a considerable margin of error in such figures and one cannot properly apply the result of work in the dead house of the city clinic directly to the determination of the degree of infection prevailing throughout the population as a whole.

Neither can one consider figures derived from the autopsies held upon patients dying in a public clinic as equally applicable to those individuals who enjoy a better environment.

Nevertheless they indicate the extraordinary frequency of tuberculous infections in the past, now, happily, lessening.

Burchardt reported finding evidences of tuberculosis in 91 per cent. of 1452 autopsies.

Many other confirmatory investigations are available and one must conclude that at least 80 per cent. of living individuals above thirty years of age carry tuberculous lesions, for the most part obsolete.

(*j*) *The absence of the localizing physical signs of the severer positive reactions of the old subcutaneous injection test is more than compensated for by the lesser risk, the avoidance of confusion due to existing fever or temperature lability on the part of the suspect, the simplicity and the definite results yielded by the allergic tests.*

* R. Staehelin, "*Handbuch der innere medizin*," L. Mohr u. R. Staehelin, 1914, vol. ii, p. 465–652.

Comment.—All statistics show the great value of a tuberculin test (von Pirquet especially) in suspected infants and very young children in whom the infection is likely to be, or in infants must necessarily be, recent, but the liability to infection with increasing years progressively diminishes the positive value of the test.

(*o*) **Family History.**—The family history may throw light upon latent infection, predisposition, or the resistance of the patient, and should be carefully investigated, as regards both incidence and course of cases, in brothers and sisters, parents and collaterals.

(*p*) **Past Health Record.**—This may be of the utmost significance and importance. Past pleurisy, "winter cough," "inflammation of the lungs" of suspiciously long duration, "spitting of blood," and the like, merit careful investigation.

PHYSICAL SIGNS.—*A diagnosis must often be made in the absence of positive physical signs, as is evident from the genesis of the disease. Furthermore, the X-ray negative will reveal always changes of far greater extent and importance than those elicited by the physical examination of the same case.*

Area of involvement exceeds physical signs.

Inspection.—*The physiognomy of advanced tuberculosis is too well known to need a description, the laity recognizing it as readily as the physician.*

Advanced cases.

It is merely a composite of emaciation, hectic, exertion-dyspnea, and usually of faulty congenital chest conformation.

Incipient cases.

Incipient cases have no distinctive physiognomy, though an asthenic build is *usually present*, and anemia may or may not be evident. *Unilateral wasting of muscle in the suprascapular region is of much importance.*

Value of Litten's test.

The impairment of chest movement may be imperceptible in the incipient stage, though reduced expansion may be shown by the diaphragm phenomenon or the fluoroscope.

Frank cases.

Advanced cases show marked impairment of chest movement, and oftentimes localized contraction, abnormal pulsation along the pulmonary-cardiac boundaries and the characteristic, phthisical chest.

Misleading color.

Some cases present a high color similar to that noted in the mitral disease of young persons and due, apparently, to interference with the pulmonary circulation.

Often negative.

Palpation.—Palpation may be wholly negative in the incipient stage, though yielding most exquisite signs in the advanced cases where it reveals lack of expansion, inequality of voice transmission, signs of cavity or marked infiltration (see "Consolidation" and "Cavities").

Percussion.—In the earliest stages percussion may be negative or actually misleading.

Important data.

Any decided inequality in the percussion notes of the two sides, particularly at the apex, should attract attention. It should be remembered that, *normally*, the note on the left side is less intense but lower pitched than that of the right.

Hyperresonance.

Hyperresonance at the apex, especially if unilateral, is a valuable suggestive sign of an incipient process; in the later stage it is of course likely to indicate vicarious emphysema or the presence of cavities.

Patches of dulness may be made out, or extensive areas of infiltration, with or without cavity formation, according to the status of the case.

Auscultation.—This is by far the most important procedure.

Unilateral variation.

Unilateral diminution of the breath sounds or of voice conduction may be as significant as an increase in their intensity.

Cog-wheel breathing.

Harsh breathing and the so-called "cog-wheel" inspiration must be carefully noted and the latter must not be confounded with the inspiration associated with an overacting heart, such a condition being frequently met with in tuberculous subjects.

Undue prolongation of expiration, though it be low-pitched, may be a sign of importance and in advanced cases with marked infiltration and cavity formation the breath sounds are frank and characteristic.

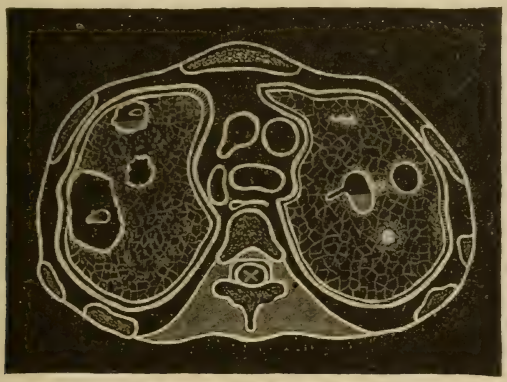


FIG. 154.—Various types of cavitation, illustrating some of the various factors affecting physical signs.

Râles may be absent for a considerable period in the incipient stage. One often hears only fine sibilant sounds, most significant if detected at the apex even in the absence of other signs, and particularly so if unilateral.

Cough maneuver.

Every patient should be made to cough in order to bring out râles, and deep inspiration following cough may be attended by sibilant, crackling or bubbling râles or by the so-called mucous click, according to the stage of the disease. In advanced cases the râles are of all types as described elsewhere.

Comment.—Certain points are absolutely essential to the proper examination and early diagnosis of tuberculous cases. A knowledge of the sounds produced by the various maneuvers in the normal chest is absolutely necessary.

Know the normal.

Unilateral variations are infinitely more important than bilateral ones in the incipient or early cases. Inasmuch as the disease usually commences at the apex, this region is the most important for the diagnostician. (Both are affected almost coincidently far more often than is generally supposed.)

Apex signs

As judged by the *physical signs,* the earliest recognizable primary lesion is commonly slightly posterior to the center and about 1 to 1½ inches below the apex. The disease tends to extend both upward and downward along the interlobar fissure and in front along the inner margin of the upper lobe.

Line of march

Posterior vs. anterior.

Posterior apex auscultation is usually more productive than anterior auscultation in the early stages of the disease.

The important auscultation areas are the apex, anteriorly and posteriorly, the inner lung borders anteriorly, the apex of the axillary space, and the region of the interlobar fissure posteriorly as roughly indicated by the scapular border when the arm is placed upon the opposite shoulder.

X-ray.

As previously stated the lungs are usually much more extensively involved than physical signs would indicate, as anyone may prove by the use of the X-ray and this fact no doubt accounts for the errors in prognosis and failure in treatment in cases judged wholly by the physical findings.

In fact, the systematic use of fluoroscopy or skiagraphy in these cases has a most chastening effect upon any man who believes himself able to accurately determine the number, position, character and extent of tuberculous lesions by other physical methods.

Height important.

Apex Movement.—It is important to employ auscultatory percussion over the apices in order to determine the height at which they stand and the difference in level as between inspiration and expiration. Marked retraction of one apex is an important sign of either an old apex lesion or an active and advancing one, but does not absolutely prove it tuberculous. The diaphragm phenomenon ("Litten's Sign") has already been described.

Warning.—*Tuberculosis is not the only disease capable of producing abnormal signs at the apices.*

PULMONARY INFARCT

(*Pulmonary Apoplexy, Hemorrhagic Infarct, Pulmonary Embolism*)

Genesis.—*All emboli in the branches of the pulmonary artery originate in the right heart, in thrombotic systemic veins, or from a thrombus in the artery itself, and may be septic or non-septic.*

Associated ailments.

The former occur in pyemia, ulcerative endocarditis, septicemia and exceptionally severe acute febrile infections. The latter result from the detachment of vegetations from the tricuspid or pulmonary valves, from globose thrombi or polypoid vegetations, from the trabeculæ of the right auricle, from the chordæ tendineæ or apex angle, or from a more remote non-septic thrombus.

In severe infections associated with marked myocardial toxemia the disengagement of such particles may follow sudden movement of the patient.

Peculiar area.

The result is usually the blocking of a branch of the pulmonary artery which causes a localized pneumonia primarily representing the distribution of the affected vessel and in some instances leading to abscess or gangrene. The areas are usually wedge-shaped with their bases at the periphery and may vary in size from those no larger than a tiny seed to the huge infarcts affecting an entire lobe.

Great variation.

SYMPTOMS.—Sometimes Obscure.—*Tiny infarcts may be wholly symptomless or produce only slight cough, dyspnea, and perhaps hemoptysis.*

*If branches of moderate size be involved these symptoms are more pronounced or indeed extreme and if the pulmonary trunk itself or its main branches be wholly blocked, rapid dissolution or instant death results.**

Diagnosis easy, or impossible.

Frank Cases Easily Recognized.—*A sudden onset with such symptoms as are described below, accompanied by the physical signs of circumscribed pneumonia, pleuro-pneumonia or possibly an acute edema, over an area usually basal or primarily axillary in location, together with the presence of recognized causative factors makes the diagnosis easy.*

The typical case.

If the embolus be septic the case becomes one of septic pneumonia with pulmonary abscess or gangrene.

If the septic element be absent prognosis is favorable as regards the lung condition. Multiple successive small infarctions may occur and their location sometimes may be determined easily, as in a case recently observed, and, in typical cases, the picture presented, viz., *sudden localized pain, dyspnea and bloody sputum*, is characteristic even though the physical signs be obscure. The middle and lower lobes of the right lung are the regions most affected.

Multiple lesions.

Sudden pain.

Dyspnea and hemoptysis.

It must be remembered that even large pulmonary infarcts may be unattended by bloody sputum.

Thrombi of the Pulmonary Branches.—Ribbert maintains that thrombosis is far more common than has formerly been supposed and that many of the silent infarction areas owe their presence to gradually induced thrombi in the pulmonary branches, rather than to embolus.

Silent infarcts.

PULMONARY ABSCESS.—*The abscesses may be single or multiple and due to tuberculosis, septicemia, pyemia, septic emboli, lobar and broncho-pneumonia, malignant endocarditis and indeed to suppurative disease of any organ or structure adjacent or remote.*

There is usually an associated empyema with embolic-infarction abscesses which are often multiple. Other abscesses may be distinctly localized primarily, but tend to extend.

Symptoms.—*The symptoms are those of sepsis* (see "Septicemia" and "Pyemia") *and the physical signs may be vague or lacking* if the septic focus is central, or simulate an encysted empyema if superficial. Perforation readily occurs (most commonly in a bronchus) and is associated with the sudden appearance of a considerable quantity of purulent sputum which then persists with or without distinct and demonstrable cavity signs.

Often obscure.

Perforation.

The sputum is then usually foul, and, in contradistinction to gangrene, contains elastic tissue. At first or at intervals it often shows more or less blood. It will be noted that the symptoms of sepsis apply to all forms; but closed abscess *may* yield no symptoms whatever or only percussion dulness and compression.

Foul sputum.

Silent cases.

Difficulties Seldom Insurmountable.—Despite this fact, in actual practice the author has not found the diagnosis of even closed pulmonary abscess so difficult a matter as one might assume.

* It has seemed to the author that otherwise silent blocks in the pulmonary area may account best for many of the extreme abrupt, but transitory periods of urgent dyspnea and precordial oppression so often observed in decompensated cardio-vascular cases.

The septic temperature is rarely absent and if localized dulness (often showing a central maximum and shading into a circumferential hyperresonance) can be determined, the free use of a clean aspirating needle introduced if possible at the center of maximum dulness point usually makes the diagnosis positive.

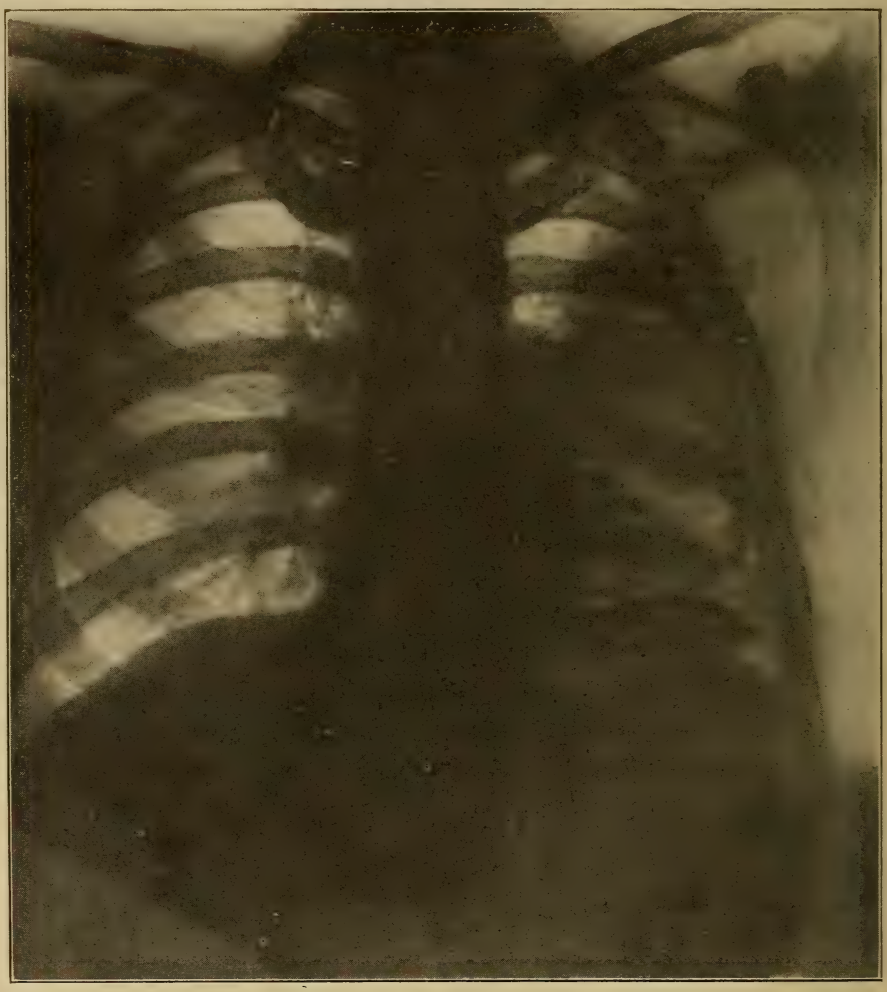


FIG. 155.—Closed abscess in left lung.

The roentgen-ray is often of the utmost value in these cases.

Encysted empyemas.

In localized and walled-off (encysted) empyemas the symptoms may be precisely the same as closed pulmonary abscess, though the area of percussion dulness or flatness is usually more sharply delimited than is that of abscess.

Always serious.

Prognosis.—Embolic abscess is almost invariably fatal and the prognosis is bad in all, though their course may oftentimes be prolonged or recovery achieved through surgical interference.

Rare.

PULMONARY GANGRENE.—*The etiologic factors are chiefly those of septic pneumonia and pulmonary abscess. It is a rare complication of diabetes*

and pneumonia, exceptionally rare in tuberculosis, and varies greatly in extent, being either circumscribed or diffuse and usually but not always affecting the lower lobe.

The involved areas are surrounded by consolidated, congested or edematous areas. An associated empyema is common and pleurisy invariable in the peripheral lesions.

Perforation.

It shows the same tendency to perforation as does abscess, most commonly into a bronchus, more rarely into the pleura or even the pericardium, esophagus, or through the diaphragm.

Odor diagnostic.

Symptoms.—*The only symptoms differentiating the disease from pulmonary abscess or certain excessively foul bronchiectases is the peculiarly horrible odor of the sputum and the usual but not invariable presence of shreds or fragments of lung tissue,* characteristically gangrenous and simulated by no other condition except certain rare cases of putrid bronchitis with interstitial pneumonia and pulmonary carcinoma *with gangrene.* Owing to some peculiar fermentative action, *the sputum often contains no elastic tissue, a fact of some importance.*

Prognosis.—Embolic, malignant, and diabetic cases invariably die; in others the prognosis is unfavorable, but not absolutely hopeless.

Primary rare.

PULMONARY TUMORS.—Only the malignant variety need be considered. Primary sarcoma is a clinical curiosity* and carcinoma rare, both being usually metastatic. The most common primary focus is of course the mammary gland, less often uterine, gastric, rectal and osseous growths. Carcinoma is the type of growth oftenest encountered and is most common in middle age and in the male.

Frank or obscure.

Symptoms.—The disease is often strikingly symptomless for long periods or baffling and indeterminate, the size and location of the growth being the chief factor; obstinate and violent cough may or may not be present. Sputum may be entirely absent or like prune juice or currant jelly, blood-streaked, or even purulent and of gangrenous odor, and contains compound granule cells. Involvement of the pleura may produce severe pain, and the dyspnea usually present in some degree may be strikingly paroxysmal; pressure symptoms may be marked and are often identical with those of aneurysm. Fever is often noted as the disease advances, profuse hemorrhage occasionally occurs and there may be marked displacement of the heart. Secondary growths are usually suggested by the primary lesion, but primary ones are frequently beyond a positive diagnosis. The X-ray may prove valuable in such cases and if the growth be accessible and of considerable size it may yield percussion dulness, usually without tubular breathing, and increased fremitus, or, more rarely, if attached to a large bronchus, both phenomena. Cachexia is of relatively slow development in carcinoma of the lung.

Pressure symptoms.

X-ray.

* In a case of primary sarcoma of the lung observed recently by the author no positive diagnosis could be made during life, though the ailment was suspected by reason of the peculiar but extremely faint shadows shown by the X-ray. The physical signs were indeterminate, the heart signs and pressure symptoms slight but somewhat suggestive of aneurysm and the autopsy showed small disseminated growths, bilateral in distribution though chiefly affecting the left lung.

Effusions.

MALIGNANT GROWTHS OF THE PLEURA.—What is said under the head of malignant growths of the lungs applies here save that the *primary* signs and symptoms are *pleural*. Cardiac displacement is usually less marked than in simple effusions, but usually a differential diagnosis depends upon the withdrawal of blood-stained fluid in amount disproportionate to, or without achieving a proper diminution of, the area of flatness.

mportant point.

The statement as to the frequency of primary growths of the lung is even more applicable to malignant growths of the pleura, but endothelioma is more frequent in this region than in any other portion of the body.

This usually takes the form of multiple nodules with infiltration and thickening of the pleura and in full-developed cases may yield to light percussion a flatness equal to that of the bloody fluid with which it is associated.

Primary sarcoma is also encountered in rare instances, and sequential involvement of the lung is the rule.

Prognosis.—Death invariably results though only after a period of several months.

Important areas.

DISEASES OF THE BRONCHIAL GLANDS.—The most important of these glands lie in the angle of the tracheal bifurcation about the main bronchi. The smaller glands follow the course of the bronchi lying in the interlobular connective tissue. They must chiefly be considered in connection with the ailments of children or adults, as possible sources of secondary enlargement with or without symptoms. It should also be remembered that gangrene of the lung may involve them and that through dusty occupation (inhalation of dust), they may become pigmented and somewhat enlarged.

Age.

Occupation.

Associated Ailments.—The following diseases may affect them: (*a*) Severe acute bronchitis. (*b*) Scarlet fever, measles, whooping cough, typhoid and similar ailments. (*c*) Broncho- and lobar pneumonia. (*d*) Pulmonary gangrene. (*e*) Mediastinal, or, by metastasis, remote, malignant growths. (*f*) Hodgkin's disease and leukemia (especially the lymphatic form). (*g*) Pulmonary tuberculosis. (*h*) Tuberculous or malignant disease of the *abdominal or retro-peritoneal* structures.

Tuberculosis.

The greater number of cases in children are due to tuberculosis, the primary source of infection being either gastrointestinal, tonsillar or bronchial.

Symptoms.—Many enlargements are symptomless, in other cases the effects are those of pressure within the mediastinum combined with physical signs chiefly observable in the interscapular region and upper sternum. These signs are usually so indefinite, occurring as they do in areas most unfavorable for percussion or auscultation, that the early diagnosis rests usually upon pressure symptoms, an X-ray picture which is often distinctive, and the knowledge of an adequate cause. (The subject of pressure symptoms is thoroughly discussed under the head of "Aneurysm.")

X-ray.

Much stress has been laid upon D'Espine's sign previously described (see page 310) and also that of Eustace Smith which is a venous hum heard over the jugulars and *over the sternum* just below the level of its notch. To elicit it the head is tipped backward.

Neither sign is of much importance and usually both are absent.

Pressure.

Pressure symptoms are practically identical in all forms of mediastinal growth or tumor, though the most *distressing* cases ever observed by the author have been associated with the glandular enlargement of Hodgkin's disease.

In some cases a mediastinal new growth yields transmitted pulsation strongly resembling, though seldom accurately simulating, the *expansible* pulsation of aneurysm.

Pulsating dermoid cyst.

The pulsating dermoid cyst is the new growth most likely to cause this error. If, however, the clinical auscultatory phenomena of aneurysm are demanded as indispensable to a positive diagnosis of that condition no mistake is likely to occur.

Rare and obscure.

MEDIASTINAL ABSCESS.—This excessively rare condition may be acute or chronic, and occurs chiefly in the male in connection with traumatism, the acute infectious diseases, pulmonary abscess, gangrene or advanced tuberculosis. It is recognized by the symptoms of sepsis associated with severe radiating substernal pain and marked pressure symptoms. It may rupture and produce a fluctuating tumor in an intercostal space or discharge into the esophagus or trachea. The danger of confounding aneurysm with a pulsating mediastinal abscess is slight save in those cases in which an aneurysm by its continued pressure has produced necrosis and ultimate abscess in some adjacent structure. The use of a *fine* hollow needle involves no considerable risk and usually establishes the diagnosis. One need never hesitate to employ such a procedure if other measures of differentiation fail in so urgent a condition as acute mediastinal abscess. In this connection the cold abscesses usually arising from spinal caries should be borne in mind. The X-ray may be of the utmost value in such cases.

Possible error.

Use of hollow needle.

Fibrosis.

CHRONIC INTERSTITIAL PNEUMONIA (*Fibroid Phthisis, Pulmonary Cirrhosis*).—In spite of years of observation the conditions characterized by extensive fibroid changes remain imperfectly classified. In a broad sense it is best treated under the one heading and we may assume that lobar pneumonia, broncho-pneumonia, old pleurisies, syphilis, echinococcus cysts and *tuberculosis* may operate to produce a lesion, the chief characteristic of which is fibrosis.

Types.

Chronic diffuse interstitial pneumonia is excessively rare as a sequence of acute lobar pneumonia, though a few cases have been described with some accuracy. *Chronic broncho-pneumonia* is more common, but is usually tuberculous and *interstitial pneumonia* following pleurisy (pleurogenous) undoubtedly occurs as a result of prolonged compression, it being associated usually with a greatly thickened and adherent pleura. The cases of interstitial pneumonia due to continuous dusty occupations (pneumonoconiosis) and the syphilitic tuberculous and echinococcus forms are better understood.

Important signs.

Morbid Anatomy.—The process may be lobar (massive) or lobular (peribronchial, broncho-pneumonic, insular). *The sound lung is markedly emphysematous* and the heart is thus pushed and drawn toward the diseased

Retraction.

lung, pulmonary shrinkage and adhesions being usually marked, though sometimes lacking (*e.g.*, in pneumonoconiosis).

The lung itself may be marvelously shrunken and show chronic bronchitis, multiple bronchiectases and perhaps aneurysmal dilatations of the pulmonary artery. *The heart is enlarged*, its right chambers being especially dilated.

The heart.

The varied nature of the lesion makes possible wide differences in post-mortem findings. The most extreme cases represent the massive form of the disease, while gummata, apical tuberculous cavities or echinococcus cysts may indicate the specific primary cause as may the peculiar pigmentation of the tissue observed in cases of *anthracosis* (coal miner's disease) and *siderosis* (due to metallic particles), *chalicosis* (grinder's rot, stone cutter's phthisis).

Symptoms.—*The symptoms accurately follow the morbid anatomy as stated, being those of emphysema, chronic bronchitis, pleural adhesion, bronchiectasis or chronic phthisis, according to the nature and extent of the lesions, combined with physical signs bearing the same relation to causation.*

The displacement of the heart, retraction and immobility of the thorax on the affected side, scoliosis, and the depressed shoulder are emphasized by comparison with the voluminous opposite side. Symptoms of marked *infiltration* are usually found, but vary widely with the degree of involvement.

Consolidation.

The drag exercised by the diseased lung upon the mediastinum and its contained structures reënforced by the thrust of the bulky emphysematous opposite lung may produce an extraordinary displacement of the heart and its great vessels.

Extreme chronicity.

The disease is not only remarkably chronic but one which permits considerable activity for many years or even for a reasonably long lifetime.*

PULMONARY SYPHILIS.—There are no characteristic symptoms of this disease which most commonly manifests itself as interstitial pneumonia. It occurs both in congenital, and tertiary acquired, syphilis. The so-called *white pneumonia* is of no clinical importance, being found only in the lungs of dead babes usually stillborn. The latter process is frequently associated with tuberculosis. In acquired syphilis, gummata, single or multiple, varying in size from a small seed to a hen's egg may be encountered. The physical signs are in no way peculiar to syphilis. Whether there is an actual destructive disease of the lung and true syphilitic phthisis is still debatable. There are certainly rare cases in which softening is associated with caseous gummata and similar cases not infrequently occur in connection with tuberculosis.†

The lower lobes are usually sites of election.

* One case observed by the author during a period of nearly twenty years has passed through several severe illnesses and has lived to see his two healthy brothers die of acute disease; another case, showing the classical signs of inherited syphilis and every evidence of massive chronic fibrosis, has undergone an appendectomy and nephrotomy and several attacks of influenza. Both men have been almost continuously at work during the whole period.

† No one can have failed to encounter cases of undoubted acquired syphilis in which a rapidly advancing proven tuberculous process showed a marked improvement following the use of specific medication.

PULMONARY ACTINOMYCOSIS.—In all essential particulars this disease presents the picture of chronic bronchitis, pulmonary tuberculosis, or pulmonary abscess, and its diagnosis almost invariably depends upon the recovery of the specific organism from the sputum. It should be suspected if in such cases tubercle bacilli are absent, the bases chiefly or primarily involved and superficial swellings or brawny inflammations noted.

Differential factors.

In several cases encountered by the author it has taken the form of "closed" pulmonary abscess revealing its true nature only when explored.

ASPERGILLOMYCOSIS.—This rare disease is due to the aspergillus fumigatus and has been observed chiefly in those who handle infected flour, meal or grain. The symptoms are essentially those of pulmonary tuberculosis lacking the tubercle bacilli and the diagnosis can be made only by the discovery of the mycelium. It may also occur as a secondary disease in connection with the various chronic pulmonary ailments and may assume a predominatingly bronchial, nodular, or cavernous form.

Resembles tuberculosis.

OIDIOMYCOSIS.—This curious ailment sometimes closely simulates pulmonary tuberculosis.

NOCARDIOSIS.—The *nocardia* are peculiar microörganisms resembling *bacteria* on the one hand and *moulds* upon the other. They are branching, thread-like, aerobic, spore-producing bodies, growing readily upon nearly all culture media at ordinary temperatures.

They are widely distributed, are found especially on grasses and grains and their point of entrance is usually the pulmonary tract.

They produce symptoms and actual pathologic changes closely resembling tuberculosis, the germs of which disease they may closely simulate if fragmented.

They are readily distinguished by their behavior on culture material and by their staining properties.

The pulmonary symptoms are in themselves identical with those produced by the tubercle bacillus.

They do not resist acid and are decolorized by alcohol.

These three conditions are dealt with more fully in their appropriate section.

PULMONARY DISTOMATOSIS.—("Lung Fluke disease").—See *Paragonimus Westermanni*.

PULMONARY AND PLEURAL HYDATIDS.—Diagnosis.—So long as hydatid cysts are central and small, few or no symptoms are produced. If they enlarge, and particularly if they reach the pleura, cough and pain may be severe. Fever is usually absent, and dyspnea slight. Physical signs may be baffling, indeterminate, or merely those of consolidation with weakened breath sounds, as in an area of massive pneumonia (blocked bronchi) or a closed pulmonary abscess. The superficial (pleural) cases will strongly resemble encysted empyema but are more often like superficial pulmonary abscess in their central intensification of dulness and may reveal a most suggestive friction opposite this point, pleuritic effusion with the usual pressure displacements, weakened breath sounds and diminished fremitus.

Symptoms variable.

Peculiar cases.

If rupture occurs, the immediate symptoms may be urgent, the watery nature of the fluid is suggestive and characteristic and hooklets and fragments of the membrane may be present. Hydatid thrill and superficial rounded tumors may be found. Fowler emphasizes the *relative* absence of *mediastinal pressure* symptoms (see "Aneurysm") despite the evidences of a *large tumor or exudate.*

Zapelloni has reported positive results with the "deviation of complement" test in 93 *out of* 114 *operated cases of echinococcus disease.* Eosinophilia was present in but 60 per cent.

Echinococcus cysts usually involve the right lower lobe and in open pulmonary cases may show the characteristic hooklets in the sputum.

DERMOID CYSTS OF THE LUNG.—Dermoid cysts in the lung are of rare occurrence, usually originating in the anterior mediastinum. They offer great difficulties to the diagnostician and may vary in size from that of a small marble to that of a large grapefruit. Occasionally rupture occurs into a bronchus or into the pleural space or the lung itself. In rare instances, through pressure, escape of the fluid may occur into the pericardial cavity or the great vessels about the heart.

Exploratory puncture.

Chylous Pleurisy.—A true chylothorax results, rarely, from occlusion or rupture of the thoracic duct or receptaculum chyli, usually as a result of malignant or tuberculous disease. The diagnosis can only be made by the discovery of a pleural effusion and the removal of a portion for examination. *Such cases lack any history of acute pleurisy.*

EXAMINATION OF HEART AND BLOOD VESSELS

THE HEART.—Boundaries Usually Encountered.—The heart lies in the mediastinum between the lungs and presents, when the patient is recumbent:

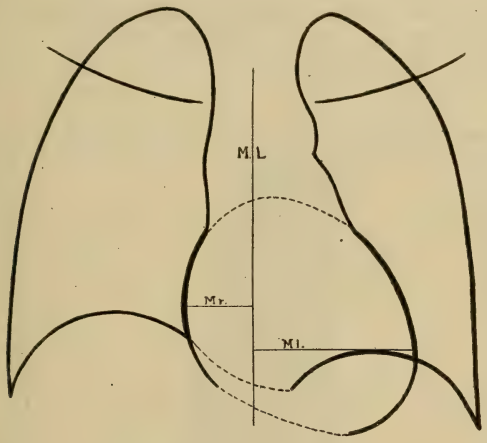


FIG. 156.—The two dimensions of chief importance in teleroentgenography, viz., Mr. at level of 4th interspace; and Ml. at level of 5th. Both Ml. and Mr., however, should represent the maximal distances from the median line to the heart borders.

FIG. 157.—Silhouette of the "sthenic" or "ideal" heart. The single convexity on the left of the diagram represents the right auricle. The four convexities on the right, from above downward are: 1st, the aorta; 2d, the pulmonary artery; 3d, left auricle; 4th, a narrow strip of the left ventricle. This figure would represent the heart outline when the diaphragm is in the neutral position. Compare with the ptotic type of heart shown on the following page.

(*a*) **A base** at the level of the upper border of the third costal cartilages.

(*b*) **A right border** formed chiefly by the right auricle, save in the case of the "drop heart," curving from the right base downward to the sixth chondrosternal articulation and attaining *in robust individuals* a distance from 3.0 to 4.5 cm. from the *midsternal line* under the fourth interspace.

Minimal and maximal figures.

The total of the right and left transverse heart diameters seldom exceeds 13.5 cm. and may not be greater than 8 or even 7.5 cm. in emaciated congenitally asthenic subjects (see "Drop" Heart).

Important.

In the latter cases the right border may not pass beyond the right edge of the sternum.

(*c*) **A left border,** which sweeps outward, downward, and then slightly inward, to a point in the fifth interspace, varying between 5.0 and 9.0 cm. (2½ to 3½ inches) from the median line, *according to the weight and build of the patient and the type of heart.*

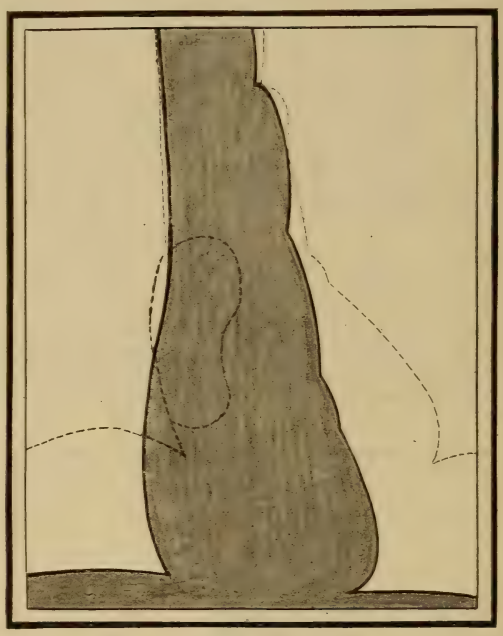


FIG. 158.—The "drop" heart of congenital asthenia and visceroptosis. Normal heart contour and elevation shown by dotted lines. (*Schwarz modified.*)* Found in a considerable proportion of the population and in all grades it constitutes a most misleading variant and is responsible for a vast number of missed diagnoses of an existing cardiac enlargement.

Small hearts.

In persons of the congenital asthenic type, be they fat or thin, the total dimensions normally fall short of the measurements ordinarily given as the normal.

(*d*) **A lower border** connecting the lower extremities of the lines representing the right and left borders.

Variations in posture as between the sitting and recumbent position affect the total *transverse* measurement but slightly.

* The position of the auricle in the typical "drop" heart is indicated also by a dotted line. The angles or notches of the left border are exaggerated as compared with the usual roentgenographic picture. Such hearts obviously must be so modified by the effects of valvular diseases and myocardial degenerations as to largely lose their characteristics. Whenever decided visceroptosis is present, whatever the apparent or actual heart lesion, a "drop" heart may be assumed as having existed primarily.

"Rule of thumb" inapplicable.

In order that the relatively small size of the average normal heart may be the better appreciated, the author has inserted the tabulation of Dr.

DATA AND MEASUREMENTS OF THE HEARTS OF SOLDIERS EXAMINED

No.	Weight Kg.	Height Cm.	Age Yrs.	Transverse diameter Cm.
1	53	157	21	13.4
3	54	164	40	12.0
5	55	158	29	12.8
7	56	173	24	13.8
8	56	171	27	12.3
10	57	160	19	12.3
11	57	168	24	12.6
15	58	168	25	11.4
16	58	170	25	11.2
17	59	175	19	12.5
22	60	174	29	11.3
24	60	162	23	13.3
25	60	164	29	13.3
26	60	183	25	12.5
27	60	174	21	10.8
29	60	170	24	12.5
31	61	174	24	11.8
35	62	163	24	11.2
36	62	166	30	14.2
37	62	173	29	11.9
43	63	163	26	13.5
44	63	174	40	13.8
46	63	167	24	11.8
47	63	164	21	12.8
48	63	169	28	12.0
49	63	178	23	12.7
50	63	170	23	11.3
52	64	167	30	14.4
59	64	181	29	11.2
60	64	171	26	12.8
62	65	177	28	14.2
64	65	168	20	12.8
66	65	176	21	12.8
68	65	172	27	12.8
69	65	170	23	12.7
72	66	164	27	13.5
74	66	167	26	12.4
77	67	161	25	14.5
78	67	173	29	14.6
79	67	167	25	13.7
81	67	175	23	12.4
82	67	167	26	14.4

Alfred E. Cohn of the Rockefeller Institute for Medical Research* very slightly abbreviated.

DATA AND MEASUREMENTS OF THE HEARTS OF SOLDIERS EXAMINED

No.	Weight Kg.	Height Cm.	Age Yrs.	Transverse diameter Cm.
83	67	175	28	12.4
84	67	168	27	14.0
85	67	169	23	13.2
86	67	170	24	15.0
87	68	167	49	13.5
89	68	171	21	13.4
90	68	175	24	12.8
91	68	174	18	11.8
94	68	175	26	14.0
97	69	175	42	15.7
100	69	169	26	14.8
101	69	174	31	13.8
103	69	177	25	11.8
106	69	172	26	13.6
108	69	180	28	12.3
111	70	171	30	12.8
113	70	176	29	13.3
117	70	175	22	11.8
118	70	175	18	13.0
119	71	172	22	13.7
122	71	170	29	14.2
123	71	177	34	13.3
125	71	178	23	12.1
126	71	172	23	11.3
127	72	179	24	12.7
129	72	174	22	14.0
132	72	174	20	12.4
133	72	174	29	14.7
135	72	173	21	12.8
137	73	178	21	14.1
138	74	174	28	14.4
140	74	171	24	13.7
141	74	172	23	12.3
142	74	172	25	13.0
143	74	178	27	13.3
144	74	186	23	12.7
147	75	173	25	16.0
149	75	165	27	13.0
150	75	175	30	13.2
154	77	179	28	12.3
155	77	181	30	12.8
157	78	170	28	14.3
158	83	180	27	14.3
160	83	179	26	13.8
161	86	180	26	13.8

All transverse measurements given are those of returned soldiers who had been subjected to the privation and exertion incident to active service on the Western Front during the "Great War" just past.

Infantrymen were chosen by preference or those whose service entailed an equivalent amount of privation and exertion.

All measurements included in this modified table were made in the inspiratory phase by fluoroscopy at a six-foot focal distance.

No special criteria other than those mentioned above governed the choice of these men and obviously a considerable number of hearts abnormally enlarged must have been included.

Nevertheless the table abundantly supports the contention made originally by the author over a decade ago and set forth in the paragraphs following:

Variations in Size of the Heart.—*The size of the normal heart varies considerably in different persons and its bulk in general corresponds to the osseous and muscular development of the individual, not to his weight, breadth or thickness, though height and* **muscular development** *combined are important factors.*

A Source of Gross Error.—A general lack of appreciation of the relatively narrow maximal limits allowable for normal hearts and a disregard of the structural factors necessary to the just application of a maximal allowance of 14 cm., even for robust, mature men, lead to a multitude of serious errors with relation to minor but not negligible incompensations and enlargements *and the existence of serious myocardial disease.*†

Total transverse measurements

Forced expiration materially increases the transverse diameter.

Many cases of cardiac dilatation of pathologic degree, especially (but by no means exclusively) such as occur in the extremely common, narrow, so-called "drop heart," of the congenitally asthenic individual, are overlooked and neglected because of a general adherence to "rule of thumb" measurement and faulty initial concepts.

Neglected at dilatations.

Furthermore, the application of the same rule may result in errors of the grossest type with relation to major dilatations of the small hearts.

The Aorta.—From the base of the heart, the aorta sweeps upward and to the right, its right border projecting slightly beyond the sternal margin. *It then passes backward and toward the left in such a manner as to leave the manubrium resonant under normal conditions.*

Manubrial percussion note.

This area assumes great importance in certain cases of aneurysm or enlargement of the thyroid or thymus gland.

Mobility of the Heart.—*Suspended* within the pericardial sac by the great vessels at its base, the heart is movable, responsive to all changes of posture, and laterally displaceable to an astonishing degree in cases of "drop" heart (*"corpendulum"*) or under certain pathologic conditions (ascites, tympanites, pleural effusion, pneumothorax, etc.).

"Corpendulum."

* Archives of Internal Medicine, May 15, 1920, vol. xxv, No. 2.

† Up to the present time the author has encountered no heart exceeding (teleoroentgenographically) 14 cm. in total transverse diameter in the inspiratory phase whose possessor did not show positive evidence of heart disease. This has held true even in the case of men of unusual musculature and athletic tendencies.

Chiefly right ventricle and auricle.

Relation to Anterior Thoracic Wall.—*Anteriorly it presents its right chambers; chiefly the right ventricle to the left of the mid-sternal line, and the right auricle from midsternum to the true right border which it wholly forms.**

The left ventricle is normally represented by a mere strip of heart muscle along the left border and the normal left auricle is hardly in evidence.

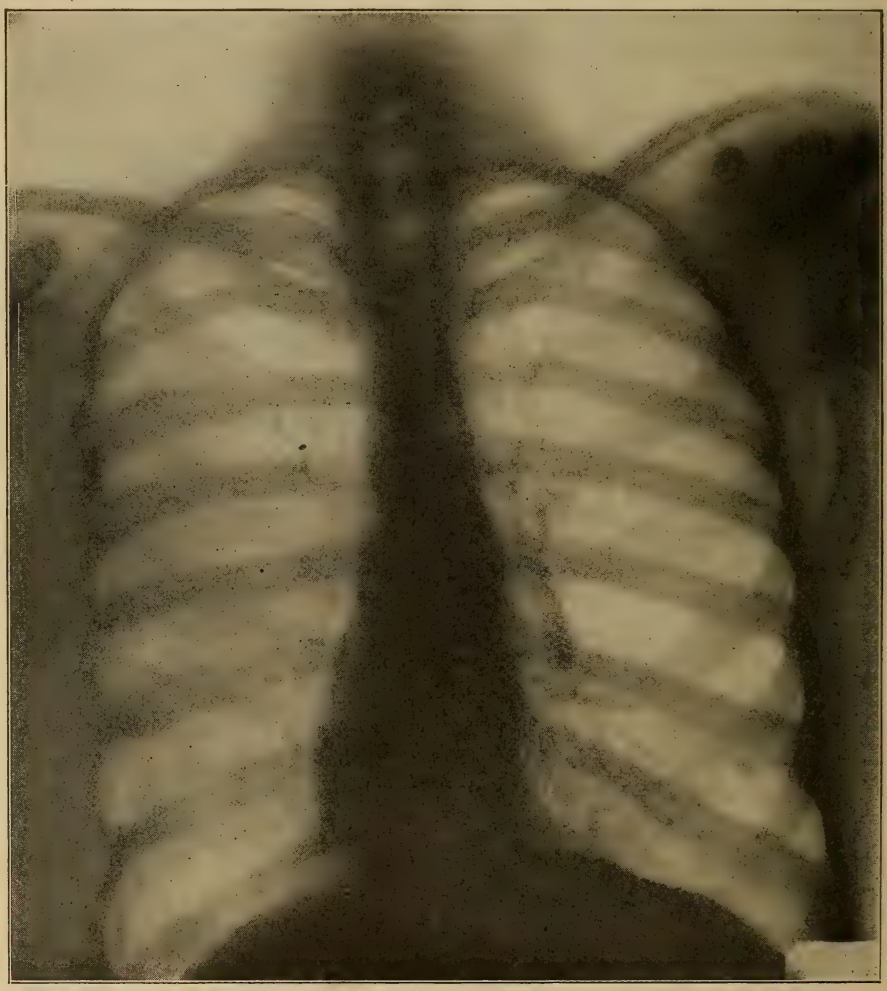


FIG. 159.—"Drop" heart. An extreme example in the adult male. Total transverse diameter 7.5 cm.

Cardiac Outline.—Changes in the shape of the relative cardiac dulness or the radiographic shadow usually indicate enlargement of the heart itself, suggest the individual chambers involved and hence the underlying lesions.

Of utmost value.

As will be seen later, such changed outlines serve as a check upon the diagnosis of valvular lesions and pericardial effusions, and the student must

* Save in the "drop" heart and its minor modifications.

never forget that even serious dilatation thus indicated may be unattended by any valvular murmurs.

Murmurs may be absent.

THE HEART VALVES.—*The mitral, tricuspid, aortic and pulmonary valves are* **anatomically** *located within so small a space that a large stethoscope bell will nearly cover them all, but their exact position is of little importance in clinical work.*

Anatomic position unimportant.

THE CLINICAL VALVULAR AREAS.—The points of maximum audibility arbitrarily fixed for auscultation are:

Arbitrary auscultation areas.

(*a*) **The mitral area** *which corresponds to the heart apex.*

(*b*) **The tricuspid area,** *corresponding to the lower half of the sternum.*

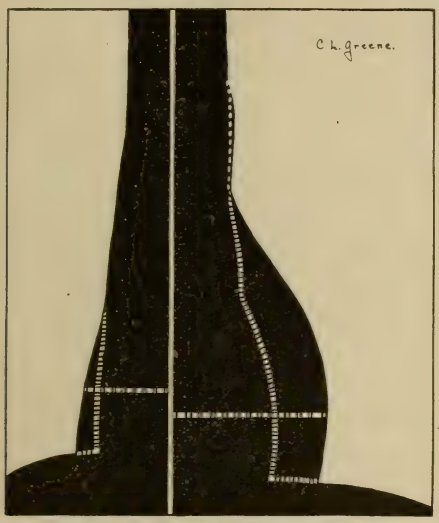


FIG. 160.—Dilated "drop" heart. (Outlines drawn from original roentgenograms.) Cause of dilatation, over-exertion following an unusually prostrating attack of influenzal bronchitis. Note that breadth of the *dilated heart* was but 12.1 cm. and after treatment 9 cm. (See also case of D. B. under "Drop" Heart, successive roentgenograms.)

(*c*) **The pulmonary area,** *corresponding to the second left intercostal space at the left sternal edge.*

(*d*) **The aortic area,** *corresponding to the second right and third left intercostal spaces, adjacent to the sternum, the former being most useful in connection with aortic systolic murmurs and aortic second sound accentuation; the latter, in auscultation of diastolic aortic murmurs. In this last lesion one also finds the lower end of the sternum a point of aortic transmission.*

The Suprasternal Notch.—The use of this area may enable one to hear weak heart sounds not audible over the selective areas mentioned above.

INSPECTION

FACIES.—No internal ailment presents more external signs to the observer than does established heart disease in many instances. In some forms the external signs are relatively prompt in appearance and apparent at a glance. In others they are obscure and relatively late or never appear.

Often strikingly objective.

The most strikingly objective lesion, over periods embracing many years,

Jerking vessels.

is aortic regurgitation, usually associated with pallor and meagerness of face, yet seldom with marked actual anemia. In such cases the jerky throb of the carotid, temporal, or any other superficial artery, may at once strike

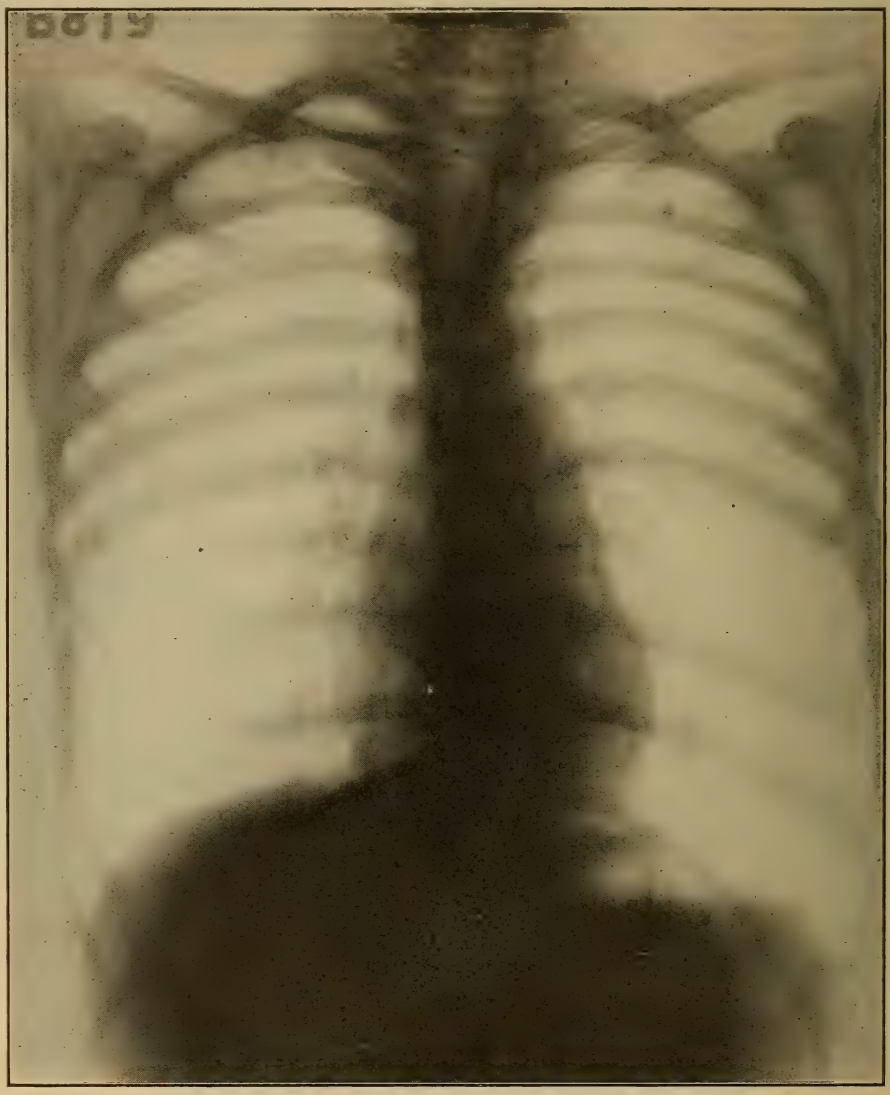


Fig. 161.—"Drop" heart with evidence of slight dilation. Note borders, equidistant from median line. Calcified focus in upper left lung field. A mitral systolic murmur was present in this case. (*Dr. Frank S. Bissell.*)

the eye, and even the head nods rhythmically in some cases or the foot jerks in time with the heart beat if the knees are crossed.

Capillary pulse.

In rare instances the eye may catch the rhythmic blushing and paling of the capillary pulse if some skin area becomes congested or even in the lips or nails.

One may find jerking vessels and a capillary pulse in thyroid cases,

lacking aortic regurgitation, but possessing overacting hearts and extreme vasomotor relaxation, and the condition of this gland should always be noted, together with the presence or absence of the peculiar stare, actual exophthalmos, or tremor, which may indicate a frank hyperthyroidism.

"Tracheal tug."

In rare instances the tracheal tug of aortic aneurysm may exist in so marked a degree as to catch the eye, or pupillary inequality or unilateral flushing or sweating may point to this or some other form of mediastinal tumor and resultant pressure.

Facial edema will of course be noted and in cardiac disease will seldom be marked, unless as a part of general edema or when actually due to a complicating or primary nephritis.

FIG. 162.—Peculiar orthopneic variant. Urgent dyspnea associated with listless rolling of the head from side to side. Usually a forerunner of death.

Mitral Stenosis.—Mitral stenosis is cyanotically objective in some instances and its outward signs are chiefly those indicative of oxygen deficit and may be closely simulated by advanced mitral regurgitation whether valvular or primarily myocardial, or, by certain pulmonary diseases which cause a relative blocking of the lesser circulation and insufficient ventilation of the blood.

Misleading "color."

In advanced mitral stenosis, especially, and particularly in young women, the rosy cheeks may appear vouchers for good health if the eye fails to note the duskiness which clouds the red and shows especially in the lips and ears.

A striking picture.

In terminal mitral lesions, especially, the countenance may appear blurred and an *orthopneic mitral case*, if breathing hard and *rolling the head listlessly from side to side is* almost certainly nearing the portals of the hereafter.

Ambulants.

All grades of cyanosis must be noted and investigated and, as stated previously, but three forms of *extreme* cyanosis in walking cases are encountered: That of the child with congenital heart disease, and advanced emphysema or the *red cyanosis of* "erythremia" in the adult.

Subicteric facies.

In old cases of right heart decompensation of long standing, a *subicteric* facies is common and underlain by a pallor, contrasting sharply with deeply cyanotic lips.

INSPECTION OF THE NECK AND TRUNK.—This should be both direct and *tangential*, general and local. The most important regions and the conditions to be noted are:

1. **The valvular areas** already described. Important pulsations may often be observed and their character may yield information of value.

2. **The manubrium and its neighborhood.** Here is found most commonly the *expansile* pulsation of aortic aneurysm.

3. The area lying between the inner edge of the left scapula and the spinal column, in which the heaving pulsation of an aneurysm of the descending thoracic aorta sometimes appears.

4. The Superior Abdominal Quadrants.—*In these we may find: The presence of manifest enlargement, pulsation, or rhythmic displacement of the liver; ascites; epigastric fulness, pulsation or retraction; pulsation of the abdominal aorta.*

The fulness and rigidity of vessels and the number, nature and time of pulsations in the veins of the neck must be noted.

General or localized edema, any evidence of marked sclerosis, local or general, venous or arterial, should catch the eye.

Bulging or deformities of any portion of the thorax together with its form and movements should be carefully investigated.

Dyspnea in all its forms from Cheyne-Stokes breathing, Biot's periodic apnea, or an exertion dyspnea, to a mere inability to hold the breath, is of importance and orthopnea is always a serious matter.

Area and position.

THE APEX BEAT.—This should be represented normally by a gentle rhythmic uplifting of the fifth interspace over an area not more than an inch in diameter, well within the mid-clavicular line.

Force.

It is not always present either in health or disease, because of overlying fat, a weak impulse, emphysema or an intervening rib, and the beat is often excessive, even in the case of a normal heart, because of temporary excitement, exertion, indigestion, narcotism, psychasthenia, and like conditions.

"Heaving" vs. "wavy" apex beats.

A heart that is the seal of extreme hypertrophy causes a distinct and widespread heaving (uplifting) impulse, and the beat of a badly dilated righ heart is wavy, diffuse and indeterminate. In either case the area of pulsation is greatly increased.

Reversed Apex Beat.—*In hypertrophy or dilatation of the right heart the right ventricle may wholly form the apex beat,* in which event there is visible in

Systolic recession.

the area between the left sternal and parasternal lines, a recession of the third, fourth and fifth or even the second left interspaces, along the region adjacent to the left sternal border, with each systole. In many such cases the left ventricle *does* reach the wall for a fleeting interval.

Extreme type.

In cases of extreme right heart dilatation, usually associated with relative tricuspid insufficiency, systolic *recession* may occur in the fourth and fifth *right* interspaces and even in the third, though the last is rare.

May deceive the eye.

Indeed the combination and coincidence of a visible left ventricular systolic apex beat with a right ventricular retraction is by no means uncommon in cases of mitral stenosis with thin chests and gives a peculiar appearance of *undulation.*

A misleading sign.

Various Precordial Retractions.—A strongly acting nervous heart and thin chest wall may cause systolic recession (right heart) in the second, third, fourth or fifth left interspace and in the upper portion of the epigastrium, even though the organ shows little or no departure from the normal outline,

* The lesser and commoner degrees of hypertrophy do not reveal themselves by a heaving apex beat.

but in such cases it is often associated with ailments which end to overtax the right heart, such as pulmonary tuberculosis, pulmonary fibrosis and emphysema of the lungs.

Congenital asthenia with psychasthenia and subnutrition, hyperthyroidism and profound anemias, are quite competent to produce such visible pulsations in thin chests and especially in those of reduced anteroposterior diameter. In most of these instances an atonic, irritable and overacting "drop" heart is present and the actual apex beat is systolic, diffuse, sharp, and left ventricular in origin.

Aneurysm.

Pulsation Adjacent to or Involving the Manubrium.—*Such an impulse, if marked and deliberate, always suggests aneurysm of the aortic arch, and pulsation of the manubrium itself is of special significance, because of its association both with aneurysm and certain vascular forms of mediastinal new growths.*

Mediastinal tumor.

Systolic Retraction Extending Beyond the True Apex Beat.—This is commonly mistaken for a systolic thrust and leads the unwary to overestimate the enlargement of an hypertrophied or dilated heart.

It may be caused by the drag of pleuro-pericardial adhesions with or without shrinkage or retraction of the lung itself, but can result when the contraction of any very greatly enlarged heart causes negative pressure areas in the lungs.

Solitary systolic pulsation over the second left interspace near the sternum is an exceeding common event as a result of right ventricular overaction. It is also observed early in certain cases of pericarditis and in the rare instances of pulmonary regurgitation.

Pulsation over the second right interspace is a well-known accompaniment of free and established aortic regurgitation. It is wholly different from the heaving expansile and usually deliberate pulsation of aneurysm of the first portion of the aortic arch.

Broadbent's sign.

Systolic Retraction in Mediastino-pericarditis.—It is one of the signs of adhesion of the pericardium to the heart itself, when associated with an indurative mediastinitis or general polyserositis. It causes a systolic drag upon the diaphragm, but to be of diagnostic value in this connection it should involve the posterior interspaces or ensiform cartilage as well and be unaffected by respiration (Broadbent's sign).

Precordial Lifting.—This may occur in great hypertrophy or dilatation or, in minor degrees, even in the case of strongly beating, excited, normal hearts in chests of small anteroposterior diameter.

The Position of the Apex Beat.—Abnormalities of position must follow inevitably any decided changes in the cardiac outline or actual displacement of the heart.

Displacement of the Apex Beat.—Upward. (*a*) *High position of diaphragm.* (*b*) *Tympanites.* (*c*) *Ascites.* (*d*) *Abdominal growths.* (*e*) *In some cases of pericarditis with effusion (often at nipple level).*

Upward and to Left.—*Effusion into right pleural sac;* (*a*) *Liquid.* (*b*) *Gaseous.*

Downward.—(*a*) *Aortic aneurysm.* (*b*) *Mediastinal tumor.* (*c*) *Senility.*

(*d*) *Hypertrophy of left ventricle (downward and to the left).* (*e*) *Collapse of abdominal viscera.*

To Right or Left.—(*a*) *Effusion of gas or liquid into pleural sac, i.e., pneumothorax, hydrothorax, etc.* (*b*) *Unilateral or vicarious emphysema.* (*c*) *Pleural adhesion and retracted lung.* (*e*) *Marked solid enlargements of lung or of the left lobe of the liver.* (*f*) *Respiratory lateral displacements may occasionally be visible in right-sided pleural effusion. These are best demonstrated by fluoroscopy, as originally reported by the author.*

Important in diagnosis.

The assumption of the lateral posture results in a corresponding displacement of even the normal heart, but is incredibly exaggerated in certain cases of "drop" heart (cor pendulum).

The apex beat may be invisible *by reason of:* (*a*) *Interposition of a rib.* (*b*) *Fat chest wall.* (*c*) *Feeble heart.* (*d*) *Emphysema.* (*e*) *Edema of chest wall.* (*f*) *Pericardial effusion (marked).* (*g*) *Pleural effusion.* (*h*) *Transposition of the viscera. In this condition the beat is present on the right side.*

Often misinterpreted.

Its area and apparent force may be increased because of: (*a*) *Mere overaction.* (*b*) *Hypertrophy or dilatation.* (*c*) *Retraction of lung.*

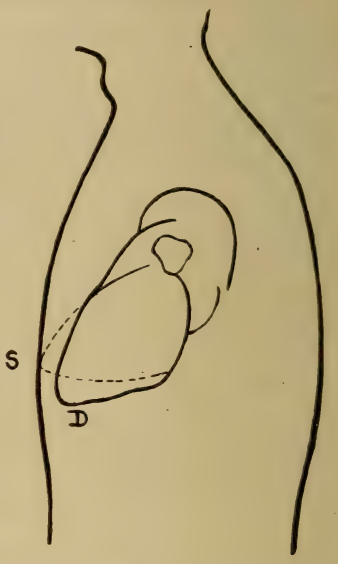


FIG. 163.—The production of the apex beat by the *normal* heart. (*After Staehelin and Ortner.*)

Precordial Bulging.—Aside from the presence of an actual growth this is usually due to three causes, viz.: aortic aneurysm, massive pericardial effusion, or, most commonly, excessive enlargement of the heart. It is most marked in those cases originating in early childhood in which the growing osseous structures have yielded readily to the pressure beneath. One may thereby often approximately fix the date of the heart lesion seen in advanced life. Such a precordial prominence is known as the "precordial boss" or "voussure" (arch).

Value in diagnosis.

Epigastric Pulsation.—This may be of several types: (1) *A systolic recession* as timed by the carotid beat, in *right heart dilatation* (Mackenzie).* (2) *A systolic pulsation,* as timed by the abdominal aorta, in certain cases of free aortic regurgitation, goiter hearts, anemias, or any case combining an overacting heart with decided vasomotor relaxation. A relaxed abdominal wall makes these pulsations obtrusively manifest. Transmitted aortic pulsation and true aneurysm are always to be considered. The latter is always *expansile and usually deliberate.* (3) A systolic protrusion in excessive *left heart hypertrophy.* (4) Actual systolic pulsation of the engorged liver as seen in certain cases of tricuspid insufficiency.

Right heart dilatation.

Aortic pulsation.

Left ventricular pulsation.

Hepatic.

* Mackenzie found that in one such case a needle, introduced after death, over the maximal area of epigastric pulsation, entered the right ventricle.

PALPATION

Scope and value.

Palpation serves to confirm and amplify inspection, and to detect *thrills* associated with valvular lesions or anemia, together with *points of tenderness* and the general characteristics of tumors of any kind. It is of special value in connection with the *expansile pulsation and "diastolic shock"* of aneurysm and the *apical presystolic thrill and systolic shock* of mitral stenosis.

Caution.

No examination of the diseased heart is complete that does not include the palpation of such related organs as the lungs, liver and spleen and determine the presence or absence of ascites in the presence of abdominal distention.

Aortic.

Pulmonary.

Mitral.

Congenital defects.

Thrills.—A systolic thrill in the second right interspace (aortic area) suggests aortic sclerosis, aneurysm or aortic stenosis. In the second left, if maximal at that point *and associated with extreme cyanosis*, it points to pulmonary stenosis, usually encountered in congenital lesions. Such thrills are common in exophthalmic goiter. Lacking cyanosis, a diastolic thrill in the second left interspace means aneurysm or aortic regurgitation. A presystolic or diastolic apex thrill is almost pathognomonic of mitral stenosis. A systolic *apex* thrill usually means mitral regurgitation; over the lower sternum or at its left edge, tricuspid regurgitation; and in children or young people, with profound cyanosis it points to congenital defects. The presystolic thrill of a tricuspid stenosis is very rarely encountered.

PERCUSSION

Never omitted.

Percussion is often decisive in the differential diagnosis of heart lesions.

One usually percusses from the more resonant to the less resonant areas, save in determining the superficial dulness and seeks to determine (*a*) *the deep or relative cardiac dulness;* (*b*) *superficial dulness;* (*c*) *the resonance of the region of the aortic arch.*

Abdominal distention and pulmonary hepatic or splenic engorgement are also important factors if the heart is abnormal.

Cardiac boundaries.

The prevalent "flat-finger" percussion must not be used, nor is any method unfailing in results, but by *orthopercussion*, auscultatory percussion, threshold percussion, or a combination of these, it may be very closely approximated in most cases.*

Manubrium.

The student should accustom himself to the sound of the normal dull tympany of the manubrium sterni, as this is one of the most important regions.

Tenderness.

He should also carefully note and localize any pain or tenderness attending percussion. Syphilitic periostitis, for example, may be encountered over the sternum and ribs and might prove of the utmost importance in a diagnosis.

In many atrophic or degenerated hearts, and in minor dilatation or mere atony, associated with chronic overstrain, tenderness often is marked at the left lower border and perhaps within it.

* In skilled hands the correspondence of outlines obtained by such methods of percussion to those obtained by long-focus roentgenograms (teleoroentgenography) or the orthodiagram is usually relatively close, *but in certain cases the grossest error occurs.*

During and following *Angina Pectoris major* most extensive areas of hyperesthesia may exist and often prove misleading if their *maxima* are remote from the heart itself.

Superficial Cardiac Area.—*The notched inner border of the left lung leaves a portion of the right ventricle uncovered and in such close proximity to the chest as to yield decided percussion dulness over a somewhat triangular space having its base at the left sternal margin from the fourth to the sixth chondrosternal articulation and its apex at or just within the apex-beat, the triangle being completed by lines connecting these points.*

Determination of the Superficial Cardiac Area.—*In determining the superficial cardiac area it is best to commence at its known normal center and percuss from that lightly and in radiating lines.*

Significant Changes in Percussion Area.—An increase in the area of superficial dulness may mean: (*a*) Enlargement of the heart. (*b*) Retraction of the lung. (*c*) Pericardial effusion. (*d*) *Pleural adhesions, solidified lung, or new growth.*

An Important Region.—*This area of marked dulness* is of great importance inasmuch as its extension to the right indicates clearly displacement of the heart as a whole, enlargement of the right ventricle, or pericardial effusion.

Enlargement to the right.

A valuable sign differentiating between displacement and enlargement of the right heart is given us by von Oestreich.

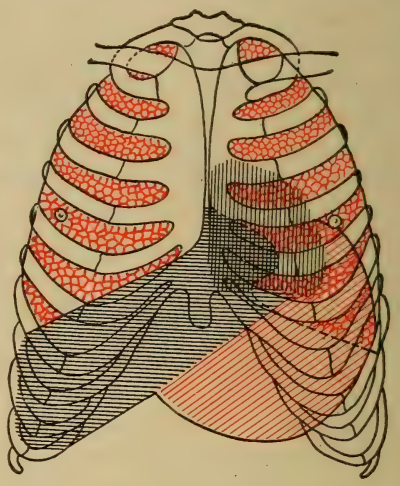


Fig. 164.—Superficial and deep relative cardiac percussion dulness, flat-finger method. This actually outlines only the exposed area (cross-hatching) and the general profile of the anterior surface of the right and left ventricles. More accurate delineation of the entire cardiac outline is now possible by means of a modernized technic, the results closely approximating the roentgenographic outline in most instances; though gross error is possible. (Repetition of figure 92 for added clearness.)

If the *superficial dulness* passes to the right as a straight border or one *concave and opening to the right*, we may assume that the heart as a whole is displaced or that a greatly enlarged left ventricle is crowding over its fellow of the right side (v. Jagic). If its right percussion boundary is broken by a *bulge* to the right the change represents right ventricular enlargement.

Extension to the left.

On the other hand, extension of the superficial area to the left alone or to the left and downward indicates left ventricular hypertrophy or dilatation, and if to the left and upward, an added left auricular enlargement. Cases in which decided right and left extension both are encountered are common as the result of combined valvular lesions.

In pericardial effusion it may be carried far beyond the right edge of sternum as a parabolic curve, joining hepatic flatness below.

Area of Relative Dulness.—It has been so difficult formerly to accurately outline the normal right heart that many modern diagnosticians still use the flat-finger method and the two arbitrary percussion areas shown in Fig. 163 as representing the normal, and these serve fairly well to determine and measure any variation in the precordial area of ventricular dulness. The orthopercussion method is far more accurate and greatly to be preferred. (See next page.)

Working outline.

The most valuable information with relation to the right border lies, as stated, in the extension of the *superficial* cardiac area to the right beyond the median line as indicating right ventricular dilatation or hypertrophy or pericardial effusion and the determination of an unduly wide extension of *relative* dulness to the right, as determined by orthopercussion, threshold percussion or auscultatory percussion, indicating right auricular dilatation.*

Value of orthopercussion.

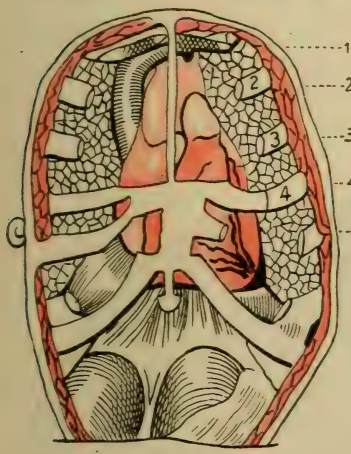


FIG. 165.—Note that in the normal heart the right border is formed by the right auricle. Compare with Fig. 158 ("drop" heart) showing right ventricle forming this border.

Value of the Sensation of Resistance.—In the case of the highly trained observer, percussion is almost as well carried out on the basis of the resistance felt as on quality and pitch of elicited notes.

The direct method of Ebstein is practically a soundless percussion, and in fact, *mere thrusting* will determine a cardiac or hepatic boundary with considerable accuracy and constitutes an excellent drill in concentration of attention.

"Tastpercussion."

The sense of resistance may be intensified by various forms of plessimeters which also sharply delimit the areas of differing resistance and pitch, or, by the use of the flexed finger in orthopercussion, which is quite as accurate and more convenient.

Firmness and equality in pleximeter pressures and plexor strokes over the contrasted areas under percussion is of cardinal importance.

The pressure may be varied to suit the conditions and need never be made painful to the patient.

An Uncertain Method.—*The old flat-finger percussion, still in general use, and most valuable for other purposes, is extremely uncertain and inaccurate in relation to right heart boundaries and the true left heart profile,* **and should be wholly abandoned in cardiac percussion.**

Threshold Percussion.—*Goldscheider's method of heart percussion demands the lightest percussion stroke audible to the percussor and is at present much used.*

"Schwellenwerts" percussion.

If a heart border be approached with a stroke so light as to be barely audible to the percussor's ear when applied over the adjacent resonant lung tissue, it should be entirely lost with the shortening of the vibrations induced

Threshold of audibility.

* Save as stated in the case of "drop" heart.

by an underlying heart border. The contrast between "little" and "nothing" is sharper than that representing mere differences of degree.

Preferred method.

It is difficult to carry out in any place not absolutely quiet and, in the author's hands, no more accurate than the combined method next to be mentioned, indeed much less so if the heart dulness passes far to the left.

It is an excellent method for the expert, but in using it the inexpert are likely to carry borders too far out. The greatest obstacle to its application lies in the difficulty of maintaining an exact equality in the force of the percussion strokes and the extremely quiet environment necessary to its most effective employment.

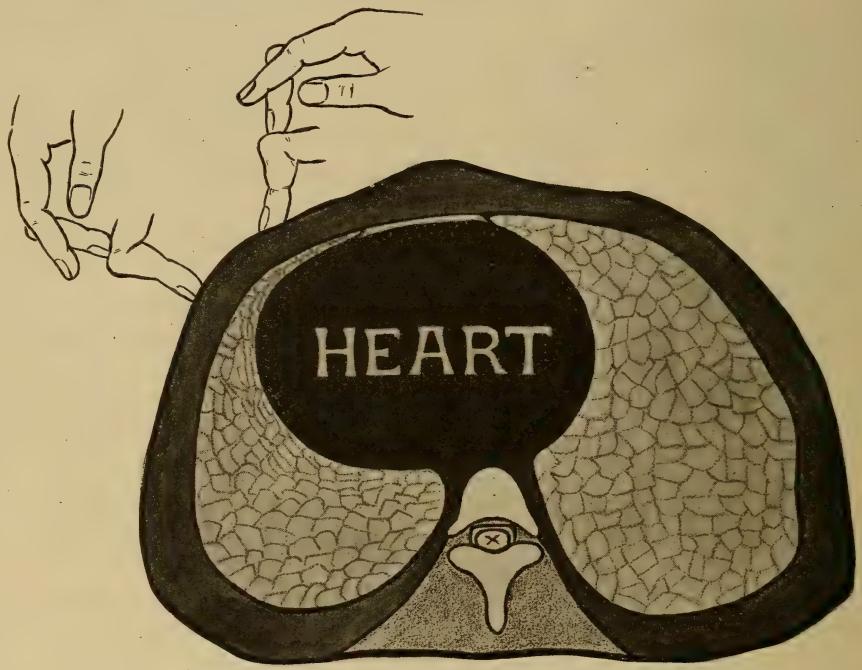


FIG. 166.—The illustration demonstrates the technic to be preferred in delineating outline by percussion. The actual stroke is, as nearly as possible a mere dropping of the wrist varied, as may be necessary, however, by a modulated stroke of bare audibility (threshhold percussion) or the strong stroke sometimes most effective. This may readily be combined with auscultation and constitutes a good method. The need of preserving the vertical direction of the stroke itself is manifest. *The upper hands* define the true profile boundary; the lower, a false one.

Orthopercussion.—*As previously stated, in the percussion of a widely extended left heart border in a chest which falls away at the sides before the limit of relative dulness is reached, one must maintain a stroke vertical to the anterior plane of the body rather than to the curving lateral surface of the chest.*

If this technic is not observed, one carries the border of a "cor bovis" around the curve of the chest into the axilla. One merely needs to lay off the distance thus obtained upon a centimeter rule to demonstrate the absurdity of this older method. He will find his heart border thus projected into space beyond the left thoracic border, *for he will follow the heart too far and elicit the relative dulness of the lateral aspect of the left ventricle* (see Fig. 166).

A measure of inaccuracy.

The older technic does not meet the modern demand for accuracy, emphasized by the frequent occurrence of great disparities between roentgenographic outlines and those obtained by the older methods.

Older methods inadequate.

Methods of Preference.—The author personally prefers to carry out percussion with a light yet firm, sustained and clearly audible stroke. The impact of the percussing fingers falls, not upon the base of the flexed terminal phalanx of the pleximeter finger as recommended by Korányi and Goldscheider, but upon the base of the second phalanx of the middle finger which is flexed at the proximal phalangeal joint and with the hyperextended tip applied firmly to the chest.*

The more nearly this represents the mere dropping of the hand, with a loose wrist, the more uniform are the strokes. If this method is employed, the tip of the pleximeter finger should be so firmly applied to the surface of the body as to overextend its terminal phalanx and render the vertical phalanges as rigid as possible, but need not constitute painful pressure save in exceptional instances.

Pleximeter finger.

Such percussion seems to check accurately with the "threshold" method, which should always be used secondarily and is preferred by many for right border percussion. One will find usually the former method the more useful and greatly value the sense of resistance and sharp definition which it yields.

According to the author's experience almost equally good results may be obtained *by lightly tapping the surface with the finger-tips* of one hand during auscultation over the heart, at distance, *i.e.*, combining *direct* percussion and auscultation. No assistant is needed if this last method be used. A procedure of still greater accuracy in the author's hands has been the use of orthopercussion by the method here described combined with auscultation over the heart. Results so obtained seem to check more accurately with fluoroscopic or plate work than any other.

A simple method.

The author personally violates established usage by percussing from within outward until the sudden and decided change of note marks the crossing of the frontier.

The student should try out the various modern methods and choose that which he finds most suitable to his own needs and most accurate in his own hands. Almost every clinician develops his own preferences and obtains better results by so doing. In the modern clinic many opportunities offer to compare percussion outlines with the chastening orthodiagraphic and teleoroentgenographic findings. He should remember however that when a busy practitioner he cannot always secure such quiet as is necessary to the use of threshold percussion.

AUSCULTATION

Auscultation aids in determining: (a) The condition of the heart muscle. (b) The competence and condition of its valves. (c) The abnormal increase or decrease of tension present in the aortic and pulmonary circuits. (d) The

* This technic is generally credited to Plesch, but has long been used by various clinicians as an alternative method.

condition of the lungs (congestion, edema, effusions). (e) Abnormalities of the aortic arch. (f) The presence or absence of murmurs in the tributary veins and arteries. (g) The presence or absence of extracardial sounds of cardiac rhythm. (h) The determination of abnormal sounds of cardiac origin, not the result of primary valvular defects.

The first and most important consideration is the *quality of the heart sounds* in the valvular areas and the carotid arteries.

Of fundamental importance.

Know and Seek the Normal.—*As in the case of the lungs, a common mistake in the teaching of students lies in the failure to drill them thoroughly in the normal characteristics of, or common variations in, the heart sounds. Students and physicians alike are too often satisfied if no murmur is heard, and do not seek and demand normal heart sounds and accentuation.*

Avoids blunders.

In no other way can we explain the frequent failures to recognize the serious significance of abnormal quality and accentuation, the decided diminution or almost complete absence of heart sounds, associated with some of the most serious lesions of the heart.

The student should possess a thorough knowledge of normal heart tones, permissible variations and normal heart border limitations, and must then primarily seek to determine their presence. Abnormalities existing will thus be emphasized.

HEART SOUNDS*

Systolic tone.

The First Sound.—A thorough understanding of the mode of production, quality and accentuation, of the normal heart sounds is absolutely essential to the intelligent interpretation of heart murmurs.

At the Apex the Normal Accentuation is on the First Sound; at the Base on the Second.—*Disregarding conflicting theories and fine distinctions, one may say that the essential elements in the production of the first sound are the initial, sudden sharp contraction of the ventricular walls and the coincident closure of the auriculo-ventricular valves.*

Diastolic tone.

The Second Sound.—*The second sound is produced by the simultaneous closure and tension of the aortic and pulmonary valves immediately following ventricular contraction.*

Hence this second sound must primarily depend upon the integrity of the valves themselves, and measure the amount of that recoil which is the resultant of the forces of propulsion and the resistance encountered in the artery.

Abnormal Accentuation of the First Sound.—*It follows that abnormally increased accentuation of the first sound, as heard in any of the four auscultation areas, usually indicates an overacting ventricle whether this overaction is temporary, and caused by excitement or overexertion, or persistent, and due to mere increase in strength, as in hypertrophy from valvular disease, to excessive radius of contraction of a partly filled ventricle, as in mitral stenosis, or to aortic rigidity or heightened arterial tension, as in arteriosclerosis and interstitial nephritis.*

Pulmonary and Aortic Accentuation.—*The aortic valve being sound, any increase in general arterial tension will produce an accentuation of the aortic*

*See also "Rationale of Organic Heart Murmurs," page 455.

second sound and similar increased pressure in, or obstruction to, the pulmonary circulation will result in an accentuation of the pulmonary second sound.

The degree will depend largely upon the soundness of the valves, the strength of the ventricles and the integrity of the mitral and tricuspid leaflets. The second sound may also be markedly intensified in sclerosis of the first portion of the aorta or pulmonary artery.

"The Diastolic Echo."—The *"third sound"* reported by Barrie in 1893, and again by W. S. Thayer and the late Dr. Geo. Gibson of Edinboro in 1906, is present occasionally in normal individuals; is often extremely faint; and audible over the apex in certain slowly beating hearts when the patient is lying on the left side. It seems to correspond in time to the protodiastolic "h" wave of Hirschfelder as occasionally seen in the venous tracings of the polygram. It constitutes the protodiastolic element of the "protodiastolic gallop-rhythm" and has long been recognized as occurring in certain cases of mitral stenosis and aortic regurgitation, but though interesting is of slight practical importance.

Of slight importance.

The term "diastolic echo" well describes it, for it immediately follows the second sound and probably corresponds to the termination of the primary rapid rush of blood into the ventricles and preliminary apposition of the auriculo-ventricular valves (Hirschfelder).

Probable cause.

Practical Applications.—Much information is afforded in both acute and chronic disease by a study of the variations in accentuation. For example, lobar pneumonia must inevitably be accompanied by marked accentuation of the pulmonary second sound and a dangerous tendency to dilatation of the right ventricle. If this dilatation becomes extreme the pulmonary second sound is markedly weakened and if tricuspid regurgitation occurs it may be wholly lost, a sign of the gravest import.

Pneumonia.

Tricuspid leakage.

The pulmonary congestion incident to mitral stenosis and regurgitation makes accentuation of the pulmonary second sound an important diagnostic feature, and, as in pneumonia, a loss of this accentuation or of the entire sound may be a serious symptom.*

Mitral lesions.

Changes in Timbre.—*A tone may be weak, short, sharp, muffled or distant, "murmurish" or impure, hollow or ringing, "slamming," "fetal" or metallic.*

Important variants.

The first sound, as heard at the apex, may be wholly lost or replaced by the murmur in some cases of mitral regurgitation or aortic stenosis. To a less degree, aortic incompetence tends to obscure the second sound alike in the aortic area and in the carotid artery, though in the latter lesion a marked accentuation may be present if a high degree of sclerosis is present in the cusps, or when, in spite of incompetence, the valve segments still swing freely and achieve an imperfect apposition.

Modified 1st sound and aortic 2d.

It is probable that in *most instances of diminished intensity* some degree of actual aortic stenosis co-exists.

In mitral stenosis the aortic second sound is weakened, the mitral first sound

* As stated elsewhere a diminution of the pulmonary second sound accentuation in mitral regurgitation may mean a small leak or a powerfully acting left ventricle which keeps the pathway clear.

at the apex being accentuated and sharp or slamming unless extreme regurgitation co-exists.

In aortic regurgitation and stenosis the first sound at the apex is dulled and in combined mitral and tricuspid regurgitation all sounds are greatly diminished.

An excessively weak heart may yield relatively loud, *sharp*, *short* tones if, as is usually the case, the rhythm is rapid and hurried.

Lost or obscured tones.

A distinctly weakened first sound at the apex, in the absence of emphysema or purely external factors interfering with sound conduction, should always *suggest* cardiac weakness with or without *silent leakage*, and demands a thorough investigation of the cardiac area and the symptoms presented by the patient, *whether these be subjective or objective.*

"Metallic clacking" is frequent in tachycardial arrhythmias, especially if the stomach be distended by gas. **Fetal sounds** and a slamming mitral first sound are always pathologic. *A ringing*, accentuated second aortic tone is often associated with aneurysm of the arch; commonly, with sclerosis of the vessel, high arterial tension and, in many instances, with pure aortic regurgitation.

Multiple factors.

Murmurish Sounds.—The same may be said of *persisting* "murmurish," impure, unduly *sharp and short*, or muffled sounds, though due allowance must be made for mere pulse acceleration and disturbances of rhythm.

Acute Infections.—*In acute infections* such as rheumatism, prostrating diphtheria, influenza, scarlet fever, and the like, both during the attack and *in and after convalescence*, the heart must be carefully watched for signs of serious myocardial or endocardial mischief, *such as may be indicated only by subjective weakness or dyspnea, minor dilatation, and a labile pulse.*

A duty often disregarded.

Mere Muffling of Both Sounds.—When this is evident especially at the apex, it may be due to a fat or edematous chest wall, to emphysema, exudative pericarditis, fatty overgrowth, serious myocardial disease, or cardiac displacement.

Attention to these changes in quality, intensity and character of the heart sounds are of special importance in connection with early endocarditis, acute or subacute myocarditis and the silent, often abrupt, dilatations and insufficiencies so often encountered in chronic primary myocardial degenerations, atrophy, and in the narrow ptotic hearts of congenitally asthenic individuals.

Displaced Heart.—In fibroid phthisis, pleural effusion, mediastinal growths, diaphragmatic hernia or even extreme meteorism or ascites, the cardiac displacement may be suggested by the changed site of audibility of the sounds or murmurs, the whole mediastinum being oftentimes markedly displaced.

Deceptive conditions.

One can seldom with certainty assign to the individual valves, murmurs heard under conditions of cardiac displacement, as they may disappear wholly or in part when the dislocation is corrected. The pressure of pleuritic effusion and that of ascites or meteorism offer the most frequent examples.

In excessively dilated hearts, and especially in such of these as show marked associated arrhythmias, an accurate primary interpretation, differentiation and summary of the murmurs, and hence of the individual lesions present, is usually impossible.

A Useful Maneuver.—It is well to become accustomed to the auscultation of the normal heart sounds as heard in the median inferior hollow of the neck "*fonticulus gutturis*," as one may often test there the legitimacy of the basal heart sounds.

Increased Audibility.—Aside from cardiovascular causes already given, this may result if the lung is retracted from the heart, and hence at times coincides with a change in the site of **maximum audibility,** *e.g.*, fibroid lung or

Extracardia causes.

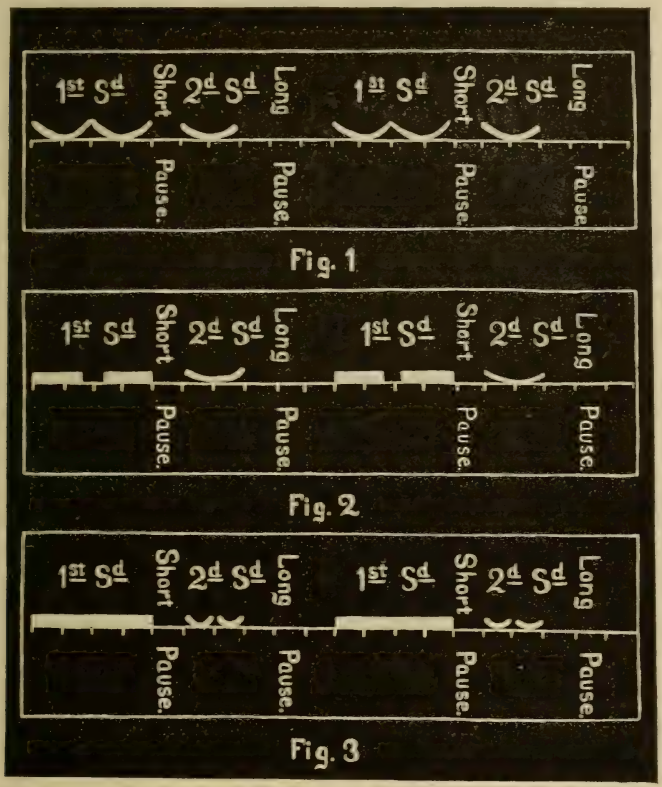


FIG. 167.—Division and reduplication of the heart sounds. Upper figure.—Splitting of first sound (*blup* dup). Mid-figure.—Division of the first sound apex (*lupup* dup). Lower figure.—Division of second sound (Lub *letup*).

adjacent excavation. Sounds are occasionally transmitted loudly and over wide areas by such pulmonary consolidations or cavities.

Reduplication of Heart Sounds.—*The normal heart sounds consist of two systolic and two diastolic sounds so blended as to form one systolic and one diastolic tone because of the synchronous closure of the valves of the right and left heart.*

Under many conditions this synchronism is so interfered with as to produce splitting or actual reduplication of either sound.

In true reduplication the elements of the double sound are clear and well-defined though short. In other words, a third sound appears in the cycle.

The triple rhythm.

The split sound elements are less clearly separated and sometimes cannot be differentiated from the shortest possible murmur.

The phenomenon of reduplication has been compared aptly to the sound produced by the asynchronous closure of double swinging doors. A normal heart, if temporarily overacting, may be the seat of doubling or splitting of the heart sounds, but, in general, it may be said that, hearing an apparent reduplication of the *second sound* (a presystolic third sound) at the apex only,

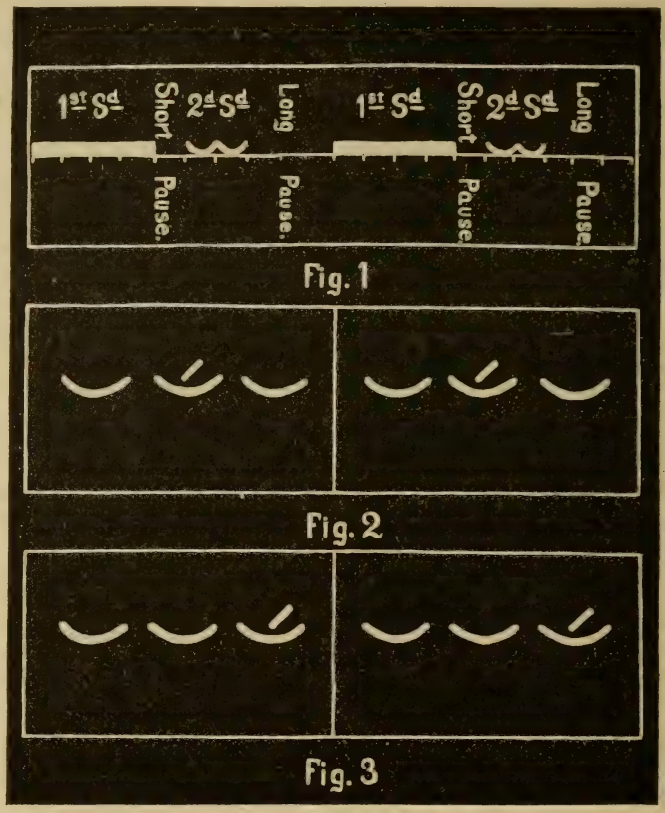


FIG. 168.—Reduplication and division of the heart sounds. Upper figure.—Reduplication of second sound (lub tlup). Mid-figure.—Presystolic gallop rhythm (lup *lup* dup). Lower figure.—Proto-diastolic gallop, rhythm (lup lup *lup*). Gallop rhythm is always a suggestive and serious phenomenon and probably represents in many instances a unilateral block. (See "Heart-block.")

and especially if it be associated with diastolic blubbering or a *bruit*, one may assume that it is due to the presence of mitral stenosis.*

If a reduplicated second sound is heard over the aortic area and, perhaps, the whole heart, myocardial degeneration often combined with cardiac overstrain and arterial hypertension is the probable cause.

Curiously circumscribed.

A Split Second Tone.—A split second sound at the base, *limited usually to the third left interspace*, has been found by the author with especial fre-

*Less commonly in regurgitation unless associated with myocarditis of marked degree or arteriosclerosis.

quency in slightly dilated or merely atonic insufficient hearts, usually with a history of past temporary prostration from acute illness or overstrain.

It has been present in certain cases of early luetic aortic disease, in the "drop" hearts of persons of the congenitally asthenic type, who so often present the picture of so-called "neurasthenia," and in middle-aged males with minor signs of arteriosclerosis and various grades of dilatation with or without arterial hypertension. It has been associated invariably with a distinct capillary pulse and increased "pulse pressure." An intermittent short diastolic murmur in the third left interspace is occasionally observed which may wholly disappear under treatment, revert from time to time to the original split second sound, or, especially in middle-aged patients, become after months or years a persistent, short murmur of aortic regurgitations.*

Many of these "split-second" cases, with or without "drop" hearts, have been under the observation of the author over long periods. The periods of to-and-fro shifting from the split sound to the actual diastolic *bruit* sometimes observed are most interesting.

Fetal Rhythm.—(*Embryocardia*).—In this the heart sounds are rapid and equally distant, resembling the actual fetal heart sounds, and the condition has been well named "embryocardia" (Stokes-Von Huchard). The expert clinician recognizes its significance and importance, *as indicating a marked enfeeblement of the heart muscle associated with excessively rapid contractions.* Certain cases of auricular flutter, alternation and paroxysmal tachycardia must be placed under this head.

Ticking rhythm.

Auscultation Areas.—*The four primary surface areas for auscultation are:*

1. **The mitral area** (*apex*).
2. **Tricuspid area** (*lower half of sternum*).
3. **Pulmonary area** (*second left intercostal space*).
4. **Aortic area** (*second right intercostal space and along the line connecting this point with the third left interspace*).

As stated previously, these points, all adjacent to the sternum, do not correspond to the exact anatomic location of the valves which may be all included in the area of a large stethoscope bell, but represent the region in which the sounds of the respective valves or their associated murmurs are best heard.

HEART MURMURS.—Definition.—A heart murmur is an extraneous sound of cardiac site and rhythm tending to obscure, partly or wholly replace, or immediately or laggardly precede or follow, a heart sound.

* The author feels that possibly minor aortic insufficiencies, transient or permanent, may be less rare than has been believed and has found no clinical evidence to sustain the theory that a pulmonary insufficiency is responsible for the murmurs, or, that in young individuals, an aortic ring which yields under excessive temporary strain is of necessity persistently damaged.

Any one familiar with the "drop" heart and its modifications must recognize the special liability of its possessors to cardiac overstrain, especially when such individuals form as they do a considerable proportion of those seeking athletic honors and entering into the more strenuous forms of labor and recreation alike.

A reiteration for emphasis.

Common Sources of Error.—*It should never be forgotten that, as stated, all the extraneous sounds produced by a heart that is overacting, excessively weak or dilated, tumultuously beating, or mechanically dislocated, can seldom be accurately interpreted. Under such conditions, existing organic murmurs may be obscured or unplaced in rhythm; or, as more commonly happens, murmurs may*

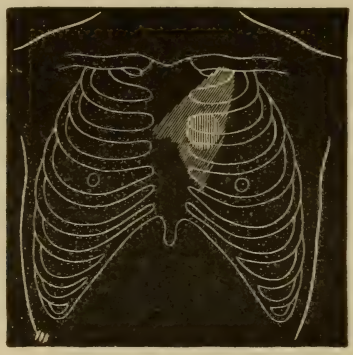


FIG. 169.—Usual site of murmur associated with anemia and asthenia. (*Sansom.*)

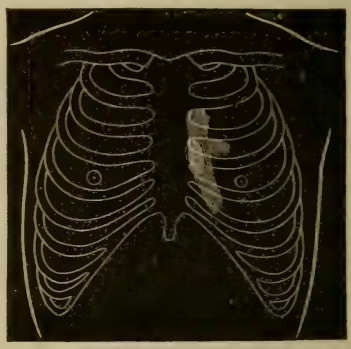


FIG. 170.—Murmur over conus arteriosus in anemia and myocardial asthenia.

be present, purely temporary in character, which may be and often are wrongly regarded and treated as organic.

The utter impossibility of accurately differentiating all the murmurs heard over extremely weak and widely dilated or dislocated hearts should be better understood.

Transient Murmurs.—*Transient dynamic or accidental murmurs and harsh heart sounds, associated with abnormal accentuation, are extremely common.*

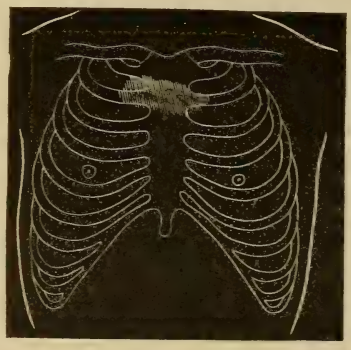


FIG. 171.—Murmur limited to (*supra*) aortic area in anemic cases. (Eleven per cent. of cases, according to *Sansom.*)

FIG. 172.—Coexisting pulmonary and apex murmurs in anemia. The apex element must be viewed with suspicion in all such cases of assumed hemic origin. Sansom held such to be true mitral regurgitations.

They call for rest, reassurance, the administration of test doses of such drugs as digitalis and the bromides and always for repeated examinations.

Attitude important.

Postural Modifications.—*All patients should be examined, if possible, both when recumbent and when erect, after a rest period, and, if their condition permits, after some brief brisk exercise.*

The murmurs of valvular leakage are *usually* best heard in the recumbent position, those of narrowing (obstruction) in the erect posture; while sometimes, as in the case of mitral or tricuspid stenosis, a typical *presystolic* murmur may appear only at the moment when the patient reaches the upright position and then rapidly subside. The diastolic murmur of aortic leakage often may be increased by raising the arms above the head or may require brisk exercise to make it audible. Useful maneuvers.

The Intensity of the Murmur.—*The stronger the heart and the narrower the affected opening, the louder and higher pitched is the murmur, and, speaking roughly and broadly, it may be said that the louder the endocarditic murmur the better is the prognosis, inasmuch as it often indicates an hypertrophy of the chamber most affected and most vital to compensation.* Loud vs. soft.

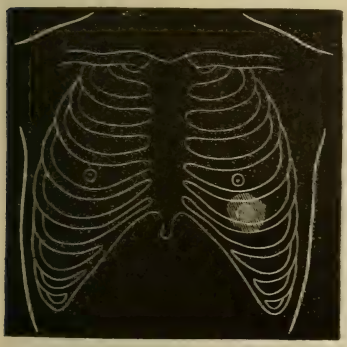


Fig. 173.—Systolic murmur, at apex *only*, in cases of anemia. All such murmurs justly may be regarded as only in part, if at all due to the physical condition of the blood. They probably represent actual though often transient and atypical mitral regurgitation.

The general rule previously stated is, of course, a rough one subject to many exceptions, nor would it cover certain excessively loud or even musical murmurs which are often associated with and derive their loudness and peculiar quality from calcareous vegetations. Murmurs must, in any event, take their pitch and timbre in large degree from the form of the orifice and the density, tension, shape or surface irregularities of its margins.

Cases with manifest signs of inveterate and extreme cardiovascular disease, but yielding faint murmurs elicited with difficulty, or those in which no murmur is present though the heart is manifestly incompetent, are oftentimes of the most serious import, as indicating extreme myocardial exhaustion and at times an excessively large leak. Faint murmurs.

Variations in Timbre.—The greatest possible diversity of timbre is encountered in murmurs of cardiac origin. "*Blowing*" *of all degrees*, "*sighing*," "*cooing*," "*gurgling*," "*whistling*," "*hissing*," "*sawing*," "*grating*," "*musical*," "*squeaking*," "*blubbering*," "*rolling*," "*roaring*," *and* "*rumbling*," are some of the terms quite applicable to the varied sound expressions of cardiac regurgitations and stenoses quite apart from the "shuffling," "creaking," "squeaking," "grating" and "rubbing" of pericardial or pleuro-pericardial friction.

ASTHENIC AND "HEMIC" MURMURS.—The murmurs associated with decided anemia are *systolic* in time and soft and blowing in character,* seldom widely transmitted, and usually best heard over the pulmonary area (second left intercostal space). Related to 1st. sound. Usual maximum.

* Rarely they may be loud and harsh as in a case of exsanguination due to acute hemorrhagic gastric ulcer observed by the author. In this case the murmurs were auto-audible and to be heard when the ear was several inches from the chest.

Lack of transmission.

They are heard less frequently over the apex, aortic area, etc., and such apex bruits differ from the true murmur of *endocarditic* mitral regurgitation in the fact that they are usually softer and less clearly defined; are almost invariably associated with a *more decided* murmur of the same type in the pulmonary area, and have not the same transmission to the axilla and back.

They do not, however, differ from many relative and consecutive mitral insufficiencies, either as to quality, or imperfections in transmission.

So also, when heard over the aortic area proper (second right intercostal space), they lack the transmission into the carotids and subclavian, characteristic of true aortic stenosis.

Associated anemia.

Their association with anemia and disappearance under appropriate treatment further serve to differentiate them in practice from true *endocarditic* murmurs, but involves as well factors quite apart from the condition of the blood. Associated dilation is far from uncommon and is itself often attended by apex murmurs of a similar quality in undernourished individuals of the asthenic habitus, *even when any marked degree of anemia is lacking.* A few cases of diastolic hemic murmurs have been reported.*

Adequate Factors.—The author can see no vital objection to the assumption that, in *marked degrees* of anemia with or without associated asthenia, a lowered specific gravity and diminished viscosity of the blood combined with hypotonia, and a rate of flow which may reach five times the normal, may readily produce audible murmurs.

On the other hand, it would appear to him that, in the greater number of instances, other associated factors play a large part; for such murmurs often persist after all signs of anemia have disappeared, subsiding only when coexistent general muscular weakness and subnutrition have been overcome.

Cardiopulmonary murmurs.

The author believes, therefore, that many of the so-called accidental murmurs heard at the apex or over the superficial cardiac area, now classed as hemic, cardiopulmonary and the like may be in whole or part murmurs of papillary muscle insufficiency due often to mere temporary heart muscle atonicity.

Papillary muscle insufficiency.

In some instances the yielding of these structures might throw the murmur into the mesosystolic or prediastolic phase (post-systolic) which is precisely the rhythm of "cardiopulmonary" murmurs.

In the anemias of pronounced congenital asthenia the elements of myocardial atony and slight chronic or recurrent dilatation are usually superadded.

The great majority of such *persisting* murmurs of apical site act with respect to audibility as exactly like endocarditic murmurs as they could be expected to do when produced by the insufficiency of structurally *normal* valves and constitute most valuable signs when occurring *in association with subjective symptoms of myocardial inadequacy.* They, of course, merely reflect deficiencies of tonus but *these may be associated with myocardial*

* One case of typical diastolic murmur of aortic localization reported by the author occurred in the terminal stage of pernicious anemia, the autopsy showing a normal valve. He would now regard it as a dilatation of the aortic ring.

degeneration, and if we are not to note these deficiencies we are not to go far in early diagnosis. Furthermore their changes under treatment are exactly what one expects under the foregone assumption and not in the least what would be the case were they cardio-respiratory or cardio-pulmonary.

The author does not believe that, in general, murmurs heard over the *apex* of the heart, even though they lack the quality, transmission, and secondary signs of organic valvular lesions are to be classed as trivial, especially in the case of men and women above thirty or thirty-five years of age.

To admit this is to permit the elimination of an important symptom of minor myocardial insufficiency associated with actual disease.

A review of recent literature balanced against a large personal experience with bruits of this type has convinced him of the falsity of any such assumption, whether the murmur heard be directly systolic or post-systolic. It is to be feared that there is a general lack of understanding of the readiness with which, in the tonus deficiencies, the mitral ring or the papillary muscles become incompetent and permit leakage in a structurally sound valve.

Impairment of tonus.

One is impressed by the apparent lack of appreciation of the extreme readiness with which myocardial tonus is impaired even in health in the vastly greater number of instances temporarily and harmlessly. Any exertion which is excessive for the individual and produces profound fatigue means temporary trifling impairment of tonus. Excessive heat and vitiated warm air affects it in many individuals. Nutritional deficit tends to impair it. Psychic stress, strain and shock, if intense, may be subtly as potent as physical overstrain; and finally, the first effect of toxemia from whatever source upon heart muscle is the reduction of tonicity. One has only to review experimental work done not only upon the heart but upon other hollow muscle organs to realize how exquisitely sensitive such tissue is to such influences.

In the diseased or congenitally inadequate hearts, every fresh accession of an existing concealed septic focus; any decided advance in an already seated inflammatory or degenerative process, will affect to a greater or less degree myocardial tonus and consequently the reserve power of that heart. Many or most of these impairments will be temporary and trivial, but from time to time those of a severer grade or greater persistence occur, the existing pathologic process is accelerated, or the primary congenital weakness is intensified.

As stated previously, the more pronounced systolic hemic murmurs have their maximum at the pulmonary area in nearly every instance and hence simulate in time and location a murmur of pulmonary stenosis, but the harsh murmur of this rare and almost invariably congenital lesion is wholly different in intensity, in its common association with a systolic thrill, intense cyanosis, and other striking and extreme clinical phenomena.

Other Forms of Accidental Murmurs.—A cardiorespiratory or cardiopulmonary murmur is a "systolic whiffing" heard best during inspiration

and in the region of the *left lung border*, along the lines of the maximum recession due to the systolic contraction and recession of the right ventricle.

It is superficial and usually mid-systolic or post-systolic in time and is most likely to be heard when the heart is overacting. If true to type it should be absent in forced expiration over the area of cardiac flatness or move inward in full inspiration and outward in expiration.

These murmurs are supposed to be due to the influx of air into interposed lung during right ventricular systolic recession.

Their frequency is probably not nearly as great as has been supposed but, undoubtedly, they deserve mention.

Pleuro-pericardial Murmurs.—*When the apposed layers of the pleura and pericardium become inflamed in the extension of a pleuritis or pericarditis, a murmur may be present which is usually distinctly frictional and superficial in character and heard best usually along the border of the superficial cardiac area.*

This sometimes endures for a long time after the original disease subsides, usually lacking the quality, and, almost invariably, the transmission, of a valvular murmur. The murmur corresponds to cardiac rhythm but is distinctly affected by the phases of respiration. It is obvious that the disappearance of such a murmur in full inspiration would indicate that contact of the costal pleura and pericardium was responsible for the murmur and that this has been destroyed by the interposition of the inflated lung.

Disappearance of the murmur in full expiration would indicate that it arose from contact between the pulmonary (visceral) pleura and pericardium which has been broken by the expiratory recession of the lung.

Conversely the appearance or intensification of such a murmur in full expiration indicates inflammation of costal pleura and pericardium and its appearance or intensification in full inspiration points to involvement of the visceral pleura and pericardium. The sign is occasionally one of great importance as indicating a more or less sinister extension of an acute or chronic pericardial or pleural infection or the presence of tuberculosis.

Curious Crackles.—*Some curious showers of tiny crackling murmurs* may be heard rarely along the left edge of the sternum, particularly in connection with advanced and extreme pulmonary emphysema.

Mediastinal Emphysema.—The presence of air in this space may be due to external injury, the induced pneumothorax at present so much in vogue, or to ulceration or rupture of any portion of the respiratory tract in direct relationship.

There may be subcutaneous or subfascial crepitation, often first observed in the cervical region, or merely crepitation of cardiac rhythm and loss of heart dulness.

Splashing.—*If the pericardium contains both air and liquid, churning or splashing sounds of cardiac rhythm may be audible. This may be simulated by sounds produced in large contiguous cavities as in marked gastric distention, pneumothorax and advanced phthisis, but the true murmurs are so bizarre and peculiar as to be practically pathognomonic of the pericardial condition.*

THE RATIONALE OF ORGANIC HEART MURMURS.—Varieties.—*Organic murmurs may be valvular, arterial, myocardial, pericardial or pleuro-pericardial in origin.*

The valvular murmurs are of two kinds, namely, the *regurgitant and the obstructive;* the one being due to a leakage and back flow, the other to a narrowing of the valvular opening, a stenosis.

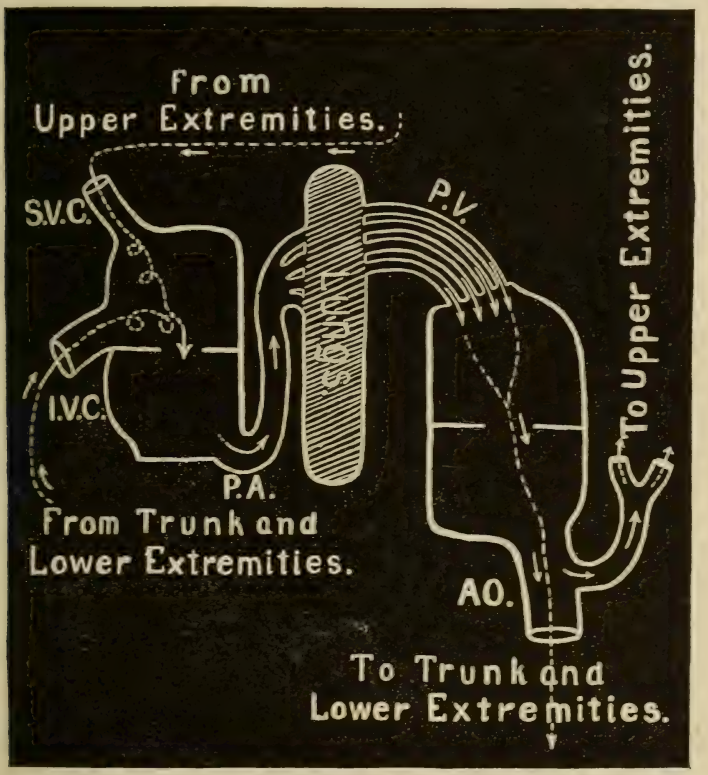


FIG. 174.—The right and left hearts. S.V.C. Superior vena cava. I.V.C. Inferior vena cava. P.A. Pulmonary artery. P.V. Pulmonary veins. AO. Aorta. The lungs are in the position of a self-cleansing blood filter and purifier, receiving venous blood from the right ventricle and delivering arterial blood to the left auricle.

Preliminary Remarks.—For the sake of clearness and vividness of description in relation to the production of heart murmurs, no effort to attain a finically technical description is attempted.

The student needs to create primarily a simple mental picture such as he may retain and readily reproduce when in contact with the patient.

Mode of Production.—*The heart must be regarded simply as a double pump in constant action, or, as two double-chambered hearts, firmly united and synchronous in action. It may be represented diagrammatically as two hearts with the lungs between them;* the systemic arteries, capillaries, and veins

Cardiac pump

Relation of lungs.

carrying the blood from the left ventricle through the tissues and back to the right auricle, whence it passes to the right ventricle, is driven by it through the lesser (pulmonary) circuit and returned to the auricular reservoir of the left heart, for such is the plan of the circulation.

Ventilation.

Practical Synchronism.—Pouring constantly into the right and left upper chambers, respectively, comes the blood from both the greater and lesser circulations, led through the lower and upper caval conduits to the right auricle and through the pulmonary veins to its fellow of the left heart.

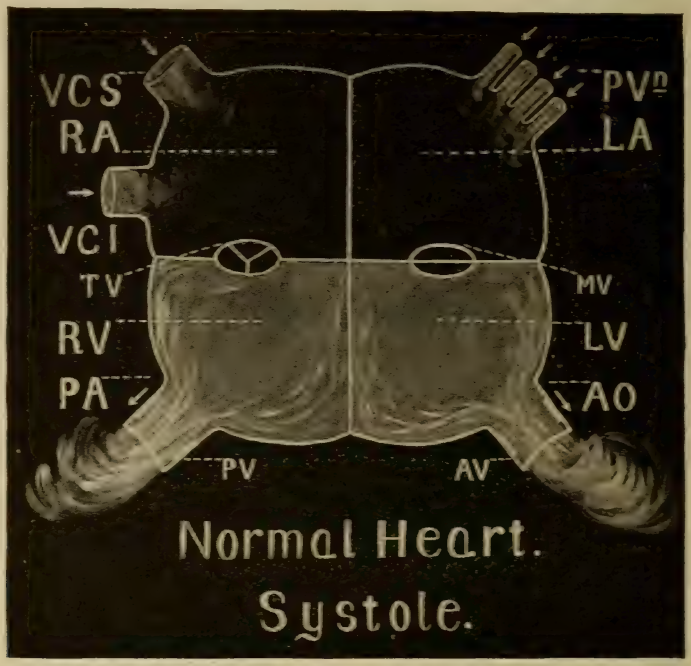


FIG. 175.—The normal heart in (beginning) systole. The ventricles, filled with blood which they have received from the auricles during diastole, are now contracting and driving their contents through the semilunar valves, just now forced open, into the pulmonary artery and the aorta. The mitral and tricuspid valves have just closed (first sound), shutting off any reflux from the ventricles to the auricles which latter must fill during the period of this ventricular contraction (*systole*). M.V., mitral valve. T.V., tricuspid valve. A.V., aortic valve. P.V., pulmonary valve. L.A., left auricle. R.A., right auricle. L.V., left ventricle. R.V., right ventricle. V.C.S., superior vena cava. V.C.I., inferior vena cava. AO., aorta. P.V^{s}., pulmonary veins. P.A., pulmonary artery.

Course of blood.

Into the right auricle the superior and inferior venæ cavæ pour their dark, impure, venous blood, while to the left auricle the four pulmonary veins bring a constant supply of bright, red, arterial blood that has undergone ventilation and purification in the pulmonary air cells.

Ventricular contraction.

During the recurring periods of ventricular relaxation (*diastole*) which follow each ventricular contraction, the blood passes from the auricles into the ventricles through the opened auriculo-ventricular valves; *the mitral on the left, the tricuspid on the right.*

Systole.—With the *initiation* of systole (presphygmic period) these valves abruptly close (**first sound**) the inlets to prevent backflow into the auricles and the blood is forced by the continuing ventricular contraction (systole) along the paths of least resistance, *i.e.*, through the pulmonary valve and into the pulmonary artery on the right, and through the aortic valve and into the aorta upon the left; these exits remaining fully open during systole, but shutting smartly as the systolic ventricular contraction ends (*second sound*).

First sound initiates systole.

Second sound initiates diastole

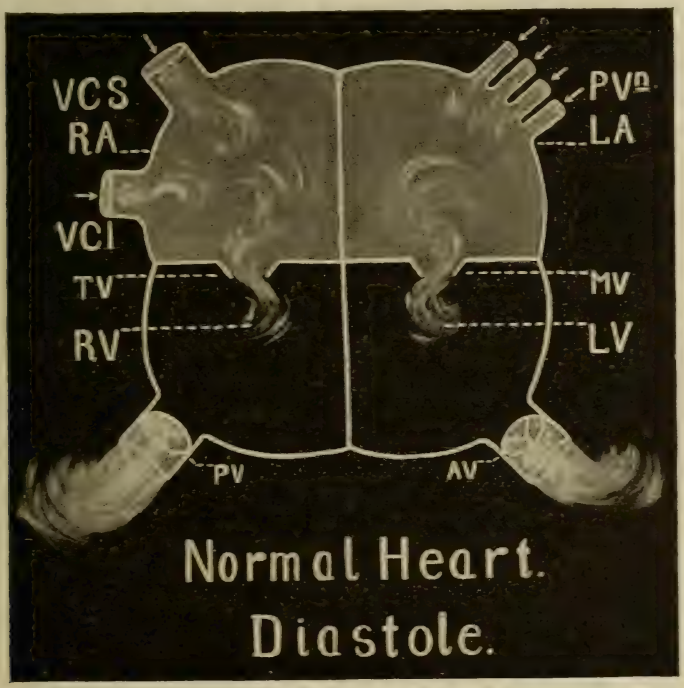


FIG. 176.—The normal heart in beginning diastole. The ventricular contraction has ceased, the aortic and pulmonary valves, closed (second sound), are shutting off and supporting the blood column; the ventricles are filling from the open mitral and tricuspid orifices above. M.V., mitral valve. T.V., tricuspid valve. A.V., aortic valve. P.V., pulmonary valve. L.A., left auricle. R.A., right auricle. L.V., left ventricle. R.V., Right ventricle. V.C.S., vena cava superior. V.C.I., vena cava inferior. P.Vn., pulmonary veins.

Blended Sounds.—As both sides of the heart contract simultaneously, or practically so, the sounds produced by each double valvular closure whether of systole (mitral and tricuspid) or diastole (aortic and pulmonary), are ordinarily coincident and blended into one "tone" for each of these phases.

The "first sound" *obviously corresponds to the initiation of the period of ventricular contraction and the complete closure of the mitral and tricuspid valve; i.e., systole.*

Its time is practically that of the carotid pulse beat, a fact of much clinical importance.

Diastole.—The instant that this systolic contraction is completed the

aortic and pulmonary gateways close, the emptied ventricles relax, the mitral and tricuspid open. The aortic and pulmonary valves must be shut smartly to prevent backflow into the ventricles from the overfilled and distended great arteries, and thus they produce the **"second sound"** which *initiates "diastole."* During this, the period of ventricular relaxation, the blood that has been accumulating in the auricles during ventricular systole pours down through the opened mitral and tricuspid valves to fill the emptied ventricles,

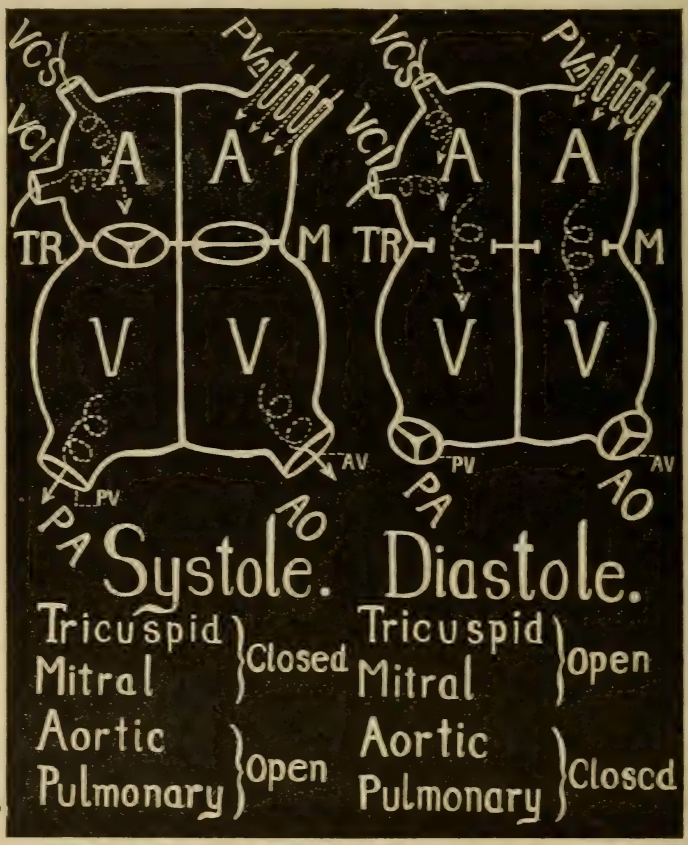


FIG. 177.—Position of the valves of the heart in beginning systole (first sound) and diastole (second sound).

Auricular contraction.

and just before the next systole the auricles themselves contract vigorously and ventricular systole almost immediately follows.

Presphygmic Period.—*The brief interval of time elapsing between the sharp closure of the mitral and tricuspid valves at the onset of systole and the further rise in ventricular pressure, necessary to open the aortic and pulmonary valves against the arterial pressure is called the* **presphygmic period** *and varies from* 0.07 *to* 0.09 *seconds.*

Mental picture.

Practical Application and Importance of a Mental Image.—For the purposes of the auscultator, systole is assumed to be initiated by the first sound and diastole by the second, and in the mind's eye he sees the cycle of events.

Systolic Events.—*Thus when hearing* **the first sound,** *he sees the ventricles contracting, the mitral and tricuspid valves closed, the aortic and pulmonary valves freely open, the blood surging into the pulmonary artery and aorta, and the auricles refilling from the venous trunks* (see Figs. 175 and 177).

Diastolic Events.—*With the second sound he sees an exact reversal of conditions: the aortic and pulmonary valves tightly closed; the mitral and tricuspid freely open and the blood rushing through them from the auricles above to the ventricles below* (see Figs. 176 and 177).

IMPORTANT DEDUCTIONS.—*It is obvious that any organic valvular murmur that is coincident with, immediately follows, replaces, or itself modifies, the normal first sound* (*systolic murmur*), must arise either from leakage in the valves which should be absolutely closed and water-tight in this phase (*mitral and tricuspid*) or from an obstruction in those that should be freely open (*aortic and pulmonary*).

Inevitable conclusion.

Hence any organic valvular systolic murmur must be due either to mitral or tricuspid regurgitation or to aortic or pulmonary obstruction (stenosis).

Systolic murmurs.

In diastole the opposite conditions prevail; the mitral and tricuspid valves being open and the aortic and pulmonary valves closed.

It is evident that any murmur coincident with, immediately following, modifying, or replacing the second sound (diastolic murmur) is due to aortic or pulmonary leakage or to mitral or tricuspid obstruction.

Diastolic murmurs.

Nine out of ten murmurs have their origin in the left heart—the hard-working side.

Rule of incidence.

Presystolic Murmurs in Mitral or Tricuspid Stenosis.—*If the auricles contract as they normally should in presystole this final contraction will increase abruptly the force and rapidity of the flow from auricle to ventricle just before the closure of the auriculo-ventricular valves (first sound) terminates diastole. The diastolic murmur of mitral or tricuspid obstruction will assume therefore a presystolic crescendo intensification. If the auricle is incapable of its normal contraction, the murmur is diastolic purely, lacking all presystolic accentuation or being actually diastolic-diminuendo because the highest speed of the blood stream is attained under the initial intra-auricular pressure, accumulating during systole and highest at the instant the second sound announces the close of the ventricular outlets and the opening of the auriculo-ventricular gateways.*

Auricular systole.

Crescendo murmur.

Diminuendo murmur.

In *typical* cases the "rumbling" or "thrilling" *crescendo* murmur seems to run against and be abruptly terminated by an intensified slamming first sound.

AGAINST THE STREAM.—*It is at once evident* that if at any point there is created a serious impediment to the free onward flow of the blood, an increased strain is thereby thrown upon the cardiac mechanism and a tendency to passive congestion or stasis in the area whose drainage is thereby impaired is at once established (see Fig. 173).

Logical sequence.

Therefore, if any damming of the flow of pathologic degree is present or any portion of the pumping machinery is defective, the ultimate bad effects will appear *chiefly and first* in those portions of the heart next nearer the

venous sources of the blood than is the lesion itself. *In short, if one may use an old expression, in the main the bad effects of a cardiac lesion work backward against the blood stream.*

An extracardial obstruction.

ILLUSTRATION.—*Interstitial nephritis raises systemic arterial blood pressure to an extraordinary degree and the left ventricle responds to the challenge of the narrowed arteries by drawing upon its reserve and increasing the strength of its individual contractions.*

Accentuated aortic 2d.

The blood is then forced so vigorously into the aorta as to make its elastic recoil and the closure of the aortic valve unusually violent. *As a result there is an "accentuation of the aortic second sound."*

Heart response.

Hypertrophy.—*Like every other muscle under sustained unusual exercise and overload, the ventricle tends to hypertrophy and to increase its strength; the wear and tear of the aortic valve and the aorta itself is also increased and an unusual and excessive pressure is exerted upon the mitral valve, which alone stands between the overacting ventricle and the weak-muscled left auricle.*

Relative insufficiency.

Secondary Mitral Leakage.—If then the mitral valve becomes the seat of acute inflammation or of sclerosis, if the papillary musculature loses its strength and can no longer stay the leaflets (papillary insufficiency), or, if the heart itself dilates so widely through high pressure, toxemia, and degenerative changes, and muscle tonus becomes diminished so greatly that the mitral ring which supports the valve yields (relative insufficiency), in any or all of these events *mitral leakage* occurs.

Extremely common.

Auricular dilatation.

Consecutive Involvement of the Right Heart.—*A backflow into the left auricle then is established; that chamber is dilated and embarrassed, and the incoming current of blood from the lungs is obstructed. The right ventricle is immediately called upon for increased action in order to relieve the passive congestion of the lungs by an extra drive. The blood is then forced so strongly into the pulmonary artery as to intensify the shock of its valvular closure and produce an "accentuation of the pulmonary second sound."*

Right ventricular response.

Accentuated pulmonary 2d sound.

Pulmonary stasis.

The lungs may thus be placed between two fires through the continued back *pressure* of blood associated with a weak left ventricle, a leaky mitral orifice and weak or fibrillating left auricle in front, and increased pressure from the overacting right ventricle behind. This effect is greatly intensified if the left heart continues to weaken.

Tricuspid leakage.

A persistence of these conditions would tend to cause ultimately a dilatation of the right ventricle with or without a temporary or permanent leakage through the tricuspid valve and a transfer of part of the overload to the great systemic veins. The effects of stasis might be manifested then in the general venous circulation by congestion of the viscera and ultimate edema or general anasarca.

Systemic stasis.

Such a complete cycle of pathologic events is common enough in practice though by no means invariable, and usually covers many years in its full course and develops but a part of the complete cycle.

Realizing what marked local symptoms may arise from even a slight chronic congestion of the brain, stomach, liver, intestines, kidneys and lungs, one can readily understand how easily in such cases misinterpretation may occur because of the *misleading localization of the symptoms* and how readily

Symptoms often misinterpreted.

the true primary and basic cause namely, *cardiovascular insufficiency*, may be overlooked.*

The Causative Factors.—Practically all true valvular murmurs are due either to stenosis or to abnormal patency of one or more of the valvular openings and these conditions result from inflammation of the valves (endocarditis), chronic sclerosis (arteriosclerosis), from associated secondary or primary myocardial degeneration, actual myocarditis, or from mere toxic or congenital myocardial asthenia (atonicity) which results in insufficiency of the papillary muscle or stretching of the valvular ring (relative insufficiency). The causative conditions are fully described elsewhere, and a study of these factors makes clear the etiology of the actual lesions.

Endocarditis and sclerosis.

Myocardial defects.

Relative Frequency.—Mitral regurgitation is by far the most common; mitral stenosis, aortic regurgitation, aortic stenosis and tricuspid regurgitation follow in order; the remaining lesions being rare.

As to combined lesions, double mitral and double aortic are most common, some placing the latter first in frequency, which is against the author's personal experience, and would vary greatly in different clinics according to the predominant sex and age of the patients.

Double lesions.

THE DIFFERENTIAL POINTS.—*To differentiate heart murmurs the following points are determined primarily:* (*a*) **Time or rhythm.** (*b*) **Point of maximum clearness and intensity.** (*c*) **The direction and extent of transmission.** (*d*) **The quality of the abnormal sound.** (*e*) **The associated signs,** *viz., arterial and venous pulsation, the radial pulse, increased area of cardiac dulness, accentuation of heart sounds, cyanosis, edema, etc.*

RHYTHM.—Organic murmurs occurring with the first sound. (*Period of ventricular contraction*): (*a*) *mitral regurgitation;* (*b*) *tricuspid regurgitation;* (*c*) *aortic stenosis;* (*d*) *pulmonary stenosis* (*rare*).

Systolic murmurs.

Systolic murmurs, nine times in ten, are due either to mitral regurgitation or aortic stenosis or are purely accidental or hemic.

Murmurs Heard with the Second Sound.—(*Period of ventricular relaxation*): (*a*) *aortic regurgitation;* (*b*) *pulmonary regurgitation* (*rare*); (*c*) *mitral stenosis;* (*d*) *tricuspid stenosis* (*rare*). The mitral and tricuspid stenosis murmurs may be purely diastolic if the auricles are fibrillating or impotent; presystolic, or diastolic with presystolic intensification, if the auricles are capable of contraction just before the first sound occurs.

Diastolic murmurs.

Time variable.

Pure diastolic murmurs maximal at the base *are almost invariably due to aortic regurgitation.*

Pulmonary regurgitation rare.

A presystolic murmur *is almost invariably due to mitral stenosis and is maximal at, or, oftener, just within, the apex-beat.*

To Determine the Rhythm or Time.—This should be positively determined by taking the carotid pulse while listening to the murmur. *The radial pulse should not be used* for this purpose, and in dilatation of the right heart, the *apex-beat* is far less constantly reliable and clean-cut than the *carotid beat* which can easily be felt at the anterior border of the sterno-

Important rule.

Use carotid for timing sounds.

* The patient with a nephritis of the extremely chronic type is likely to be snuffed out by an apoplexy or uremic seizure before he can complete this disastrous cardiac cycle.

mastoid muscle when the head is turned to the side corresponding to the artery palpated.

Hypertrophy and Dilatation.—Under their appropriate headings the variation in cardiac outline in the chief pathologic conditions and their influence upon the heart sounds will be fully shown and discussed.

"Simple hypertrophy" is usually associated with chronic interstitial nephritis, aortic stenosis or arteriosclerosis, and "simple dilatation" represents a stage of adaptation or actual incompensation in such lesions, or, in some form of myocarditis, myocardial degeneration or congenital asthenia.

As a matter of fact the occurrence of hypertrophy without some degree of dilatation is doubted at present by the greater number of experienced pathologists and the so-called "simple dilatation" (lacking hypertrophy) cannot exist *in any decided degree* without producing decided symptoms subjective or objective together with a limitation of the field of cardiac response.

CERTAIN FACTORS BASIC IN THE CONSIDERATION OF THE DISORDERS OF THE HEART AND BLOOD VESSELS

Meets vital needs.

What the Heart Must Do.—*In conjunction with the vascular mechanism it supplies the initial propulsive impulses and the sustained force necessary to maintain that uninterrupted flow of blood throughout the systemic and pulmonary vascular circuits, which is indispensable to the physical and chemical exchanges essential to health and indeed to life itself.*

Arterial currents.

The Transformer.—In the aorta and pulmonary artery alike, both pressure and rapidity of flow are excessive and rhythmically interrupted, but the shock of the expelled blood column is taken up by the elastic arterial walls.

Capillaries and veins.

In the capillaries, the flow is sufficiently slow and uninterrupted to permit the vital tissue exchanges of waste and repair, and a slowed current and lowered pressure are present with slight modification in the veins.

Arterial pressure.

Arteries Auxiliary Hearts.—*The sustained arterial pressure necessary to produce a continuous current is dependent upon an adequate initial impulse, a sufficient blood volume, efficient vasomotor control, and normal elasticity of the vessel walls.*

Pathways of least resistance.

The great arteries at first yield to the shock of systolic outflow but in their recovery exert a sustaining pressure upon the blood column which, being shut out of the heart during diastole by closed semilunar valves, must of necessity move forward along the vascular pathways of least resistance.

Sustained flow.

Arterial Conservation of Initial Energy.—Obviously the driving force of ventricular contraction is largely conserved and compensated by this primary arterial distention of the aorta and pulmonary artery which serves to *maintain* the flow during the entire period of ventricular relaxation. These great vessels constitute a storage plant, rhythmically charged and discharged.

Necessity of vasomotor control.

The Rôle of the Vasomotor System.—*It is evident also, not only that the capillary flow must be continuous, but that it must be controlled to a degree that will secure the automatic satisfaction of the periodic or even momentary variations in the needs of the body tissues.*

Hence quite aside from reflex increase of heart strength, acceleration of primary outflow and rise in pressure, tissue needs are met by a slowing or quickening of the peripheral stream and a coincident local increase or decrease in intravascular pressure through automatically regulated dilatation or contraction of the capillary channels. Rate of flow.

Speed of the Blood Stream.—In the great arteries the normal flow varies between 200 and 400 mm. per second as contrasted with a capillary flow of but 0.6 to 0.8 mm. per second. In the normal individual the body circuit is completed in about fifty-five seconds requiring about 55 heart beats. In strenous physical effort the time required is but four and one-half seconds and in marked anemia, with lowered specific gravity and decreased blood viscosity, the circuit consumes but thirteen and one-half seconds, the required beats being reduced to 23 (C. Hirsch). In erythremia, on the other hand, increased erythrocytes and a heightened viscosity are associated oftentimes with a doubling of the total blood content of the body, heightened arterial pressure, cardiac overaction, and vasomotor constriction, and the resultant slowing of the circulation becomes an important factor. Great arteries. Capillaries. Physical exertion. Complete circuit. Anemia. Erythremia.

Extreme Vasodilatation.—This may obtain in disease to a degree which actually threatens the life of the individual, such indeed as would be the dilatation if the medullary center were paralyzed, as in certain cases of sudden death from diphtheria, or if the splanchnic nerves were divided. A cause of death.

Excessive Vasoconstriction.—Conversely, vasoconstriction may become so extreme through direct overstimulation of the vasomotor centers in the medulla or the action of circulating toxins (uremia), as not only to raise arterial pressure to a dangerous degree, but also, when exerted locally, to cause actual gangrene of the part affected, as in Raynaud's disease, in which there is extreme vasomotor constriction alternating with excessive local vasomotor dilatation. Uremia. "Raynaud's disease."

Conflict of Opposing Forces.—In certain ailments, as in *aortic regurgitation*, there is a compensatory effort on the part of the body tissues to neutralize the effect of the unsupported blood column (at the aortic outlet) and modify the necessarily excessive strength of the primary flow by means of a persistent mild vasodilatation. Compensatory vasodilatation.

In a case of interstitial nephritis the resulting tendency to adverse toxemic vasoconstriction is further aggravated by a coincident impetus given to arteriosclerotic changes. This not only weakens the heart, but, by impeding conservative vasodilator action, produces a decided impairment of body nutrition. The vasomotor center itself not only may become markedly irritable or weakened through the toxins of disease or the imperfect blood supply it receives in certain cases of incompensation, but is extremely susceptible also to adverse psychic influences.* Superimposed nephritis.

The Heart's Capacity for Work.—*The miraculous perfection of this complete, wholly automatic, hydraulic plant is shown by the fact that each day's work of the normal heart is equal to* 20,000 *kilogrammeters* (*Zuntz*), *viz., the power* Daily task.

* The Dastre-Morat Law.—Dilatation of the splanchnic vessels is accompanied usually by contraction of the surface vessels.

Life's work.

sufficient to raise 20,000 kilos of water to the height of 1 meter. In a lifetime of sixty years it pumps 2,800,000,000 liters of blood (Plesch).

Cardiac reserve.

Varied Demands.—The output of the heart must be such as will satisfy the extremes represented by absolute rest on the one hand and the most violent exertion on the other, and this means that the normal heart must be able to meet instantly and adequately at least a thirteenfold increased demand.

Impaired reserve.

One of the earliest, most important, and least recognized symptoms of insufficiency is an inability on the part of the individual to meet hitherto well-borne variations in the demand upon his heart strength without conscious effort or actual discomfort. The daily work hitherto done easily becomes a burden.

Nutrition impaired.

Metabolism in Insufficiency.—It is evident that any decided interference with efficient circulation must affect the supplies of nutrient material and oxygen which in health are steadily and continuously carried by the blood to the chemically active tissues, no less than the proper combustion and complete disposal of the waste products of body metabolism which are removed through various channels in like manner and by the same mechanism.

Acceptance of Myogenic Theory.—*No attempt will be made to set forth in detail the claims of the neurogenic and myogenic theories relating to the contraction of the heart. The latter, for the most part, stands upon a firm experimental basis, so admirably serves diagnosis and therapy alike as to have immeasurably advanced the proper clinical conception and management of cardiac derangements, and is accepted by most physiologists.*

WHAT THE NORMAL HEART MUST POSSESS

MYOGENIC THEORY.—The heart cells must possess: (*a*) The power to generate stimuli to contraction, *i.e.*, "stimulus production."

(*b*) The ability to receive stimuli, *i.e.*, "excitability."

(*c*) The power to respond to stimuli by contraction, *i.e.*, "contractility."

(*d*) Conductivity.

(*e*) The power to maintain continuously a lesser but vastly important tonic contraction or muscle tonus apart from periods of active contraction, *i.e.*, tonicity.

Stimulus Production.—*The myogenic theory also assumes that the highly specialized heart muscle cells when normal, properly nourished, and free from disturbing extracardial influences, can elaborate and maintain rhythmic chemical exchanges which stimulate the muscle fibers to a constant maintenance of proper tonus and efficient rhythmic contraction (rhythmicity).*

Dependent upon chemical processes.

"All or nothing."

Explosions of Energy.—*Irritability remaining constant, temporary exhaustion attending each systole is complete, but is rapidly repaired during diastole, and, in health, stimulus production and irritability constantly readjust themselves to adequate recurrent rhythmic discharge.*

Refractory Stage.—By reason of its rhythmically recurring exhaustion, the heart muscle is wholly unresponsive to stimulation during the period of its actual contraction, *i.e.*, it loses its *excitability (refractory stage)*. The prompt restoration of excitability and contractility, as seen in health, depends upon

a proper maintenance of the balanced chemical exchanges necessary to the renewal of the cell activity.

Rhythmicity.—*The rhythmicity of those explosions of energy which give rise to the normal heart beat depends upon the generation of periodic stimuli apparently determined by the rhythmic chemical interaction of inorganic salts of potassium, calcium and sodium and their ion-proteid combinations present in the blood in certain definite proportions (balanced solution).* Chemical exchanges.

In such combinations the substitution of one ion for another can change the physical properties of the "ion-proteid" compound and it is assumed that the quality of recurring irritability (recharge of the exhausted cell) indispensable to contraction is thus explained.

The theory of Gaskell which assumes a rhythmic alternation of assimilation and dissimilation (anabolic and katabolic processes) would seem well suited to this assumption.

Restoring the Heart Beat Post-mortem.—By proper perfusion with Ringer's solution,* H. E. Hering restored and maintained for three and one-quarter hours the heart beat of a man eleven hours dead and Kuliabko obtained contraction of the heart of a dog five days dead. Modern miracles.

Adequate rest periods must be had between the recurrent discharges or neither stimulus production nor contractility of normal degree can be retained.

Therefore, the work of the normal heart is dependent upon a series of rhythmic explosions and these in turn are contingent upon the maintenance of an efficient balanced metabolism.

From the foregoing it is clear that the possession of a certain degree of anatomic integrity of the heart muscle is a paramount necessity and that the matters of nutrition and proper conduction are alike vitally important. Fiber conduction.

Efficient Contraction.—*A point or points of maximum irritability, free conductivity and definite open channels of conduction are essential to efficient and orderly contraction.*

As Hirsch well says, "the heart muscle is a homogeneous plasma mass with nuclei," a veritable aggregation of specialized cells. Its fibers freely anastomosing, are naked, wholly lack the connective tissue sheath of the skeletal muscle, *and conduct almost as freely as nerve fibers.* Thus every facility exists for rapid and harmonious stimulus transmission and coördinated response. Heart fibers naked. Free interconduction.

It has been clearly, and apparently conclusively, shown by Wm. His, Jr., that the embryonic heart can act by virtue of its own intrinsic powers of conduction before its ganglion cells are demonstrable. This does not prove that a function possessed in embryonic tissue may remain dominant after the nervous mechanism develops but is a strong link in a long chain of evidence. Automatism.

Gaskell's Bridge.—(*Bundle of His*).—The apparent barrier to the full acceptance of the theory of the heart's potential automatism was represented by the anatomic break evident in the separation of the auricles from the

* Ringer's solution is an artificial blood serum containing *sodium, calcium and potassium* chloride and sodium bicarbonate.

ventricles by the auriculo-ventricular septum, a non-muscular structure, but this gap has been bridged by Gaskell of Cambridge and Wm. His, Jr., who have shown that the interruption of normal stimuli passing from auricle to ventricle is not accomplished by cutting the nerve bundles, but rather the "bundle of His."

Gaskell foreshadowed and foretold,* and Stanley Kent and Wm. His, Jr., proved, for the human heart, that the apparent barrier between auricle and ventricle was bridged by a bundle of peculiarly modified muscle fibers ("auriculo-" or "atrio-ventricular bundle").

Purkinje fibers.

Auriculo-ventricular Bundle.—Tawara described clearly the course of these fibers, whose point of origin forms the so-called *auriculo-ventricular node* of Aschoff and Tawara at the posterior right border of the base of the auricular septum, and showed that the bundle represented an aggregation of the Purkinje fibers which ramify to every part of the ventricles.

Heart Block.—Any disease process or the administration of any drug tending to obstruct the free passage of impulses through this bundle must at least increase the time interval normally existing between auricular and ventricular contraction (*conduction period*) as expressed clinically by that separating the jugular wave (*a*) from the carotid wave (*c*) of the polygram. If the block interposed is still more decided, certain auricular contractions will fail to carry through and both delays (*prolonged a-c interval*) and elisions of the "*c*" wave ("dropped" ventricular contractions) will appear on the record, and show a rhythmic ventricular disregard and pretermission of fixed groups of auricular beats. The auricle may beat two, three, four or more times to each beat of the ventricle.

Increased "a-c interval."

Partial block.

Complete Block.—This may reach a point where entire *dissociation* of auricle and ventricle is present and the two chambers beat quite independently, each of the other, the latter developing a slow deliberate rhythm of about 30 beats to the minute, while the auricle pursues its own rate which may be two, three, or four times that of the ventricle.

Ventricular rhythm.

It is in such cases of heart block that periods may occur when the over-deliberate, and oftentimes weakened ventricle fails to send sufficient blood to the brain, thus causing convulsive seizures. It may cease for short periods, moreover, to beat at all, in which case syncopal attacks may occur and thus round out the classical "*Adams-Stokes syndrome.*"

Adams-Stokes syndrome.

Sino-auricular node.

The Pace Maker of the Heart.—*Whence come the rhythmic impulses which induce coördinate contraction of the auricles and ventricles?*

There is a normal point of *maximum irritability* represented by a network of specialized muscle cells richly supplied with nerves and imbedded in the upper anterior end of the *sulcus terminalis*, which sulcus runs from the inferior vena cava to the junction of the superior vena cava with the right auricular appendix.

Position.

* W. H. Gaskell of Cambridge University, England, actually demonstrated both myogenic conduction and heart block in the frog and tortoise in the year 1883.

This is the sino-auricular node of Keith and Flack which, from its station in the wall of the right auricle at the mouth of the superior vena cava, normally initiates each contraction of the heart.

Sinus node.

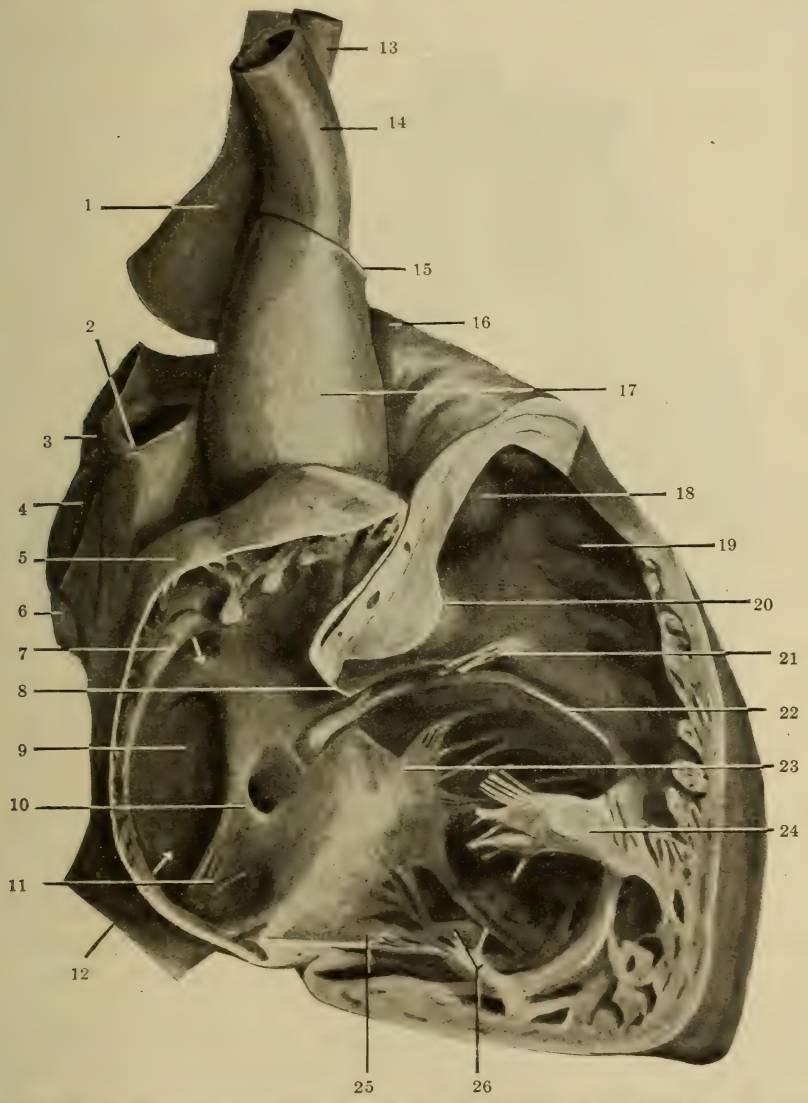


FIG. 178.—**"Gaskell's Bridge,"** "*His' Bundle.*" Interior of the right atrium and ventricle. The atrio-ventricular bundle dissected out. (*Morris-Jackson.*)

1, Aortic arch. 2, Vena cava superior. 3, Right pulmonary artery. 4, Right superior pulmonary vein. 5, Right atrium. 6, Right inferior pulmonary vein. 7, Crista terminalis. 8, Cut anterior tricuspid cusp. 9, Fossa ovalis. 10, Valvula sinus coronarii. 11, Valvula venæ cavæ. 12, Vena cava inferior. 13, Left common carotid artery. 14, Innominate artery. 15, Reflexion of pericardium. 16, Pulmonary artery. 17, Ascending aorta. 18, Left pulmonary valve. 19, Conus arteriosus. 20, Crista supraventricularis. 21, Papillary muscle of conus. 22, Atrioventricular (His) bundle. 23, Medial tricuspid cusp, partially removed. 24, Anterior papillary muscle. 25, Part of posterior mitral cusp. 26, Posterior papillary muscle.

Course of impulses.

Thence the wave of impulse spreads over the auricular walls, causing auricular contraction, then crosses the bridge of Gaskell (bundle of **His, Jr.**) to the papillary muscles and walls of the ventricles, which begin to contract from 0.12–0.18 of a second later (normal a-c interval of the venous tracing).

Normal conduction time.

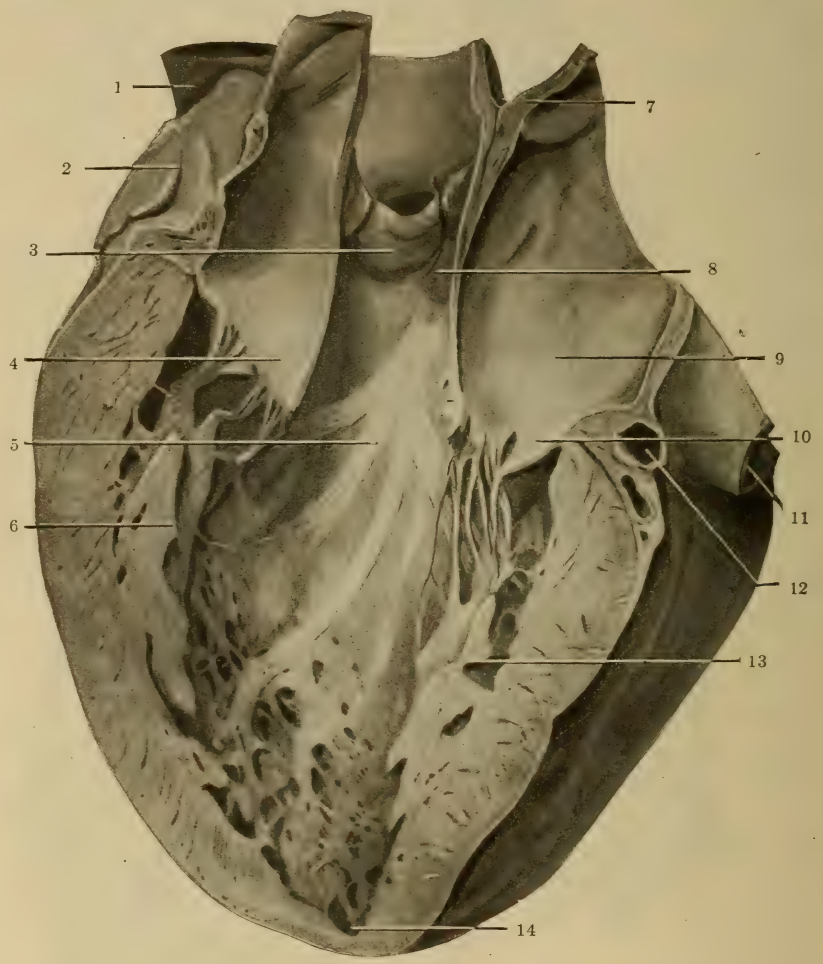


FIG. 179.—**"Gaskell's Bridge,"** *"His' Bundle."* Left ventricle and part of the atrium. (The aorta is opened through the medial cusp of the mitral valve. The plainly visible left limb of the atrio-ventricular bundle has been accentuated.) (*Morris-Jackson.*)
1, Pulmonary artery. 2, Right auricle. 3, Right aortic valve. 4, Anterior mitral cusp. 5, Atrioventricular bundle. 6, Anterior papillary muscle. 7, Cut wall of left atrium. 8, Membranous ventricular septum. 9, Left atrium. 10, Part of posterior mitral cusp. 11, Vena cava inferior. 12, Coronary sinus. 13, Posterior papillary muscle. 14, Fine meshwork of *columnæ* carnæ at apex.

Right vs. Left Auricle.—As might be expected from the position of the pace maker, the beginning of right auricular contraction actually precedes that of its fellow, the left auricle, by 0.01 to 0.03 second but clinically their contraction may be considered as simultaneous.

Extrasystoles.—In certain pathologic conditions the heart may develop abnormally placed areas of irritability which initiate imperfect beats disturbing normal rhythm though usually leaving the rate unaffected. Premature contractions.

The Strength of Extrasystoles.—Given equal stimuli, the *strength* of any extrasystole is mainly dependent upon the time elapsing since the initial contraction of the beat preceding it, *i.e.*, upon the duration of the antecedent rest period.

Stimulus Conduction.—As stated previously, conduction by the muscle fibers themselves, while a trifle less perfect than that of systemic nerves, is remarkably good. It is greater in the contractile fibers of the auricle and ventricle than in the auriculo-ventricular bundle and, as in the case of other functions of heart muscle, is momentarily exhausted by each systolic contraction.

Tonus.—*As in the case of all other muscles a certain degree of muscle tonus is normally well maintained and during life complete relaxation never occurs in the normal myocardium.*

The Neurogenic Theory.—This hypothesis assumes that the intrinsic stimulus to contraction originates in the ganglion cells adjacent to the sinus node or in those of the interauricular septum. Thence it is supposed to be transmitted by axones to the "*sinus node*" (of Keith and Flack), the auriculo-ventricular node ("node of Tawara"), the "bundle of His" (His-Tawara system), or directly to the muscles. Auriculo-ventricular node.

Independence of the Heart.—It is obviously possible that the two systems may prove to be interdependent or simultaneously active, but it is certain that the heart muscle itself is perfectly capable of automatic rhythmic activity wholly apart from the nervous system.

The Influence of the Vagus and Sympathetic Cardiac Nerves.—The vagus and the sympathetic nerve fibers are physiologic antagonists with relation to heart action.

Normally tonic vagus influence exists and manifests itself chiefly in the maintenance of a normal rate. When abnormal vagus excitation occurs, the rate of heart beat is slowed, and the interval between the auricular and ventricular contractions ("conduction time, a-c interval") is increased. Certain auricular contractions may fail to excite the ventricular contraction of the heart or either or both auricle and ventricle may stop temporarily. These constitute examples of partial or complete vagus heart block (delayed and interrupted conduction in the His bundle), such as may be seen in digitalis poisoning or in the toxemias of infections.* Physiologic antagonists. Vagus block.

It would seem that the direct action of the vagus is exerted upon the sinus

* Whether the older conception of "conduction time," which assumed that impulses to ventricular contraction were passed through the auricular tissues, was correct may be doubted. It would appear that strictly speaking the "a-v interval" represents the fraction of time intervening between an auricular and a ventricular response to individual and separate stimuli from the sinus node. The ventricular impulse passes through the auricular-ventricular node, the His bundle, and the two branches of the His-Tawara system. The distinction is of slight importance in this elementary discussion.

node (node of Keith and Flack) and that it is left vagus stimulation which chiefly affects the conduction time.

Action of sympathetic.

Conversely, stimulation of the sympathetic tends to increase the rate of the heart beat, shorten the conduction time and increase contractility and irritability. In health, these accelerator nerves constantly exert a tonic influence opposed to that of the vagus.

Common Causes of Vagus Overstimulation.—Aside from poisoning by digitalis, strophanthus, aconite, etc., vagus overaction may occur in varying degree in dyspnea and cyanosis (lack of oxygen and excess of CO_2), in tobacco poisoning, in cerebral compression, and in arterial hypertension.

Effect of atropin.

On the other hand, vagus influence is diminished or suspended under full doses of atropin and this fact is utilized in the differentiation of the slow pulse of vagus overstimulation from that due to organic disease.

Vagus overeffects may be easily obtained by deep thumb pressure over the right carotid at the middle or lower third of the neck, but the maneuver is not wholly safe.

To what an extent the diseased heart can escape the normally balanced influences of the accelerator and cardio-inhibitory nerves and run *amuck* through sheer disorganization of its own automatism and intrinsic rhythmicity, is shown later in our discussion of the "arrhythmias" associated with organic disease.

Reinforcing spirals.

Remarkable Anatomic Structure of Heart.—The arrangement of the muscle fibers of the heart is peculiarly adapted to coördination in that the ventricular musculature constitutes two spirals (Mall) each consisting of two layers running at right angles to each other. One of these spirals passes from the tricuspid area to the apex of the right ventricle, the other from the aortic and mitral orifices to the apex of the left ventricle, each being connected to the other by twisted strands running from the papillary muscles of one ventricle to those of the other.

Marvelous regulation.

Coördination of Function.—This is indispensable to the normal heart action and in addition to a marvelous correlation of functions we find a certain automatically regulated sequence of stimulation and contraction which permits selective muscular activity on the part of different portions of the heart. Thus a given stimulus being received, the sequence and degree of the resulting contraction is exactly that necessary to the economic and efficient contributive action of each part of the heart.

An understanding of this beautiful mechanism enables us to comprehend more readily the newer nomenclature of heart disorders, and the mechanics and modern interpretation both of disturbances of rhythm and of actual cardiac insufficiency.

Cardiac response.

Possibilities of Cardiac Reserve.—*Extraordinary possibilities of emergency response to effort are possessed by all normal hearts and affect both rate and force of contraction.*

Cardiac endurance.

Much cardiovascular overwork may be borne for long periods without producing symptoms, and actual insufficiency can be present for years without obtrusive manifestations.

Determining Factors in Cardiac Insufficiency.—*It is the degree of impairment or exhaustion of the reserve force, the sudden or gradual narrowing of the field of response, which determines cardiac insufficiency, one or all of the heart functions being affected.* Vital point.

Therapeutic Objective.—*Our object in treatment is to amplify, restore, or rehabilitate impaired reserve, and the main object of diagnosis is to detect early, and, so far as possible, measure the degree of, its impairment.* To maintain reserve.

BLOOD PRESSURE

Factors Basic in its Determination and Interpretation

ADAPTIVE CARDIOVASCULAR AUTOMATISM.—In order that a state of perfect body health may be maintained without undue and ultimately damaging strain upon the heart and blood vessels, it is necessary that the heart, that automatic generator of energy in the cardiovascular power plant, be itself measurably perfect as regards its controlling mechanism, and possessed of the attributes necessary to the generation of adequate horsepower and its economic delivery to the storage plant represented by the aorta and its main arterial service lines. Fundamental requisites.

Transformation and Conservation.—It is also requisite that the turbulent intermittent outrush of blood from the ventricles into the aorta should be so controlled automatically as to replace the primary jerky intermittency and rhythmic variations in the velocity of the blood stream by conditions of relative uniformity and constancy of flow, and such gradual reduction of speed, as best suits the needs of the tissues fed and drained by the capillary deltas.

Selective Adaptation and Distribution.—There must also be a selective adaptation of the *local* rate of flow and actual blood supply to the ever-varying demands of body metabolism. Obviously there is equal need of a relatively constant capillary blood pressure, varying only as may be necessary to meet the demands of tissue metabolism by means of a frictionless automatism.

A Marvelous Mechanism.—The anatomic structure of the arterial system is beautifully adapted to these purposes, and the exquisitely balanced and adjusted control exercised by the vasomotor system in health adjusts to a large degree the speed of the capillary current and the local supply of blood without any special demand upon the heart for decided changes in rate, rhythm, force or output.

Systolic and Diastolic Pressure in the Aorta.—Inasmuch as the systolic pressure and energy transmitted from the heart to the aorta far exceed primarily the amount needed to move the blood column onward, these must be husbanded in order that blood flow may be steadily maintained during the entire diastolic period. One per cent. is the amount of pressure energy *immediately* applied to support the forward movement of the blood column. Ninety-five per cent. is expended in distending the elastic arterial walls and thus becomes potential pressure-energy, the gradual release of which during

diastole serves to keep the column of blood moving steadily onward without permitting a descent of pressure below that representing the minimum ("*diastolic pressure*") of the preceding diastole.

Pulse Pressure.—Hence both systolic and diastolic pressure levels are maintained in the arteries, the former (systolic blood-pressure) representing the maximal level for the cycle, and becoming lowered as potential energy is converted into kinetic drive for the blood column. The difference between these two levels ("pulse pressure") of pressure energy represents under absolutely normal conditions the reinforcement, rhythmically dispatched to the arterial system by the heart through its recurring systolic contractions.

Regulation of Rate of Flow.—*Both initial current velocity and initial blood pressure*, as demonstrable in the larger arteries, would be disastrously excessive both in the lesser and least arteries and in the capillary spaces if the primary rate of flow was maintained throughout. Therefore, as we have seen, the rate of flow which in the large arteries varies from 200 to 400 mm. per second becomes reduced to 0.6 to 0.8 mm. in the untroubled waveless capillary lakes (C. Hirsch).

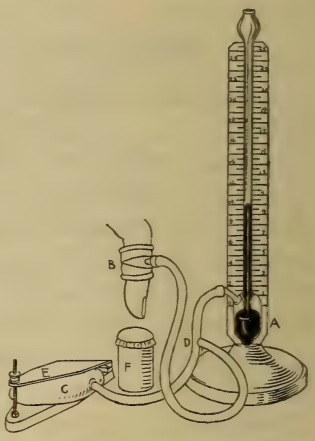


FIG. 180.—Gärtner's tonometer. One of the first blood-pressure instruments applied widely to *clinical* uses. It is used still occasionally for comparison of peripheral with brachial blood pressures.

Blood Pressure in the Small Arteries and in the Veins.—The systolic blood pressure, which in the aorta is about 150 and in the brachial may average 125 mm. of mercury, is reduced to the diastolic level of constant pressure (60 to 70) in the lesser arteries. *In the capillary deltas* it varies from 45 to 15 mm. of mercury and, in the veins, is reduced to a maximum of 20 in the superficial venous channels and 10 to 15 in the subpapillary venous plexuses (C. L. Wiggers).

Adjust to tissue needs.

DETERMINATION OF BLOOD PRESSURE.—Preliminary Considerations.—*It is evident that in clinical work we are chiefly concerned with the circumferential pressure exerted by the blood column upon the walls of the brachial artery during systole and at the end of diastole, together with the difference between these two which is "pulse pressure."*

Fundamental Factors.—*Furthermore, that variations in arterial pressure are dependent chiefly upon the volume and energy of ventricular systolic discharge, its rate, and the resistance offered by the lesser arteries and capillary field.*

It must not be considered the measure of the heart strength, which is but one factor.

Importance of Pulse-pressure.—In health the average "pulse pressure" varies between 35 and 50 mm. and this and the diastolic pressure are actually far more important in a diagnostic sense than the systolic pressure.

A diastolic pressure reaching or exceeding 100 mm. Hg. is almost invariably pathologic.

It should represent normally about 70 per cent. of the systolic reading.

Simple Device.—Fortunately for the clinician, blood pressure may now be measured quickly and with sufficient exactitude by simple and relatively inexpensive instruments nearly all of which depend upon the same general principles, viz., the translation of the pressure required to obliterate the arterial pulse, into the height of a mercury column in a manometer tube.

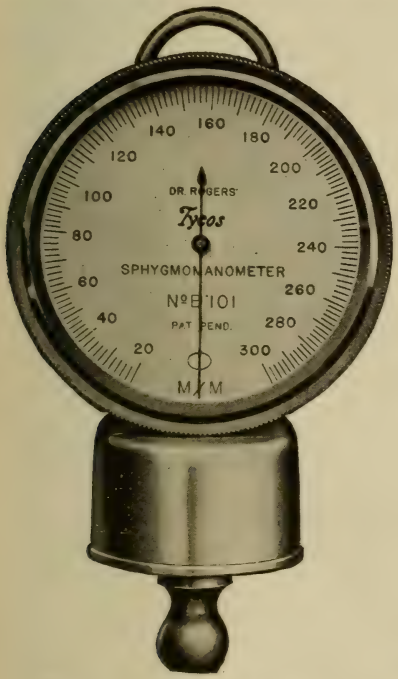


FIG. 181.—One of the several forms of pocket sphygmomanometers of the aneroid type. Convenient, compact and portable, but needing standardization from time to time.

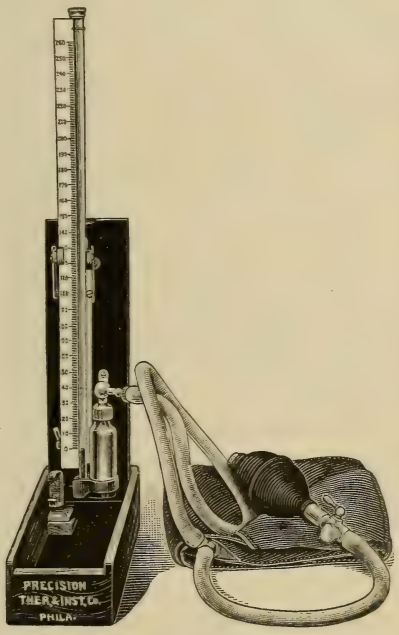


FIG. 182.—New Nicholson Princo sphygmomanometer. Small, portable and accurate. (*Courtesy of Precision Thermometer and Instrument Co., Phila.*)

The portable instruments now available are, for the most part, sufficiently accurate for ordinary clinical work, but systolic blood pressure readings which depend upon the usual *and least accurate* method of determination, *i.e.*, the first palpable return of a radial pulsation after obliterative compression, involve an underestimate of from 5 to 15 mm. of mercury.

Systolic, diastolic and "pulse" pressures.

The Older Technic of Blood-pressure Determinations.—The hollow armlet, applied midway between shoulder and elbow, is inflated by the hand bulb until the radial pulse is lost, then by the outlet thumb-screw the pressure is lowered until the pulse return is just perceptible. As the pressure is equal in all parts of the closed system, the height of the mercury column in the manometer tube is an exact index, and the reading represents the "*maximum*" or "*systolic*" pressure.

"Diastolic" or "minimum" pressure is determined by noting for ten to twelve pulsations the increasing amplitude of the pulse wave registered by the mercury column as the pressure is reduced in 5-mm. series. The point representing the base line of the maximum excursion is the **index of diastolic pressure.** Below that is a limited pressure area of equal amplitudes.

The "mean" pressure represents the average of systolic and diastolic readings and the **"pulse pressure"** or *"pulse amplitude"* represents, as has been stated, the difference between the systolic and diastolic readings.

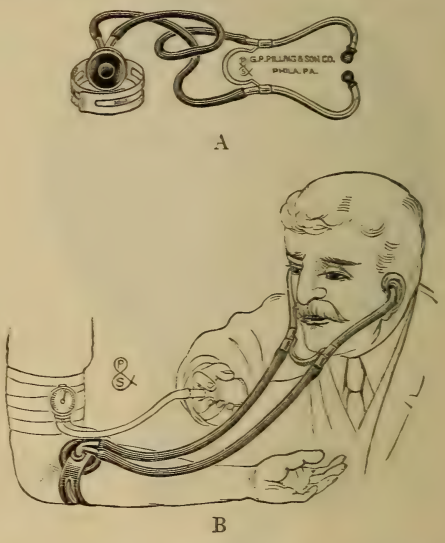
Pulse pressure variations.

Pulse pressure is usually about one-fourth the systolic pressure and runs between 35 *and* 50 *mm. Hg.*, as stated above. A pulse pressure which falls below 25 or exceeds 60 is almost certainly pathologic.

Diastolic readings run normally about 25 to 40 below the systolic; in low tension, varying from 50 to 80 mm.; and in aortic regurgitation 150 mm. or more. An excessively rapid, persistently irregular or unequal pulse makes *palpatory* diastolic pressure determination impossible.

Sources of error.

In every case the arm band should be closely adjusted, *the arm supported at the heart level* and the same position taken for a series of tests.



A

B

FIG. 183.—The illustration shows the Pilling bracelet-stethoscope, an ingenious, useful, yet dispensable device. It is less convenient but wholly possible to use the binaural stethoscope of any type alone. (See Fig. 184.)

Sclerosis.

The limit of error in calcareous arteries is but 10 or even 5 mm. and is negligible or easily estimated. The same figures respectively represent the difference between females and males and the standing and sitting posture.

Sex and attitude.

The limits of error in respect to the state of contraction or relaxation of the arterial wall itself are probably considerable and quite beyond accurate determination.

Muscular movement and the presence of edema alike introduce a variable element of misinterpretation.

An error of 20 mm. Hg. may result from the use of an improper rubber tube and old tubes should be replaced frequently by new ones.

Auscultation method.

Koratkow's Method.—This simple, clinically accurate, and *recommended* method consists merely in auscultating over the brachial artery below the point of constriction, the sounds produced during the obliteration of the brachial pulse *by the arm band of any of the ordinary instruments.* The use of a special arm band carrying a stethoscope is a matter of increased

convenience but is not a necessity. The sounds heard over the brachial artery as the obliterating pressure is gradually released are divisible in five phases of audibility, viz:

First Phase.—Systolic Pressure Level.—A clear thumping sound, which must be translated into the reading of the manometer scale at the *exact time of its occurrence* represents systolic pressure. This sound remains clear and distinct usually until the mercury column has dropped 15 mm. and represents the first projection of blood into an empty artery.

A clear sound.

FIG. 184.—The auscultation method. (*Norris.*)

Second Phase.—The clearness of tone of the first phase becomes a blending of multiple eddy murmurs and this blurred picture lasts until an additional recession of about 20 mm. of mercury has occurred.

Confused multiple bruits.

Third Phase.—A sharp sound appears and grows progressively more distinct and louder *during a recession of about 25 mm.* of the mercury in the manometer column.

Reappearance and intensification of clear tone.

Fourth Phase.—Diastolic Pressure Level.—This represents the abrupt muffling of the tone of the preceding (third) phase and lasts for only a short period represented by a recession of about 5 mm. of mercury.

Abrupt muffling of tone.

The scale reading of the manometer at the *exact time of the appearance of this dull muffled sound* is taken as representing the diastolic pressure.

Fifth Phase.—This is merely the period initiating entire loss of sounds and by Koratkow and others was considered formerly as representing the diastolic pressure point.

The use of the fourth phase is probably the more accurate and certainly the more generally applicable method inasmuch as complete disappearance of sounds may not occur in aortic regurgitation or in certain cases of exophthalmic goiter.

Normal Readings.—*The normal systolic pressure readings according to*

Janeway are: for young adults, 100 *to* 130; *older adults* 100 *to* 145; ***children*** 90 *to* 110; *infants under two years,* 75 *to* 90.*

Age.—The following table is that of L. Gordon, and represents the results of an extensive study of the relation of age in normal children to systolic blood-pressure.

Years	Mm. Hg.
Under one	71.0
One	73.0
Two	79.3
Three	81.0
Four	83.0
Five	86.5
Six	88.5
Seven	85.0
Eight	93.0
Nine	100.0
Ten	95.0
Eleven	104.0
Twelve	105.0

Effect of excitement.

Excitement may cause a rise of 40 *mm. or more and physical effort a* ***slight*** *increase or, if extreme or of an unusual nature and abrupt, a rise of* 30–60 ***mm.*** *may occur. Accustomed effort of the same grade may cause a rise of only* 10 *mm. or less.*

The rise of pressure under exercise and excitement alike is predominantly systolic, and, in the case of the former, pressure may actually fall if the individual is in good training and the effort is not one of an abrupt, violent, nature (high jump, throwing the hammer, 100 yard dash).

Experience teaches us that a persistent pressure of 130 or more in the young adult is a *suspicious* finding, and that even in middle age a systolic pressure of 150 is almost certainly pathologic.†

A great need.

We need greatly a large series of "normal" readings based upon the auscultatory method and upon cases thoroughly and completely investigated with respect to the presence of incipient disease and the structural types encountered. The variations in the figures at present available are too great to represent wholly normal departures.

Faulty figures.

The reported *average* systolic reading of 150 mm. at age sixty for example, to which about 10 mm. must be added to allow for the constant error inherent in the palpatory method, must have involved the inclusion of maximal readings so high as to raise serious doubts concerning the legitimacy of the figures. The mortality reports of life insurance companies show the fallacy inherent in the application of the word "normal" to any such figures.

Results in practice.

* The author feels that the lower figures *for adults* are to be accepted only with decided reservation inasmuch as he has usually found them only in association with more or less profound subnutrition or circulatory depression. Low readings are especially common in tuberculosis even during the incipient stage.

† The average normal for the *sixth* decade is 138 mm. Hg. *Maximum* obtained in large group 150 mm. Hg.

Important Statistical Data.—*J. W. Fisher has recently published a most valuable article reporting the average systolic pressures obtained as the result of* 19,339 *readings representing that number of accepted candidates for life insurance.*

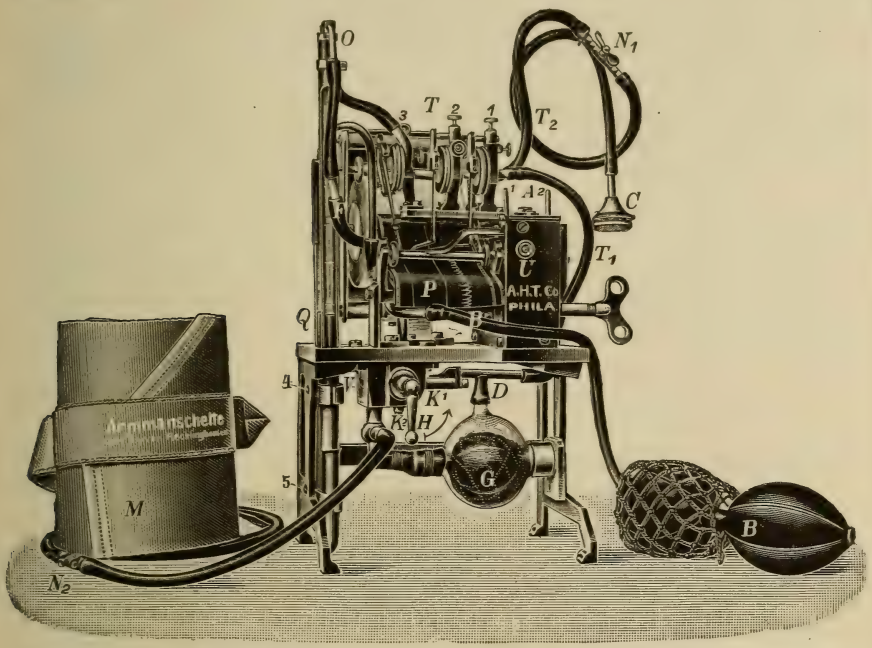


FIG. 185.—The Uskoff sphygmotonometer. This instrument simultaneously records blood pressure in millimeters of mercury, together with the brachial pulse at varying pressures, and one other tracing (jugular, carotid, apex beat, etc.). (*Courtesy, A. H. Thomas Co.*)

The following table shows that **even those who had attained the later years of the sixth decade of life** *showed an average systolic blood pressure slightly under* **135** *mm. of mercury.*

DR. J. W. FISHER'S TABLE

Ages	Number	Average blood pressure
15–20	281	119.85
21–25	785	122.76
26–30	791	123.65
31–35	689	123.74
36–40	2,111	126.96
41–45	6,740	128.56
46–50	4,471	130.57
51–55	2,371	132.13
56–60	1,100	134.78
Total.....	19,339	128.91

On 525 applicants accepted in earlier years who showed an average systolic pressure of 152.58 mm., the Northwestern Life Insurance Company

Excessive mortality.

suffered an excess mortality exceeding by over 30 per cent. the general average of that company. On another group of 1970 lives showing an average systolic pressure of 161.44 mm. representing the rejected risks, followed only in part and under great difficulties, the mortality rate was almost two and one-half times greater than the general average of the company.

*Yet, of this group, at the time of examination, 1082 showed no other impairment than this relatively high systolic pressure and the individuals composing this subgroup, and showing but the single blemish (B. P. 161.44 mm. Hg.), yielded an excess mortality more than double that properly to be expected.**

The experience of Dr. Fisher and other medical directors in relation to life insurance corresponds accurately with the clinical observations of the author extending over a period of several years.

Faught's Formula.—Faught suggests that one may determine approximately the normal limit of blood pressure (palpatory) for any age by assuming 120 mm. Hg. as the normal for the male adult of twenty years, and adding 1 mm. for each two years of added age.

An arbitrary deduction of 10 mm. Hg. is made at all ages in the case of women.

ABNORMALLY HIGH PRESSURE.—*In chronic interstitial nephritis* the elements of arteriosclerosis, high peripheral resistance from toxic hypertonus of the blood vessels and increased heart energy bring about high systolic readings (160 to 300 or even higher) and a markedly high diastolic level. *In both "acute nephritis" and "the chronic parenchymatous lesion,"* the pressure is usually decidedly raised though seldom to the same degree as in the *chronic* interstitial save during uremic seizures. It may be significantly high in acute nephritis before edema is manifest and attain and maintain a high level throughout the acute stage. *In amyloid kidney the pressure* is not raised, but is usually abnormally or even extremely low.

Important temporary increase.

In abnormal nervous excitement temporary high pressure may be observed and this may be very decided in victims of preëxisting chronic hypertension.

In secondary myocarditis with associated splanchnic spastic crises, lead poisoning, gout and actual or impending cerebral hemorrhage, relatively high readings are present.

Cerebral Arterial Crises.—Fleeting aphasia, vertigo and syncope, associated with decided hypertension, have been observed frequently by the author. Less often an actual transient hemiplegia has occurred and in most instances this has proven a prelude to an actual apoplectic "stroke."

Lead Poisoning.—The chief characteristics of the arterial hypertension of lead poisoning are high systolic and *relatively low diastolic* pressure resulting in *increased pulse pressure* with a low diastolic level. This is in rather decided contrast to the high diastolic and systolic levels of interstitial nephritis. The distinction is lost, however, if, as so often happens in chronic plumbism, renal involvement develops. Marked exacerbations of pressure occur, especially in cases of encephalopathy, and apparently are associated with a

* "The Value of Blood-pressure Readings in Examinations for Life Insurance," J. W. Fisher, M. D., Lancet-Clinic, Cincinnati, No. 7, vol. cxiii, Feb. 15, 1915.

vascular spasm, usually visibly reflected in the condition of the retinal arteries.

Four-fifths of the workers in lead show high arterial pressure, even in the absence of other symptoms of plumbism (Norris and others). Bradycardia is commonly observed (55 per cent.). Excessively high (250–300 mm.) systolic readings in cases showing tremor or obscure cerebral symptoms in renal disease should always *suggest* the possibility of lead encephalopathy.

Transient stupor or coma, fleeting delirium or transient or persistent delusions, maniacal excitement, hysteroid manifestations and epileptiform seizures are some of the symptoms encountered and uremia may be closely simulated.

In impending uremic seizures *a rise in pressure to* 300 *has been several times observed by the author. The pressure may recede sharply but is likely to remain high until a balance is again attained or the fatal issue approaches.*

A reading of 420 mm. with recovery has been reported in puerperal eclampsia.

In general arteriosclerosis alone it is unlikely that pressure readings of over 170 are often observed and these only in exceptional instances. *On the other hand, in well-marked instances a distinctly low pressure may be found and it is probable that our former views with relation to the matter must be greatly modified.*

In aortic regurgitation *the "pulse pressure"* is unusually high (60 to 120+) because of the high systolic and extremely low diastolic pressure characteristic of that lesion, and is largely due to increased capillary flowage, to the left ventricular hypertrophy, and the unsustained blood column.* High pulse pressure.

In middle-aged individuals carrying this lesion the systolic pressure may run from 170 to 230, with diastolic readings of 30 to 60 respectively. The highest systolic-diastolic readings with *relatively* lower pulse pressures are seen in cases showing more or less renal involvement and arteriosclerosis.

In free regurgitation with a large, powerfully contracting ventricle, one may obtain systolic readings of 230 mm. or more with a diastolic of 30 mm.

The pulse pressure in aortic leakage is of some value in determining the freedom of regurgitation. An abnormally high variation between the arterial tension in the arm and the leg (60–160 mm.) exists in pure and fully compensated cases.

Intracranial Pressure.—*High Tension.*—This form may be due to meningitis, tumor, thrombosis, meningeal hemorrhage, and the like and produces high pressure through brain center anemia and a reflex extreme vasomotor constriction of the cerebral vessels which in its turn excites the heart to powerful contraction. (Apoplexy is usually included in this group, but is more *likely to follow* a long-sustained hypertension than to initiate it.) *In conditions of coma* this may be most confusing and lead to an erroneous diagnosis

Brain center anemia or asphyxia.

Misleading hypertension.

Coma.

* Wiggers believes that the element of vasodilatation plays little if any part. From a purely clinical standpoint one would strongly affirm the constant presence of moderate vasodilatation, meeting in some degree the demand of the tissues for a more stable capillary flow.

Epileptic seizures.

of uremia. *Some epileptic seizures are also temporarily* most misleading because of attendant hypertension, but the pressure falls promptly to normal or oftentimes below normal as soon as the attack subsides, unless it be of

An exception.

the Jacksonian type in which event it may behave as do other forms of increased intracranial pressure.

Splanchnic crises and lightning pains.

Locomotor Ataxia.—Splanchnic spastic crises and lightning pains may produce transitory high arterial pressure and in this, as in intermittent claudication, vasoconstriction seems to be the fundamental cause.

Angina Pectoris.—In most instances, according to the author's experience, this is associated with a sharp or even maximal rise in pressure oftentimes followed by a sudden drop to or below the antecedent pressure and this hypertension is most extreme in cases complicated by interstitial nephritis.

The Adams-Stokes syndrome may or may not be associated with high arterial tension but the readings during the severe seizures with feeble or absent systole are, of course, reduced to an approximate zero.

Increased viscosity and blood volume.

Erythremia (*Chronic Splenomegalic Polycythemia*).—Many cases show a blood pressure much above the normal. In two, observed by the author, the pressure variations resembled those of an interstitial nephritis. One showed marked arteriosclerosis, the other none.

In these cases both viscosity and total blood volume are greatly increased.

Often overlooked.

The Heightened Pressure of Stasis.—This is a form of contributory hypertension extremely common and unfortunately often overlooked, disregarded or misinterpreted.

An asphyxial phenomenon.

It occurs most typically in cases of interstitial nephritis and mitral disease with a weakening or failing myocardium and is due to an asphyxial irritation of the centers and resulting vasoconstriction associated with an overload of carbon dioxide in the blood.

In such cases an acidosis is usually present which greatly reduces the tolerance of the respiratory center to CO_2 and may thus produce dyspnea without marked pulmonary stasis.

A vicious circle.

The fact that digitalis and rest are the best remedies shows that the immediate indication is increased heart strength and blood flow. Although an asphyxial phenomenon no extreme signs of stasis, such as deep cyanosis, need be present. Inasmuch as it is the center itself which is chiefly affected, under a carbon dioxide blood overload, the vessels contract and the heart coincidently slows and loses tonus, thus increasing the overload and establishing a vicious circle.

In diphtheria the blood pressure may be of the utmost importance in relation to the not infrequent deadly combination of acute dilatation and the splanchnic paralysis of central origin. It should certainly be watched carefully in such cases for it is unlikely that either condition is unheralded by significant blood-pressure variation in the way of hypotension.

Shock and hemorrhage.

Abnormally Low Pressure.—*The conditions giving the lowest readings are toxic paralysis of the vasomotor center, shock, collapse, visceral perforation, and concealed hemorrhage.*

The lowest recorded persistent low reading followed by recovery is 50 mm. Hg. (Neu).

In visible hemorrhage attended by nervous excitement, fear, and apprehension the pressure may be raised though if actual exsanguination be induced it will be low.

The acute infectious diseases, pleurisy with effusion, cholera, dysentery and severe diarrheas, anemias and cachexias and the terminal stages of all diseases usually show low pressure.

Acute Infections with Persistent Hypotension.—The low pressure is probably due to vasomotor relaxation from toxemia of the centers together with, or without, profound myocardial weakness, and sudden death may occur from either one or from both causes. Death may result from mere emotion or slight exertion in such toxic cases.

Toxemic centers.

The degree of the hypotension manifest in all severe cases of diphtheria measures roughly the grade of toxemia, but in laryngeal cases an "asphyxial" higher level is present until the obstruction is relieved by tracheotomy or intubation.

A sudden drop in blood pressure is an ominous sign, and a progressive lowering of the level during the second week of the disease is of grave significance.

May be a danger signal.

A similar but less extreme hypotension occurs in dysentery.

Cholera.—Systolic readings of 60 mm. are reported as occurring during the algid stage and 70–75 is a relatively common reading.

As stated by Dr. G. W. Norris in his admirable book* dealing with blood-pressure the response of a lowered systolic level to saline transfusions and epinephrin constitutes "a satisfactory criterion" of the amount required.

A useful criterion.

Pneumonia.—The late Dr. Geo. Gibson of Edinboro first stated that a pressure *persistently* and decidedly below normal in pneumonia was "of evil omen," and that a decided drop "bodes disaster." He proposed the following formula:

When the arterial pressure expressed in millimeters of mercury falls below the number of heart beats per minute the fact is of evil augury.

This may be accepted as a rough clinical rule vitiated somewhat by the fact that high pressure may have existed before the attack of pneumonia; that cases showing absolute or relative high tension or a normal level not infrequently prove fatal; and that many factors affecting the pulse rate stand quite apart from blood-pressure variations.

Fallible but useful.

Howell states that a study of the four auscultatory phases is useful and believes that clear sounds and regularity of rhythm are of good augury, and that persistence of the relatively impermanent second phase is of especially good import. One must not overlook, however, the right heart and the cardinal prognostic value of pulmonary accentuation and grade of cyanosis.

Bronchial Asthma.—This condition is nearly always associated with decided hypertension, which in cardiorenal cases may be extreme.

Hypertension occurring in the bronchial form doubtless is accounted for

* "Blood Pressure. Its Clinical Applications." Lea and Febiger, 1916.

by the fright and distress attending the attack together with the asphyxial element.

It is said to be low in the misleading cardiac form, but this has not been true of the few instances enountered by the author in which readings were obtained.

Pneumothorax.—Conflicting reports only are available with relation to blood pressures in this condition.

Obviously much depends upon the mode of onset, the degree of intrathoracic pressure, the question of a preëxistent tuberculosis and the stage of the process.

Those of sudden onset are associated doubtless with an abrupt drop in arterial tension and those showing marked dyspnea and cyanosis should show a moderate psychic and asphyxial rise in pressure.

Tuberculosis.—Active tuberculosis wherever seated is associated with moderate hypotension in most instances and one must extend this statement to cover many obsolete and inactive cases.

Many, if not most of the latter, fall under the head of "chronic congenital asthenia" elsewhere described.

Chronic Universal Congenital Asthenia.—This more or less visceroptotic functionally unstable, structurally deficient, and non-resistant group yields many instances of hypotension.

The high percentage of obsolete or latent tuberculous foci revealed by the roentgen ray and tuberculin tests in these supersusceptible yet resistant individuals renders it difficult to determine whether or not they may be considered as a group apart with relation to hypotension.

Lowered Splanchnic Tonus.—In the hypotonias of congenital asthenia in its subnutritional form one assumes a loss of splanchnic tonus similar, but less in degree, to that present in shock.

In many instances there appears to be a generalized lack of vascular tonus associated with the peculiar relative atonicity of the myocardium of the "drop"- or modified "drop"-heart characteristic of these cases.

Syphilis.—*Hypotension* is the rule probably in the more active earlier stages of syphilis, which, in its later phases, constitutes a not uncommon etiologic factor in *hypertension.*

Pericardial Effusion.—The effect of a large exudate is to produce low tension and a small pulse pressure, and the degree of reduction, or better its persistent lowering, is one of the bad signs of this condition.

Normally the pericardium tends to limit or prevent excessive dilatation and reënforce especially the weaker right heart and the left auricle.

When the sac is excessively distended by an effusion the right auricle is compressed and cannot receive and pass on the proper amount of blood to the embarrassed ventricles. Deficient blood supply and insufficient activity of the heart as a whole contribute largely to the production of ypotension.

Aortic Aneurysm.—No striking diagnostic points are elicitable with reference to this lesion. The associated conditions determine the pressure variations for the most part.

Dr. R. Edwin Morris reports, moreover, that decided *unilateral* pulse and blood pressure differences are common in individuals lacking any sign of aneurysm, marked arteriosclerosis or cervical rib.

Chronic Alcoholism.—In delirium tremens *hypotension* is marked during the stage of active delirium.

In cases of chronic alcoholism presenting vague symptoms, psychasthenic and otherwise, a curiously constant rise of systolic pressure (20–70 mm.) occurs and endures for several days. The systolic pressure remains relatively high.

Raff has found the phenomenon of considerable differential value.*

Morphinism.—The blood pressure in the morphin *habitués* of the better grades of society or those of any class who are relatively well nourished are said to show high pressures which are strikingly reduced by the free catharsis now universally used prior to withdrawal of the drug.

A suggestive finding.

Norris properly points out the fact, however, that the patients of this type admitted to the public clinics are usually in a state of semistarvation, profound cachexia and *hypotension*.

Public patients.

Anesthesia.—*Ether.*—The primary sharp and oftentimes excessive increase in arterial tension due to excessive stimulation of the psychic sphere and the struggles of a resisting patient may be a source of great danger in cases of aortic aneurysm, antecedent hypertension and myocardial weakness of any type.

Surgically important.

During full anesthesia the blood pressure is maintained usually at about the normal level by stimulation of the respiratory centers, sometimes so excessive as to cause temporary suspension of the breathing. Arrest of respiration in the stage of full anesthesia is the result of paralysis of the centers.

The combination of *oxygen and ether* produces a considerable rise in blood pressure throughout the anesthesia.

Chloroform anesthesia is characterized by an *early fall in pressure* increasing *pari passu* with the deepening of the state of unconsciousness, and in fatal cases continuing its descent until death.

It is asserted at present that this is wholly due to the persistent cardiac depression characteristic of the drug, but it would seem unlikely that this is the last word, and probable that peripheral vasodilatation plays a considerable part. Not only progressive myocardial weakness, but also an increasing dilatation are present in lethal cases.

The early temporary arrest of heart action often encountered is a respiratory reflex inhibitory effect which may prove fatal in cases of myocardial degeneration or inflammation.

Nitrous oxid slows the pulse and raises blood pressure. This anesthetic is somewhat dangerous in cases of decided chronic arterial hypertension.

Caution.

Ethyl chlorid exerts an effect wholly comparable to chloroform, but in a greatly lessened degree.

Typhoid fever is characteristically a disease associated with persistent

* Dr. Karl Raff, *Deutsche Archiv. f. klinische Medizin*, vol. cxii, p. 209.

Low tension.

and progressively increasing hypotension though this usually does not reach extreme figures (95 mm.). Sudden drops are ominous.

Important sign.

The advantage of routine observations is especially marked in relation to hemorrhage and perforation where a *sharp and sudden* drop may indicate hemorrhage, and a decided secondary rise (within from two to four hours) point to developing peritonitis from perforation or to renal involvement.

Malaria.—In malarial cachexia the blood pressure usually is low.

Scarlet Fever.—Hypotension or normal pressure are usually present. *A decided rise in blood pressure is of great importance as suggesting the coming of a complicating nephritis*, for it may precede by several days the onset of frank signs or even the albuminuria. On the other hand, albuminuria may occur without such an ascent of pressure, and may be merely that of toxemia of the lesser grades.

Norris emphasizes the following facts with relation to hypotension in acute infectious diseases. They may be summarized as follows:

1. Hypotension occurs in practically all febrile infections, being especially marked in certain forms.

2. In so-called *sthenic* fevers the fall of pressure is trifling.

3. In *asthenic* fevers arterial hypotension is pronounced and death in many cases is attributable to vasomotor failure quite as much as to a failing myocardium.

4. Toxemia rather than fever determines the degree of hypotension.

5. Degeneration or disturbed function of the adrenal glands occurs in some febrile infectious conditions, notably in diphtheria.

6. Decrease of *pulse pressure* usually represents a systolic fall and is a bad symptom; well sustained *pulse pressure*, a favorable sign.

7. Practically any acute infection may produce cardiovascular damage.

8. A convalescent patient should not be permitted to leave his bed so long as there is a marked difference between the pulse rates in the erect as compared with the recumbent posture.

9. Minor degrees of physical exertion in unfit convalescents may cause a fall of 30 or 40 mm. Hg. systolic pressure.

10. Arbitrary standards cannot be set forth in figures. The essential prognostic factors relate to the *course of the blood pressure, diastolic* and *systolic*, upward or downward, and to the *lability* or stability of arterial tension.

Shock.

Hypotension Attending the Sudden Relief of Abdominal Pressure.—The danger from shock which is associated with the too rapid removal of preëxisting abdominal pressure is well known.

In the withdrawal of fluid from the abdominal cavity, a decided temporary hypotension is induced which is much less marked if the flow is gradual and constriction of the abdomen maintained throughout.

Pleural Effusion.—A large pleural effusion whether transudate or exudate causes a rise in blood pressure; its removal, a fall coincident with its withdrawal, and to a degree measurably corresponding to the relief of intrathoracic pressure afforded, but dropping temporarily below the level of arterial tension normal or habitual for the individual. The dangers attending the

Important data.

withdrawal of exudates too rapidly or the yet greater one dependent upon undue irritation of the pleura are well known.

The fall in blood pressure during thoracentesis (20 mm.+) is more rapid in the case of chronic than in recent or acute effusions and the only fatal instance observed by the author occurred in the case of a patient suffering from a multiple serositis, whose chest had been aspirated again and again with impunity. Vertigo is a symptom to be regarded and shallow rapid inspiration may precede syncope. A fatal case.

In the case of both *paracentesis abdominalis* and *thoracentesis* the lack of efficient preliminary local anesthesia, an unnecessary fussiness, and the unwise assembling and parade of instruments, greatly intensifies the tendency to syncopal seizures. A common error.

Both operations are ordinarily trifling in their nature and the patient should be spared mental perturbation so far as possible.

Abdominal Colics.—Practically every form of severe abdominal pain of this character may be associated with moderate rise in pressure. An excessive ascent in cases of preëxisting arterial hypertension has been observed by the author several times in cases of angina pectoris with abdominal pain maxima, and this statement also applies to Pal's "splanchnic crises," possibly misnamed, certainly too often diagnosed. Pressure rises.

Blood-pressure Determinations in Pregnancy.—Significant and even critical arterial hypertension may precede the appearance of albumin in the urine. No pregnant woman should be allowed to go through delivery without receiving the benefit of repeated blood-pressure determinations especially during the later months, these being of infinitely greater importance than the examination of the urine. Any case which shows readings distinctly above normal should be carefully watched and if the rise is progressive as well as persistent and reaches or exceeds 145 or 150 mm. it constitutes a danger signal. More important than urinalysis.

In cases of pregnancy *associated with marked hypertension* the rupture of the membranes is followed by a drop of from 60 to 90 mm. of mercury, a rebound of nearly as great an amplitude, a further and second recession following actual delivery, a second rebound, and then and thereafter a gradual return to normal occupying from three to seven days (Anders and Boston). A curious sequence.

Acute heart lesions, with profound weakness, show decided low pressure unless a marked asphyxia of the centers is present.

Points of Importance.—*A low systolic coexisting with a high diastolic pressure* (100+) may prove of great significance in the early stages of an interstitial nephritis and is often present in the *terminal* stages of cardiorenal disease when the myocardial reserve is near to exhaustion.

A high systolic and normal diastolic pressure may result from purely temporary conditions such as emotional upsets or excessive exertion in an untrained individual.

A high systolic and extremely low diastolic pressure almost invariably means aortic regurgitation even though the murmur be absent. If very marked it

will be associated probably with Duroziez's sign in the femorals and a well-marked Corrigan pulse. If true aortic stenosis exists, this will be modified and the pulse pressure reduced, but usually retains its chief characteristics. According to the author's limited observations, frank exophthalmic goiter usually shows a similar but less marked pressure curve.

A low systolic and diastolic pressure in ambulant patients usually represents residual cardiovascular effects of past acute illness and in any event strongly suggests lack of cardiovascular reserve.

Of late the value of this finding as an indication of cardiovascular depression or actual myocardial weakness has been emphasized by the clinical experience of the author and the check afforded by the teleoroentgenogram, electrocardiogram and polygram.

It is encountered with especial frequency in individuals of the congenital asthenic habitus carrying concealed septic foci.

Its occurrence in terminal cases of heart disease and especially in post hypertension cases with failing hearts is well known and the author encounters a decided drop in blood pressure frequently in individuals who are experiencing a definite impairment of what was previously an adequate compensatory reserve.

In heart cases with a clear history of physical overstrain an initial low blood pressure may be seen to heighten daily under treatment.

In cases of chronic arterial hypertension a pressure of 175–185 with a diastolic pressure above 100 appears to be well borne for years (author's personal observation), but pressures of 190–200+ convey a threat, and those of 220+ mean that the sword overhead is suspended by a hair.

In this connection the author ventures to assert his utter disbelief in the widely accepted doctrine of the conservative effects of decidedly high blood pressure in cardiorenal cases.

In nearly all of the *non-terminal* cases carrying a blood pressure of 200 plus, in which, fortuitously or otherwise, a drop to 160–175 mm. Hg. has occurred, this event has been associated with immediate and decided betterment, not only with respect to myocardial reserve, but also in a number of instances, decided shrinkage in the cardiac outline. This does not justify the use of an over-radical and depleting therapy, prove that a moderately increased blood pressure is never of good augury, or that excessive abrupt falls in pressure may not sometimes be ominous, but it suggests the danger inherent in the prevalent attitude. The point has been emphasized in a certain small group of cases in which by a happy chance the author found intermittent hypertension before any albumin appeared in the urine.

In several of these individuals the adaptive changes in the heart itself were interrupted by hypertensive periods which *definitely increased* certain minor decompensatory phenomena.

In cardiorenal cases high blood pressure would appear to be primarily and chiefly a toxemic manifestation.

Those particularly affected were of the congenital asthenic type, but the same phenomena have been observed in others.

Chief Clinical Significance of High Diastolic Pressure.—*As a matter of practical application a decided and persistent hypertension with a high or relatively high diastolic level means chronic nephritis, until another cause is proven or rendered a reasonable assumption.*

This statement holds good regardless of the absence of the classical urinary findings at the time that hypertension is become manifest.

In other words, chronic Bright's disease with or without albumin and casts, but seldom without evidences of impaired renal permeability, is so commonly the cause as to place all other claimants among the "possibilities" only.

In the group described in the preceding paragraphs *periods* of impermeability coincided with intervals of increased arterial tension.

Blood-pressure Estimations in the Arrhythmias.—As stated previously, the difficulties encountered in taking the blood pressure when certain forms of irregularity are present may be insurmountable with respect to diastolic pressures.

This is true of all arrhythmias in which inequality of force plays a part, *i.e.*, auricular fibrillation, alternation, extrasystolic irregularities, etc. In such cases one can only determine the "highest" and "lowest" pressures at which the systolic sound is heard by the auscultatory method or, less accurately, record the levels by the palpatory method.

With respect to the auscultatory method, this means merely the taking of the reading at the maximal pressure at which the first sound is heard and then slowly and carefully lowering and raising it until the sound of every systolic wave is heard.

A count of the beats per minute then audible at the brachial should be made during several minutes and compared with a similar count made by direct auscultation over the heart. In both extrasystolic irregularity and fibrillation certain ventricular contractions may fail to open the aortic valves.*

Pulse Deficit.—Such comparison of the number of beats reaching the radial or brachial artery with that of the heart itself as determined by auscultation direct is a matter of considerable interest and some importance.

Venous Pressure.—The venous pressure may be roughly estimated by Oliver's method which consists in allowing the arm to hang downward so as to distend the veins, noting the height at which venous collapse occurs when the extended arm is raised.

Normally the fulness is lost ½ inch above the level of the heart apex. Each additional inch represents a venous pressure equal to 2 mm. of mercury (1.985). Other methods of measuring venous pressure involve more trouble, time and apparatus than the information gained is worth to the clinician.

* James and Hart obtain an "average" systolic pressure in such cases by making a systolic pressure record for each 10-mm. reduction of pressure, counting the systolic beats per minute for each level while an assistant counts the beats as heard over the heart itself during the same period. The systolic beats heard per minute over the artery at each level is multiplied by the reading obtained at that level and the sum of the products is divided by the number of apex-beats per minute.

High figures are often present in marked systemic stasis such as occurs in right heart decompensation from whatever cause.

THE RADIAL PULSE

"TAKING" THE PULSE.—Relative Value of Methods.—In general clinical value, instrumental methods cannot compare with pulse-taking by the old method. The palpable beat of the radial pulse represents normally the sudden systolic increase of arterial tension and it is felt only when the artery is partially compressed against some rigid supporting tissue.

What the pulse is.

Useful methods.

Pulse Palpation.—Whenever possible the pulse should be taken casually, while talking of other matters, and due allowance made for the nervousness incident to examination and the effect of any antecedent physical exertion or excitement. In children, especially, accurate determination of the actual rate is often impossible unless the youngster is asleep.

The Importance of Correct Technic.—A correct technic is of the utmost importance and the patient's arms should be similarly placed, in a position free from restraint, flexion, or muscular compression of the vessels. *The pulse should be taken simultaneously in the two radials*, the pulp, not the extreme tip, of three fingers (mid-, fore- and ring-finger) being applied lightly over the artery at the wrist. (There is no better proof of bad training than the gingerly one-finger-tip approach sometimes observed.)

Position of Arm.—*Faulty position of the patient's arm and hand may greatly modify any pulse.*

The forearm should be slightly flexed, the wrist thrown slightly backward and a trifle supinated.

Points to be Determined.—(1) *The "size" of the artery.* (2) *Pulse rate or frequency.* (3) *Rhythm.* (4) *Uniformity of strength.* (5) *Synchronism and equality of the right and left radial pulses.* (6) *The force required to obliterate them (tension).* (7) *Abnormal thickening of the artery (arteriosclerosis).* (8) *Correspondence of radial rate and rhythm to the auscultatory findings.* (9) *Peculiar variations in the character and quality of the beats.*

When the physician's fingers are applied to the artery the first four points (*"size," "rate," "rhythm" and "force"*) are determined almost unconsciously and instantaneously, the vessel being lightly rolled under the finger to get its size, which for the most part represents its "tonus" or state of contraction and not the force or efficiency of the beat.

Pressure is then made with the upper finger until the pulse is lost to the lower, the force exerted being a rough and unreliable, yet extremely useful, measure of arterial *tension.*

Tension.

The artery, thus emptied, is rolled under the lower finger to detect any thickening of its walls (arteriosclerosis) and any vessel that can thus be felt as a distinct tube when collapsed is sclerotic and hence abnormal.

Sclerosis.

Such vessels may be just palpable as a tube, definitely thickened, distinctly rigid, furrowed transversely and rigid, or carry multiple tiny plaques or beads of deposited lime salts.

Goose gullet arteries.

Determining the Rate.—It is ordinarily sufficient to count the pulse for thirty seconds and multiply by 2 to get the rate per minute, but in severe ailments or if any abnormalities in rhythm, quality, or force be present, it should be taken for at least one minute, and, if excessively rapid, it may be necessary to count each second or third beat only and apply the proper multiplier.

Proper methods.

Useful device.

Not infrequently, especially in myocardial degeneration, coronary sclerosis and certain valvular lesions, some systoles are premature and inefficient (extrasystoles), and though readily auscultated, may yield no pulse wave or a very feeble one. In such cases nurses' pulse records are often worthless with respect to actual cardiac beats. Nurses should be instructed with relation to the commoner types of intermission and irregularity.

Lost beats.

Vitiated records.

The Recurrent Pulse.—This is one which cannot be cut off by the pressure of the upper finger because of an unusually free communication and recurrent flow from the palmar arch. The difficulty may be surmounted by making obliterative pressure with the lower finger and carrying out the usual procedure above, or by compressing the ulnar artery.

Unilateral Variations.—A student frequently jumps at faulty and far-fetched conclusions if he finds an unilateral weakening or absence of the pulse. Either finding may or may not indicate deficient heart strength, but is ordinarily due to an abnormal course of the radial artery, or, more rarely, to actual blocking of the vessel, to aortic aneurysm, *cervical rib*, or to pressure of new growths. The findings must be always checked by comparing the larger arteries, such as the brachials of the two sides.

Often trivial.

Sometimes important.

In aneurysm of the ascending portion of the arch involving the innominate, the right radial and carotid may be affected; in *aneurysm of the descending aorta*, the left radial; or a delayed pulse may accompany aneurysm of the transverse portion. Direct sac pressure upon arteries abnormally constricted, deformed arterial openings, or the complete or partial conversion of intermittent into what may appear to be a continuous weakened pressure by the aneurysmal sac, make many variations possible, aside from those caused by adhesions due to associated perianeurysmal inflammation in certain cases. *Too much reliance should not be placed upon this as a sign of aneurysm, as it is often lacking or without exact significance.*

Important inferences.

In obscure chronic cases in which pain is present in the lower extremities upon exertion, whether cramp-like, rheumatoid or paroxysmal, the arterial pulse should be taken in the *dorsalis pedis and posterior tibial* arteries.

Importance great.

Such pain is often due to obliterative arteriosclerosis and vasomotor spasm (intermittent claudication) or may be associated with Raynaud's disease and always demands a careful examination of the heart and kidneys.

A full, bounding pulse is frequently mistaken for arterial hypertension because of faulty technic. Arterial hypertension may be *detected* by the finger in many instances, but correct determination requires the use of special instruments.*

A common error.

* No excuse remains at the present day for *dependence* upon the fingers for the estimation of blood pressure. Most humiliating errors are sure to occur if the unaided senses are employed

Fingers may fail one.

Auscultation is of course often necessary to reveal the true rate and rhythm of the heart beat and in certain cases of paroxysmal tachycardia pulsus, alternans, or auricular flutter, instrumental determination alone yields strictly accurate results.

Rate readily affected.

PULSE FREQUENCY.—Exertion and Attitude.—Aside from mental excitement, the digestive process and bodily exertion, which latter includes even the changing of the posture in bed or evacuation of the bladder or bowels, the position of the body affects the pulse rate.

Erect posture.

In the erect posture the rate is from 10 to 15 beats faster than in recumbency. (This variation was formerly believed to be due to rise in arterial tension, a theory exploded by the fact that the sphygmomanometer shows but trifling variations in systolic pressure and that often downward. Increased intra-abdominal pressure is undoubtedly a factor in many postural changes.)

Important sign.

The significance of an abnormally great increase of rate upon slight, unusual exertion, unattended by excitement, is very great inasmuch as it clearly indicates cardiac weakness and a limitation of the field of response (reserve).

Danger signal.

In cases of profound cardiac weakness such a maneuver frequently develops coincidently a suggestive subjective or objective dyspnea or substernal or epigastric distress.

Congenitally asthenic individuals with their narrow hanging ("drop") hearts show a remarkably labile heart action during periods of nutritional depression especially if, as so often happens, the atonic myocardium is actually dilated.

Persistent rapidity.

Alcohol, tobacco, the coal-tar products, arsenic and other toxic substances used in excess, *may occasion a persistent lability and increase in rate, and cachexias, hyperthyroidism and especially incipient tuberculosis should also receive attention in this connection.*

Infancy.

Age.—The average normal rates are for the first year of life from 130 to 140; gradually dropping from the first to the fourth year to 105 or 110, and so diminishing until at the fifteenth or sixteenth year it reaches from 75 to 80 beats per minute. During middle age and up to sixty years frequency is slightly diminished, sometimes increasing somewhat beyond that age.

Childhood.

Maturity.

Sex.—*Women show a rate which averages 5 to 8 beats faster than men, and it is slightly slower in tall than in short persons.*

Fever with Increased Pulse Rate.—Any decided persistent increase in pulse rate, lacking obvious cause, demands the use of the clinical thermometer and we find that, as a rule, the rate increases from 8 to 10 beats per minute for each degree Fahrenheit above the normal, and further that the behavior of the pulse in fever is of both diagnostic and prognostic importance.* In general an increase in the rate out of correspondence with the rule given suggests profound toxemia or diminished resistance to the disease and constitutes a danger signal. In those few diseases in which the pulse rate should be lower than the rule, excessive rapidity has still greater significance.

Tachycardia.

In fever a relatively rapid rate is of far less significance in the child than in the adult.

* See "Fever."

Slow Pulse.—*High temperature and relatively slow pulse are observed especially in tubercular meningitis, typhoid fever, and lobar pneumonia as well as in febrile ailments associated with some of the organic causes of slow heart, such as coronary sclerosis, myocarditis, aortic stenosis and especially "heart block."* Relative slowness.

Rapid Pulse.—*A rapid pulse occurs in many non-febrile ailments, most markedly in exophthalmic goiter,* and may be decided in the decompensatory stages of mitral stenosis and insufficiency, myocarditis, aortic insufficiency, acute endocarditis, pericarditis, and the pressure displacements of the heart. Paroxysmal tachycardia, alternation, auricular flutter and delirium cordis furnish striking examples of the special types of rapid pulse.

An excessively weak, thready, rapid "*running*" pulse associated with marked evidence of cardiac weakness is a forerunner of death in certain diseases and may represent auricular flutter or alternation. An ominous symptom.

Tachycardia.—(*"Swift Heart"*).—Mere excessive rapidity is known as tachycardia, and as used alone this term should be strictly limited to the pulse free from irregularity, intermittency, inequality or paroxysmal rapidity.

Scarlatina and Tuberculosis.—*In children an excessively accelerated pulse may suggest the onset of scarlatina before the appearance of the rash, and in incipient pulmonary tuberculosis a labile and persistently overrapid pulse is one of the most constant of the early signs.*

Various Factors.—In nearly all cachectic states weakness, acceleration and lability are present and it is an important confirmatory sign in severe shock and collapse. Aside from these must be considered sexual psychasthenia, the overuse of snuff or tobacco (often associated with palpitation or sinus arrhythmia), physical and mental overstrain, and idiosyncrasy.

Palpitation.—The term tachycardia covers most of the cases commonly described as "**palpitation,**" the heart action being in such cases quite normal save for a rapidity which seldom exceeds 170 beats per minute (Mackenzie), and showing no abnormalities of origin or transmission, when subjected to analysis with instruments of precision, unless it be an occasional extrasystole. In such cases the attack subsides gradually and excitement, radical changes of posture, and physical exercise markedly affect the rate.

Bradycardia.—(*Slow Heart*).—Pulse below 60. A slow regular pulse may be congenital and physiologic, but most of those thus described have subsequently been proven to be due to heart block, chronic toxemia or organic disease of the heart or brain, the latter being associated with increased intracranial pressures. Indeed any regular pulse under 50 usually indicates "heart block," *i.e.*, blocking of certain of the impulses normally passing from auricle to ventricle through the "bundle of His" or in cases of extremely low rate, a complete block and dissociation of auricle and ventricle. Seldom physiologic. Significance of excessive slowness. Often partial "heart block."

The lesser degrees of slowing may be met with as toxic effects of various drugs, in aortic stenosis, chronic myocarditis, coronary disease, cerebral hemorrhage or tumor, meningitis, epilepsy, general paralysis of the insane, melancholia, mania, myxedema, pituitary disorders, jaundice, uremia, exhausting disease and sometimes in acute ailments, especially diphtheria Many and varied conditions.

in which, no doubt, undeclared myocarditis and heart block are chiefly accountable.

Danger signal

Abrupt Transition to Bradycardia.—*As regards its occurrence in acute infections during either the active stage or convalescence, the rule may be laid down that any sudden and decided drop below the normal or usual rate, unless associated with crisis, is quite as much, if not more, suggestive of danger and need of special watchfulness and care as would be excessive rapidity.*

Toxemia, intracranial pressure and actual disease of the myocardium or bundle of His are therefore chiefly to be considered as its causes.

Slowing of the heart may occur in connection with rapidly induced arterial hypertension. This fact is strikingly illustrated at times in cases of acute nephritis or the onset of uremia.

Sinus Arrhythmias.—*In sinus arrhythmia* there is irregular spacing of the beats and by instrumental methods one discovers that this irregularity arises chiefly from a shortening of the diastole rather than of both the phases of the heart's cycle. With excessive increase of rate this irregularity disappears, with slowing it may reappear.

Such sinus irregularity is recognized by a labile *periodically* shifting pulse rate, quickening with inspiration and slowing with expiration, but showing no inequalities of force in the individual beats.

The variation in the diastolic periods is determined readily in many instances by auscultation, when difficult to recognize in the pulse (Mackenzie).

As a matter of fact if one applies the finest and most careful measurements to electrocardiographic records some variation in the spacing between the diastolic periods can be detected in almost every record.

By ordinary clinical methods these are not determinable nor is the matter one of importance.

Syncopal attacks occur occasionally in this form of tachycardial irregularity. The condition may persist for years but is rarely encountered after the age of 30.

Quite recently in the case of a youth showing a frank myocardial decompensation with septic tonsils and an ethmoiditis, sinus arrhythmia alternated with short periods of fibrillation.

It is usually of slight significance, but one cannot follow James Mackensie in regarding its (frequent) presence after an acute illness as evidence that the heart has escaped *infection.*

It may be assumed, however, that the toxemia has affected the myocardium but slightly and may thus constitute indirect evidence of no serious amage.

Either simple tachycardial "palpitation" or "sinus irregularity" may be the source of much discomfort and quite unnecessary alarm.

Unfortunately the most serious forms of pulse irregularity may give rise to a similar condition and the reader should refer to "extrasystolic irregularity," "fibrillation," "flutter," and "paroxysmal tachycardia" as described in the succeeding section.

Serious error may result either from an overestimation of the gravity of a simple tachycardia or sinus irregularity, or careless disregard of the fact that "palpitation" arises also from some of the most serious cardiovascular conditions.

In simple palpitation fear of death is readily excited by the curious subjective manifestations of precordial, "bumping," "thumping," and "turning over," of the heart, but the curious inward *conviction* of impending death sometimes experienced even in miniature replicas of angina pectoris is seldom present.

IRREGULARITY AND INTERMITTENCY.—These conditions are described fully under the "arrhythmias." **Irregularity** includes variation in volume and strength as well as in wave intervals, though the former are better described by the term "*unequal.*"

By **intermittency** is meant the apparent or actual omission of beats. The stethoscope and more fully the polygraph or electrocardiograph show that intermittency may be due either to premature and inefficient extrasystoles or the "dropped," systoles of partial heart block ("*pulsus deficiens*"). *"Pulsus deficiens."*

"*True pulse intermittency*," therefore, would apply only to the phases of recurrence of inefficient or dropped beats, which result in the transient, persistent or irregularly recurrent actual elimination of pulse waves in the arteries. *"Pulsus intermittens."*

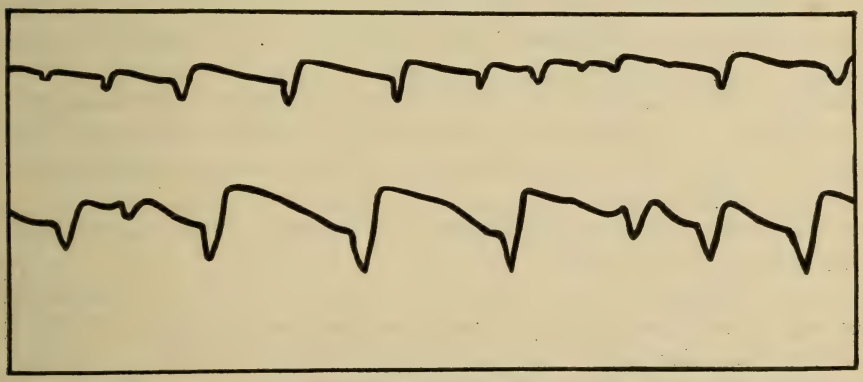


FIG. 186.—Pulsus Irregularis Perpetuus. Two radial tracings taken during auricular fibrillation. Note extreme irregularity in time and force alike. (Drawn from the original tracing of Parsons-Smith.)

If an extrasystole is of sufficient vigor to cause a lesser beat in the arteries there is established during the period of recurrence what might be termed a regular irregularity and inequality with respect to the phases in which it occurs. For the usual type of extrasystolic irregularity, as it is reflected in the pulse, the term "*interrupted pulse*" would apply more aptly.

The fundamental cause (extrasystoles of the heart) may therefore be expressed by either an intermittent or irregular radial pulse.

In the absence of definite clinical findings their significance is far more serious in middle-aged, or elderly people than in the young and both variations admit of subdivisions. (See pp. 546 and 549.) **Significance.**

Delirium cordis.

Pulsus Irregularis Perpetuus.—*Irregularity is frequently combined with inequality and excessive frequency (as in "delirium cordis" due to auricular fibrillation) and may be transient, variable, persistent or periodic.*

Alternation.

A strong beat may be felt to alternate with a feeble one (*pulsus alternans*), though alternation often escapes the finger even when demonstrable by the polygraph, or, double or triple beats may occur with a prolonged pause between the last beat of the series and the next succeeding beat (*pulsus bigeminus*) (*pulsus trigeminus*), in extrasystolic irregularity, or, the normal variation due to deep inspiration may be so exaggerated as to make the radial pulse weak or imperceptible during inspiration (*pulsus paradoxus*).*

Pulsus bigeminus and trigeminus.

"The Paradoxic Pulse."—This is most frequently observed in excessive cardiac weakness, especially if associated with obstructive pressure such as may be exerted by pericardial effusion, mediastinal tumor (whether aneurysmal, glandular or malignant), indurative mediastinitis, or laryngeal, tracheal or bronchial stenosis. The essential primary inference in any case is that there exists a weak central and peripheral circulation plus a respiratory drag upon the aorta or great veins, or, in infants and children, and in the presence of other signs, congenital heart disease.

Disease associations.

Pulsus Celer and Pulsus Tardus.—The finger and, better still, the sphygmograph, differentiates the shock-like or tapping effect of a wave of quick rise and descent (*pulsus celer*) from that of slow rise and fall. The former is exemplified by the "*water-hammer pulse*" of aortic insufficiency, the latter (*pulsus tardus*) by the pulse of arteriosclerosis, interstitial nephritis, angina pectoris or pure aortic stenosis. Exact equality in the time of the deliberate rise and fall is sometimes noted in thoracic aneurysm and Sahli rightly affirms *that even the finger* may distinguish between the pulse that is celer in its ascent and tardus in its descent, as is sometimes encountered in lesser grades of arteriosclerosis.

Full Pulse.—The artery feels full *between beats* and may be large or small and of high or low tension.

Large Pulse.—(*Pulsus Magnus*).—The *excursion* of the artery *is marked* as seen in aortic regurgitation (*pulsus magnus et celer*). It may be full as in fever (*bounding pulse*) but is usually of low tension.

Pulsus Vacuus.—This is typified by the unsustained tap delivered to the finger by the water-hammer pulse, less so in extreme malnutrition, severe anemias and asthenias, exhausting fevers, hyperthyroidism and in collapse.

It is essentially the pulse of arteriocapillary relaxation intensified in aortic regurgitation by the failure of support to the blood column due to the incompetent aortic valve.

The Small Pulse (*Pulsus Parvus*).—Aortic and mitral stenosis, myocarditis and some cases of aneurysm furnish the best examples.

* Da Costa states that inspiratory disappearance of the pulse of one side has been noted in a case of adhesion of the pleura to the subclavian artery of the side affected.

In a case observed by the author the pulse was rendered barely perceptible but not wholly lost, a similar inspiratory unilateral effect has been observed by him in several cases of aneurysm of the thoracic aorta.

Monneret's Pulse.—The slowed, soft, languid pulse so often observed in the toxemia of jaundice is sometimes given this special place in our terminology.

Wiry Pulse.—*This small hard pulse is* best marked in peritonitis.

Pulsus durus is that of high arterial tension but not small and wiry.

Dicrotic Pulse.—A recoil following the beat may often be felt more or less distinctly under proper pressure and indicates ordinarily extreme exhaustion or toxemia, early noted in typhoid fever and the typhoid state, but often found in other severe prostrating exhausting diseases of various sorts, and, very rarely, in apparent health after severe exercise.

Exhaustion.

The Capillary Pulse.—*A large pulsus celer produces capillary pulsation.* It is a rhythmic blushing and paling which may be observed over inflammatory areas, in the matrix of the finger-nail on making light pressure over the tips, or in the forehead, chest, ears, etc., by reddening the skin by friction. By steadily pressing a glass slide or even the rim of a goblet upon the mucous membrane of the lips its rhythmically oscillating border is readily seen and it may even be observed in the retinal arteries by one sufficiently skilled in the use of the ophthalmoscope.

Important and suggestive.

It is a valuable confirmatory sign of aortic insufficiency but may also be observed in exophthalmic goiter, asthenia with chlorotic anemia, certain cases of fever, pernicious anemia and acute hemorrhages.

Its associations are essentially those of the "pulsus celer" and it is occasionally observed in persons apparently in good health. The number of decided "physiologic capillary pulse" cases seen by the author has steadily diminished since his discovery of the fact that a split second sound with an area of audibility limited to the third left interspace usually represents incipient aortic leakage and precedes by a longer or shorter period the appearance of a distinct diastolic aortic murmur.

In many instances, apparently physiologic, the arteriocapillary relaxation and increased force of velocity of current necessary to produce ebbing and flowing pressure waves in the capillaries are chargeable to a masked hyperthroidism.

The Bizarre in Pulse Terminology.—The term "*gaseous pulse*" is a reminder of the "olden times" when men studied the pulse more diligently with the fingers than we moderns do with all the refinements of research apparatus at our service.

It has a place in the clinic nevertheless, for it accurately describes the soft, empty, toneless pulse often noted for some hours before death in certain cardiac cases. Gas bubbles rising and collapsing rhythmically under the finger would convey much the same sensation.

The mouse-tail pulse (*decurtate pulse*) is less descriptive of anything familiar to the author unless it applies to the pulse of extremely gradual recession and attenuation occasionally encountered in pure aortic stenosis of high grade.

The Water-hammer Pulse.—This name describes beautifully the *typical, extreme pulsus celer* of pure, free aortic regurgitation with its sharp shock-like impact and instant recoil from the finger.

Instances might be multiplied if one were to quote from the works of the older clinicians and the aptness of many of the descriptive terms together with their multiplicity, is good evidence of the possibilities inherent in the *tactus eruditus* as applied to pulse-taking.

VENOUS PULSATIONS

Indurative mediastinitis.

"Diastolic Jugular Venous Collapse."—This is now regarded as a weak, unsustained, positive venous pulse, but the collapse is particularly marked in "adhesive mediastino-pericarditis" and is rendered more striking by the diastolic recovery of the chest wall, which has been dragged down by the ventricular systole of a greatly enlarged and adherent heart, associated with diastolic intrathoracic suction (Friedreich).

Rare phenomenon.

Positive Penetrating Venous Pulse.—Very rarely and usually in association with the *"capillary pulse"* a wave may be observed in the terminal venules. Compression causes central obliteration of this pulsation but does not affect the peripheral portion.

Visible Respiratory Venous Phenomena.—The swelling of the veins of the neck during violent muscular straining or sustaining a musical note is familar to every one and it is seen in all forms of severe dyspnea, whether temporary or permanent. In chronic conditions the overdistention may become permanent and, occasionally, the usual *inspiratory diminution and expiratory fullness* is reversed, in which case indurative mediastinitis, chronic pericarditis, aneurysm, or any form of mediastinal tumor is suggested.

Common form.

Rare form.

If forced expiratory movements, with the mouth and nose closed, show overfilling of the cervical veins upon one side as compared with the other, intrathoracic venous compression is suggested.

Carotid vs. Jugular Pulsation.—*Students often find difficulty in distinguishing between venous and carotid pulsation; the former covers a wider area, is undulatory and collapsing in character, and obliterative pressure upon the carotid does not affect it.*

Proper Posture.—The patient should be in the dorsal position with the head *raised, supported* by a pillow and turned well to the right, when in about 75 to 80 per cent. of all cases the pulse will be visible.

If distention of the veins prevents visible venous pulsation the head and shoulders must be elevated until the pressure is modified sufficiently, if this be possible.

Much maneuvering and many shifts of position may be necessary to secure results and these should be carried out with the least possible effort or psychic disturbance on the part of the patient. Very little exertion or excitement, for example, may suffice to remove an extrasystolic irregularity or other interesting subsidiary findings.

INSTRUMENTAL METHODS

The True Value of Instrumental Methods

Of genuine clinical value.

Facilitate Research.—The use of the fascinating and delicate instrument of precision in the investigation of cardiovascular disturbances has been

of incalculable value in advancing our knowledge of cardiac physiology and in the clinical field the newer methods have clarified many obscure conditions and *added much of real value* to the resources of the clinician.

The author feels strongly that with respect to the electrocardiograph we may hope that present achievement, remarkable as it is, represents but a part of its capabilities as an aid to clinical research.

He now regards it as of very great value in detecting conditions of genuine importance with relation to both diagnosis and treatment such as cannot be demonstrated without its aid.

Necessary Training.—It is proper and necessary that every earnest student of medicine should know the instruments and, so far as possible, the methods of modern cardiac research, fascinating alike to eye and mind. If possible he should be accustomed to their use so far as to enable him to translate readily the simpler forms of the graphic records which they yield, or, if time and opportunity offer, become sufficiently expert to take up research along this most fascinating and attractive line or even use them clinically to his advantage and that of his patients.

Their value, especially in the *early diagnosis* of myocardial lesions, is extremely great and it is to be regretted that as yet neither the electrocardiograph nor even that simpler instrument, the polygraph, can be utilized widely by the general practitioner.

Simpler Methods Usually Adequate.—*He should realize nevertheless that the major part of the clinically important circulatory phenomena registered by modern recording instruments may be recognized and interpreted by the simpler means of ordinary clinical technic, if this be founded upon an adequate knowledge of modern cardiac physiology and pathology.*

Useful but not essential.

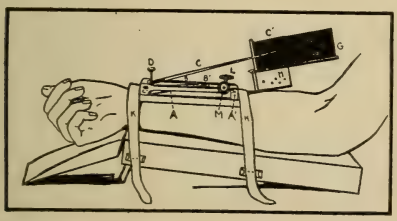


FIG. 187.—Marey's sphygmograph. Now nearly obsolete, because of its slight value and the far greater usefulness of polygraphic tracings. It was never a satisfactory instrument.

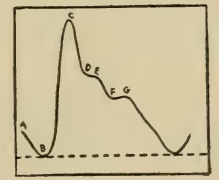


FIG. 188.—Ideal radial sphygmogram. b.c., percussion upstroke; b.c.d., percussion wave; d.e.f., tidal wave; g., dicrotic wave.

Without desiring to lead his readers to overvalue instrumental methods or to obscure the far more vital and practical aspects of clinical technic by a needless elaboration of graphic procedures, the author feels that a brief and simple reference to the chief of certain instrumental methods properly may be attempted.

The Sphygmograph and Sphygmogram.—The sphygmograph, of itself and apart from its combination with other recording apparatus, is an instru-

ment of some slight value in connection with pulse-taking, yielding, in certain directions only, more important information than may be derived from touch and making, if desired, a permanent record, but the relation of time lost to information gained and the large elements of error inherent in the technic make impossible its general adoption in private practice.

Replaced by polygraph.

It has now been almost wholly superseded by the polygraph which has been elaborated to an extraordinary degree and has thrown a flood of light upon the genesis of cardiac irregularities and the pathologic phenomena of the heart cycle.

The arterial pulse tracing represents the systolic and disastolic variations of intra-arterial pressure as registered by the expansion, retraction, shortening and elongation of the elastic artery.

In the case of rigid arteries the vessel itself may also move as a whole, as in the case of tortuous sclerosed temporals or brachials under high pressure or associated with aortic regurgitation. The rhythmic change of size and position of the artery itself is very slight normally, but sufficient to impart to the tambour of a delicate recording instrument the tidal wave of pressure generated by each systole which reflects to a remarkable degree certain important events of the cardiac cycle.

The analysis of all sphygmographic tracings depends upon a thorough understanding of that of the normal pulse.

In this we recognize an anacrotic or ascending limb which at its summit or apex becomes the katacrotic or descending limb, the latter being divided into two chief waves, the higher being *tidal or predicrotic*, the lower the *recoil or dicrotic* wave. Between these two elevations on the descending limb is a hollow representing the aortic closure. *The apex height shows the maximum excursion, the two secondary elevations on the descending limb the reactive contractions of the artery.*

These phases will be readily recognized in the ideal radial tracing shown on Fig. 188.

THE POLYGRAPH. (*Sphygmocardiograph*)

Principles Underlying Its Mechanism.—All of the simpler instruments depend upon the same general principles.

A clockwork mechanism or small motor is employed, by means of which a roll of specially cut paper is passed at any required speed under a stylus or pen attached to a lever operated by delicate tambours, which, through rubber tube connection with the jugular and radial terminals (E and C), transmit and record the impulses. Some form of time marker graduated to two-fifths of a second should also be included, and several of the instruments are capable of registering three tracings of different origin simultaneously.

The spring lever type of recorder may be used for the radial and a special receiver over the heart itself.

The simultaneous records of greatest use and value are those of the venous and carotid pulse and to obtain these the patient is placed in the dorsal

position, with the head slightly flexed, turned to the right* and resting upon a small pillow and should retain the posture for several minutes before record-taking is attempted.

If the venous pulsations are not evident in the jugular fossa because of overdistention of the veins, it may be necessary for the patient to sit up before they can become manifest. In some cases of phlebosclerosis or extreme venous pressure no proper record is obtainable.

Jugular bulb.

Technic.—Ordinarily the venous pulse is evident and the proper receiver is placed over the jugular bulb in the interval between the two heads of the

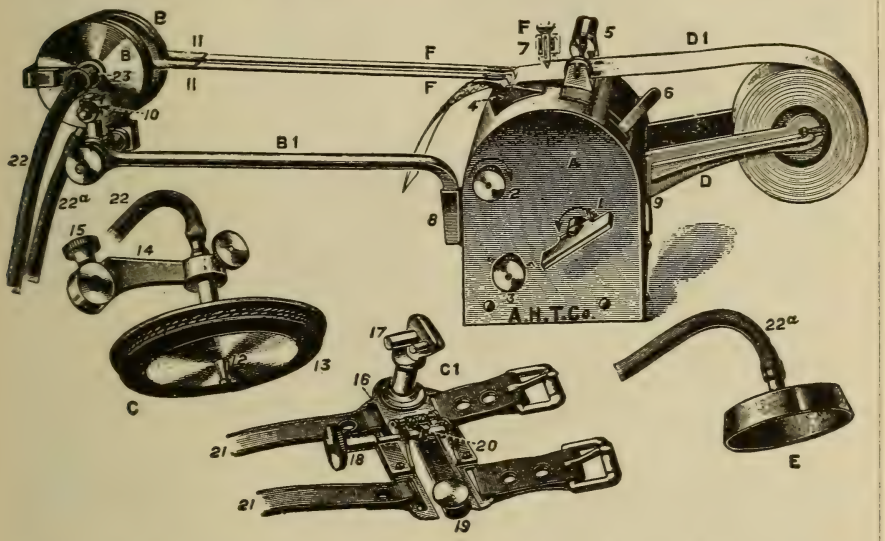


FIG. 188.—Mackenzie ink polygraph. This instrument records two simultaneous tracings only, *i.e.*, radial pulse and one other, such as carotid, jugular, apex beat, etc., the great advantage being the avoidance of smoked paper and the convenience and permanency of the ink tracings which may be continued almost to any length from the long roll of paper supplied with the instrument. The instrument need not be attached to the patient's wrist and by many is preferred because of this feature. The clock work operates at varied and controlled rates, permitting the taking of protracted records at different speeds. The original instrument was invaluable, but Dr. R. Edwin Morris has improved greatly the control of the paper strip, increased the ease and accuracy of tambour attachment and adjustment, and, furthermore, has made possible a delicate, independent and accurate control of the pens, through various ingenious devices.*

sterno-mastoid, just above the right clavicle, and such light pressure is permitted as is just sufficient to secure exact contact and exclude the air.

The proper receiver is then placed firmly over the carotid which is easily palpable just at the inner edge of the sterno-mastoid when the head is in proper position for the venous tracing, *i.e.*, turned to the *right*.

Right auricle tracings.

The tracing of the venous pulse of course represents right auricle effects but tracings representing the left auricle *may* be taken by introducing into

Left auricle tracings.

* See "Modification of Mackenzie Ink Polygraph." R. Edwin Morris, Teaching Fellow, Medical School, University of Minnesota. Jour. A. M. A., June 17, 1916, vol. lxvi, p. 1922.

† Almost all patients instinctively turn the head to the left unless watched and checked.

the esophagus to the distance required, a stomach tube capped by a rubber bag and connected externally to a suitable tambour and writing lever. This method is ingenious but not of much practical use in clinical work by reason of the nausea and retching usually induced.

Crehore registers both heart sounds and the events of the cardiac cycle by means of an ingenious apparatus termed the *micrograph*. It is valuable in cardiac research but too elaborate and time-consuming for clinical use.

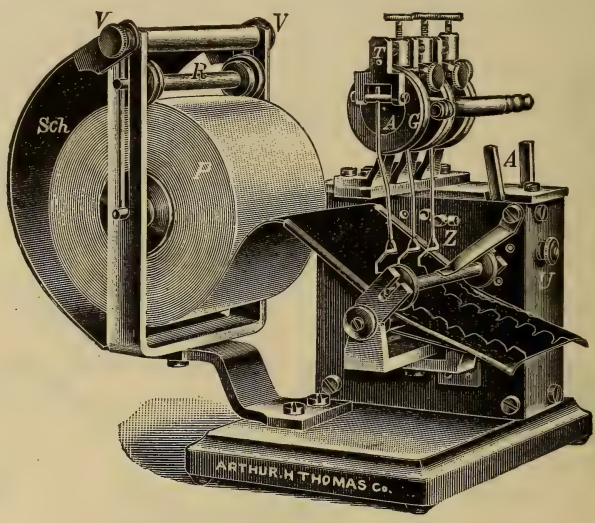


FIG. 190.—Portable polygraph, with continuous roll (20 meters) of smoked paper. This instrument consists of an accurate clock movement imparting two speeds to the paper, a time marker recording in one-fifth seconds and three recording tambours, each of which may receive impulses from three different parts of the body for simultaneous tracing. The instrument thus answers the purpose of a kymograph and is valuable for many purposes because of its extreme portability. The same instrument is furnished with a mercurial syphgmomanometer indicating blood pressure and a cuff writing attachment. In this arrangement of the instrument one of the tambours must of necessity be used for recording the tracings of the brachial pulse under various pressures, while the other two tambours may be used to record any other two tracings such as the radial, jugular or carotid pulse, apex beat, respiratory movements, etc., under an accurately determinable blood pressure as read in a manometer. The pressure applied to the cuff is transmitted to the manometer and, at the same time, to the writing tambour by means of a rubber bulb enclosed within a glass bulb, or Erlanger capsule. (*Courtesy A. H. Thomas Co.*)

There are many other special devices of extreme ingenuity and varying usefulness but none of them demand a full description here.

The Interpretation of Venous Tracings.—A venous tracing remains a thing of mystery until the simple technic of correlating simultaneous records of different forms and sources is explained.

Having fitted the receivers to the proper areas for receiving and recording simultaneously venous and carotid tracings, one first tries the excursions of the levers which should be at least 5 mm. for the venous tracings.

The position of the two recording points initiating the record should then be distinctly indicated upon the paper by exaggerated sweeps of the pens and the record taken by running a series of short tracings. The measurements

necessary to the marking and interpretation are facilitated by the use of short runs. The longer the record the greater the opportunity for error. (See Figs. 192 and 193.)

The purpose of the venous tracing is chiefly to establish the presence and normal relation of the three chief oscillations, *i.e.*, waves "a," "c," and "v."

Their place in the tracing may be definitely established by comparison with the carotid tracing in the light of their known relationship to the carotid curves and, of both, to the phenomena constituting the heart cycle.

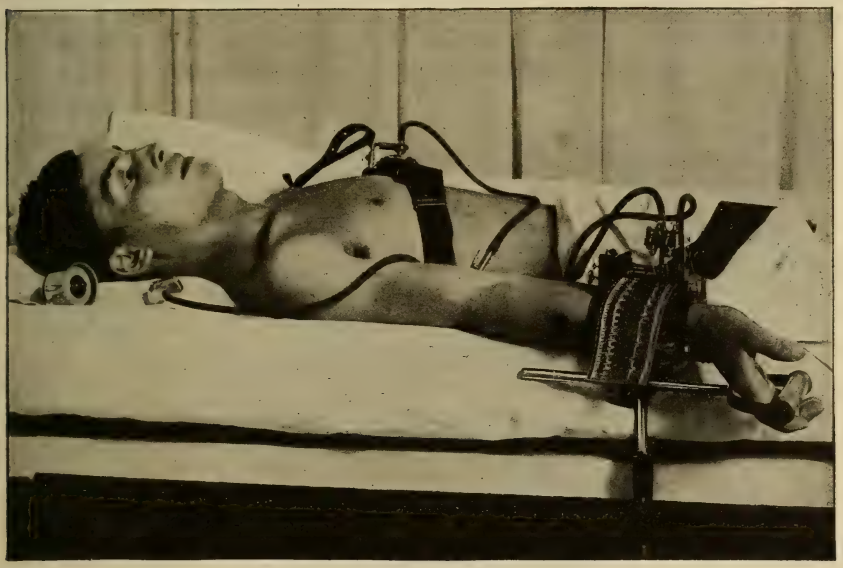


FIG. 191.—Jaquet sphygmocardiograph. In this instrument one tracing must always be that from the radial artery, over which the instrument is fixed in place, either by means of a cuff as in the older forms, or by means of an arm rest. In the single tambour type, therefore, two simultaneous tracings are made in addition to the chronograph record, *i.e.*, the radial pulse from the pelote attached to the instrument and one other tracing through the single tambour, which may be taken from the jugular, carotid, apex beat, respiratory movements, etc. In the double tambour type two tracings may be made simultaneously in addition to that from the radial pulse and the chronograph record. The double tambour type is the most widely used form of Jaquet instrument. Both types are provided with two speeds so that tracings may be greatly magnified by the use of a high speed. The illustration shows the double tambour type with cardiograph attachment in position for recording apex beat and one receiving tambour free to take either jugular or carotid.

Jaquet sphygmotonograph. By means of a tonograph attachment to the double tambour type of sphygmocardiograph, Prof. Jaquet has provided a means of recording the blood pressure from the brachial artery, under increasing and diminishing pressure, simultaneously with the tracing from the radial pulse. The brachial pulse tracing may be read in millimeters of mercury by means of a calibrating table accompanying each instrument.

By such a comparison of the more or less variable venous tracings, often obscured by wholly fortuitous notches or wavelets, with an arterial record of relative stability of character, one may readily locate its important events in most instances.

It is necessary to establish the coincidence of the "c" wave of the venous tracing with the *initial* rise of the primary wave of the carotid tracing; for a vertical drawn from this latter *must intersect* the venous "c"

wave which in its own *initial* rise marks the *onset* of ventricular systole, but just precedes the actual systolic *thrust* in the large arteries.

One has, therefore, only to measure on the carotid tracing the distance from the (previously marked) starting or finishing point of the carotid lever to the starting point of a primary carotid wave, and then lay off exactly that distance from the (previously marked) starting point of the venous pulse lever, and he will cut the "c" wave of the venous tracing, which should commence its rise just before that of the former.

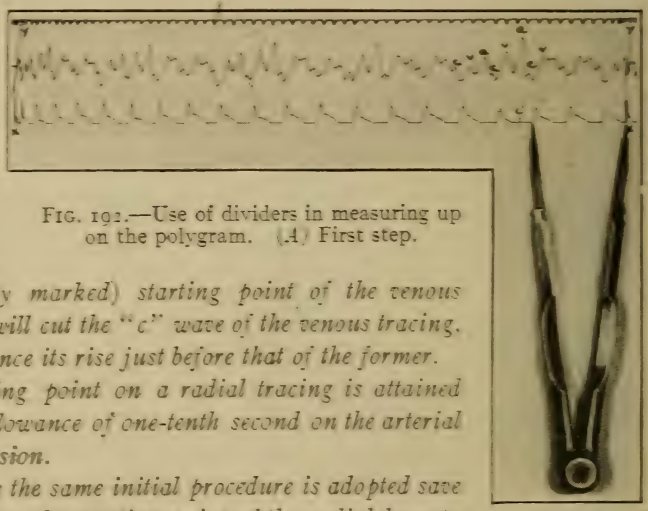


FIG. 192.—Use of dividers in measuring up on the polygram. (*A*) First step.

The corresponding point on a radial tracing is attained by an additional allowance of one-tenth second on the arterial tracing for transmission.

Therefore exactly the same initial procedure is adopted save that the distance from the starting point of the radial lever to the starting point of a radial wave is laid off and moved to the left one-tenth second of distance as per time marker. One has then attained the same point with relation to the venous "c" wave as in the former example. (See Figs. 192 and 193.)

In the author's practice little use is made of the carotid tracing.

The "a" wave summit of initial auricular systole precedes the "c" wave summit by somewhat less than one-fifth of a second. The "v" wave of protodiastole normally is about two-fifths of a second later than "c."

THE AURICULAR VENOUS PULSE.—The instrumental study of the *cardiac venous pulse*, as distinct from the respiratory oscillations, which occur in active breathing, has added greatly to precision in clinical diagnosis and the so-called "*jugular wave*" gives most valuable information as to the action of the auricles with relation to the other events of the cardiac cycle.

The jugular wave.

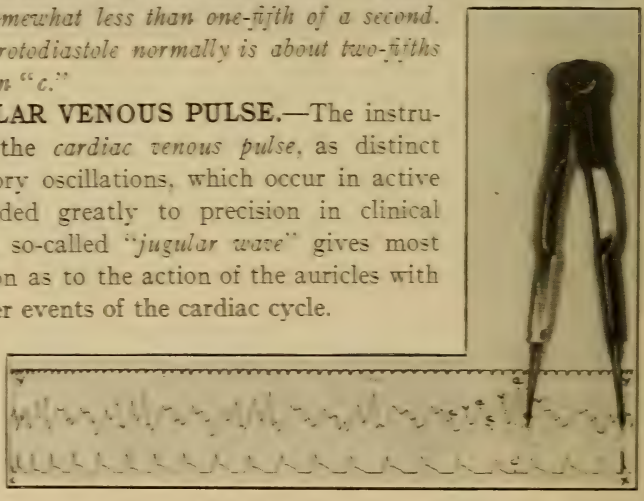


FIG. 193.—Use of dividers in measuring up on the polygram. (*B*) Second step.

General Significance.—The carotid and the venous pulsations are quite distinct in form and characteristics. One reflects primarily the *systolic* contraction of the left inferior, or pumping chamber, the other chiefly represents the effect of *presystolic* contraction of the automatic pumping reservoir of the right heart (right auricle).

Carotid vs. jugular pulse.

Of the two, the venous pulsation is infinitely the more valuable in instrumental readings.

One most easily recognizes the former by its thrust; the latter, by its collapse.

The venous pulse when normal and visible over or near the jugular fossa appears to be a phantom double wave; the first element ("a" wave of the polygram) is presystolic as timed by the heart sounds or apex-beat and *its collapse immediately precedes the commencement of the carotid pulse.*

Presystolic "a" wave.

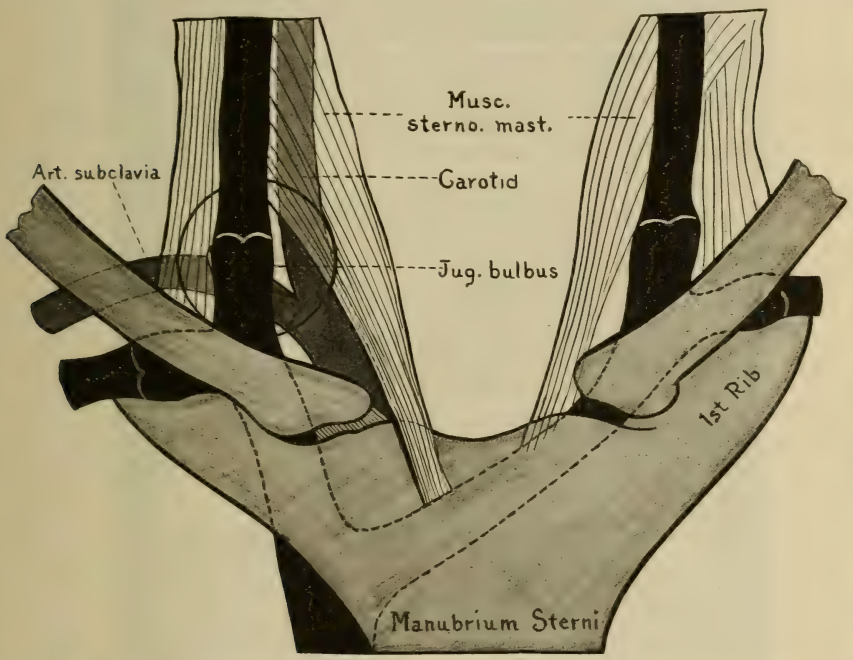


FIG. 194.—Site of election for the application of the receiver used for jugular tracings is indicated by the circle, and overlies the jugular bulb. It will be noted that the veins are shown in black wherever accessible. (*After Keith, modified.*)

The second element, "c" wave, or secondary rise, immediately follows it, just preceding the thrust of the carotid beat.

The systoli "c" wave.

Events of the Venous Pulse Cycle.—This double pulsation so often visible, in the jugular fossa, higher along the course of the vein, or, in the supraclavicular fossa over the *external* jugular, is found to be triple actually, when recorded by the polygraph.

A triple pulsation.

The first ("a" *auricular*) (*atrial*) *wave* is, as stated, unquestionably the result or the auricular presystolic contraction, being due either to a return wave to the jugular bulb or to the sudden check interposed by the auricular systole to the venous current. It is absent, therefore, in auricular fibrillation, or auricular paralysis from excessive persistent distention.

Genesis of "a" wave.

Nodal extrasystoles occur when the impulse to contraction arises (probably) in the auriculo-ventricular node, not in the sino-auricular node, its proper normal point of origin.

Presphygmic wave.

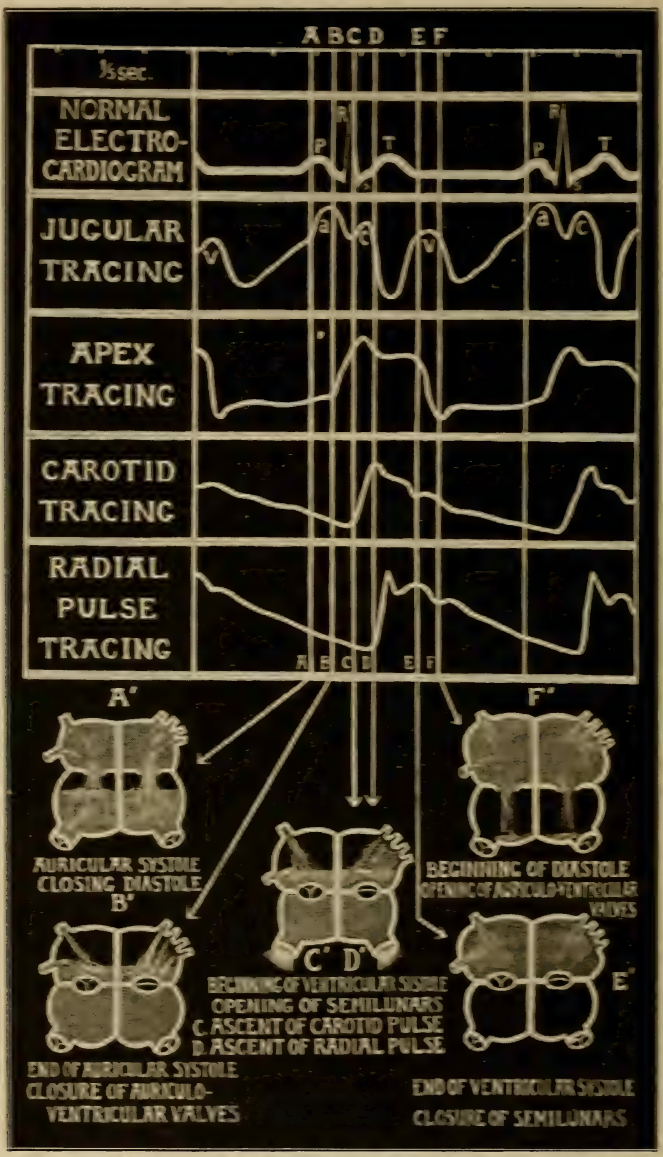


FIG. 105.—Time relationships of the phases of the electrocardiogram, and the jugular, apex, carotid, and radial tracings. At the bottom are shown schematically the corresponding phases of the heart cycle. Each of the vertical lines A, B, C, D, E, and F, is extended to indicate the corresponding heart-cycle phase. (Time marker intervals $\frac{1}{5}$ sec.)

A′ = Beginning of auricular systole (auricular presystolic contraction = ascent of "a" wave in jugular tracing = rise of P of electrocardiogram.

B′ = Closure of mitral and tricuspid valves, terminating auricular systole and ending ventricular diastole. Aortic and pulmonary valves still closed, but subjected to the increased intraventricular pressure of increasing tonus and initial contraction (isotonic or presphygmic period) = ascent of apex, crest of "a" wave and end of auricular electrocardiographic complex "P."

C′ = Opening of semilunar valves, initiation of full ventricular systole, collapse of "a" wave, ascent of "c" wave and carotid pulse, beginning of electrocardiographic systolic complex of "R," "S," "T" and, one-tenth second later, the ascent of the radial pulse.

D′ = Termination of systole by closure of aortic and pulmonary valves (mitral and tricuspid not yet open).

E′ and F′ = respectively the end of ventricular systole and the beginning of the actual diastolic period. The "V" summit of the polygram is divisible into two corresponding phases namely, V′ and V″.

Intervals B–C and E–F are slightly exaggerated.

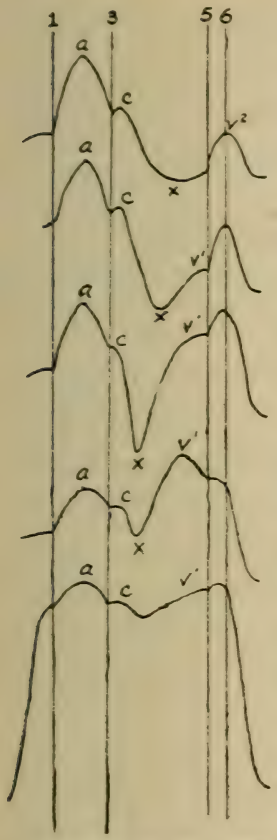


FIG. 196.—Diagram drawn from actual tracings showing the variations in records of the auricular type of venous pulse. The upper phlebogram is normal. The lowest shows the effect of free tricuspid regurgitation. Note the gradual increase of wave "v" at expense of the depression "x." (1) The beginning of auricular systole. (3) The appearance of the pulse in the carotid. (5) The closure of the semilunar valves. (6) The opening of the tricuspid valves. (*After John Hay.*)

As a result the ventricular contraction may practically coincide with or actually precede that of the auricles and in consequence a large wave embracing both "a" and "c" may result.

Such concurrent or fused waves, a-c, merely show on a tracing as interruptions and variants of the dominant rhythms followed by the "compensatory pause."

Presphygmic wave.

The second wave "c" is now known to be a venous wave independent of the carotid beat, starting probably early in the presphygmic period,* present even though the carotid be ligated and aorta held away, and instrumentally demonstrable in both esophageal and hepatic venous tracings. Indeed its form in the tracing, *as obtained with sufficiently light pressure*, is entirely different from that of the arterial wave.

Genesis of "c" wave.

Its beginning probably represents the time of the closure of the tricuspid valve and it precedes the actual carotid pulse by the presphygmic interval, *i.e.*, the time elapsing between such tricuspid closure and the forcible systolic opening of the pulmonary valve.†

The wave of valvular closure.

A study of this wave in extrasystolic frustrated contractions, *i.e.*, such hasty, weak, and premature contractions as are incapable of opening the aortic and pulmonary valves though the tricuspid and mitral may close, shows the truth of this hypothesis.

Proto-diastolic "v" wave.

The third or "v" wave is practically synchronous with the second sound of the heart, *i.e.*, with closure of the pulmonary and aortic valves, opening of the tricuspid and mitral and the beginning of diastole.‡

Between the "a" and the "v" waves is, of course, a drop in the tracing due to the emptying of the veins which accompanies auricular diastole which may be denominated the "x" depression.

After the height of auricular systole is attained a drop in jugular venous

* It is probable, however, as von Jagic says, *that in practice*, the carotid pulse is often included in the tracing of this second wave through technical error.

† Minute interval of time (0.07 to 0.085 sec.) elapsing between the closure of the mitral and tricuspid valves and the opening of the aortic and pulmonary exits in systole. It is more properly termed the ventricular isometric period.

‡ A second "v" wave (v″) is recognizable in polygraphic tracings, but as yet has been assigned no clinical significance.

pressure results which is reflected on the polygram by the "x" depression interrupted by the "c" wave.

Any encroachment upon the "x" depression by the "v" wave indicates a tendency to venous stasis and more rapid and perhaps excessive filling of the auricles.

Any of the various pulmonary and cardiac conditions embarrassing the right heart and resulting in venous stasis would tend to cause a widening of the "v" wave at the expense of the "x" depression or "phase."

Summary.—*The "a" element of the jugular wave corresponds to the beginning of auricular systole in its rise; to the close of auricular systole in its fall and immediately precedes the first heart sound.*

The "c" element of the jugular wave in its beginning marks the closure of the tricuspid valve and the initiation of ventricular systole. It coincides with the first sound of the heart and just anticipates the carotid thrust.

The "v" wave crest very nearly marks the beginning of diastole and coincides with the opening of the tricuspid and mitral valves, the closure of the pulmonary and aortic and the second sound of the heart. See Fig. 194.

Registration of the Venous Waves.—It is evident that if the venous pulse events and those of the carotid or radial are registered simultaneously in graphic form, one may draw conclusions of great value with regard to irregularities and conduction, as well as *heart strength.* Lack of balance between the venous and arterial circulations and variations in heart strength are often clearly shown.

Scope of Methods.—Such registration is now readily obtained by means of any one of several polygraphs and, in fact, the heart sounds and the direct action of the auricle as well as the electric "action currents" of the heart, can also be graphically represented by means of special instruments of precision.

Decided Limitations.—Special training is necessary to *accurate* results with such instruments and this fact and the time consumed in the necessary procedures will confine such work largely to the research laboratory, teaching hospital and, possibly, the consulting room of the specialist.

MARKING AND INTERPRETATION OF THE POLYGRAM

Systematic Procedure Indispensable.—It is absolutely necessary to proceed in a systematic and orderly manner in the marking and interpretation of a polygram. (See Figs. 192, 193, p. 502.)

Technic of Marking.—The only instrument necessary to the marking is a pair of needle-pointed dividers. Inasmuch as these are permanent records, mark the name, date, and special circumstances surrounding the taking of the record, *i.e.*, whether following exercise, special medication, accident or injury, association with painful seizures, syncopal attacks, etc. Also mark the radial and jugular tracings, indicating the side used.

It matters little what particular system is adopted. The one in use in the author's clinic is outlined below and has proven fairly satisfactory.

1. *Determine the "c" wave* from the radial record by measuring the distance from the beginning point to the first point of rise of any radial thrust. Lay off this same distance on the venous record from *its* beginning point. That wave occurring $\frac{1}{10}$ of a second earlier is the venous "c" wave of that corresponding heart cycle. Mark the "c" wave in at least six consecutive heart cycles, in the same manner. (See page 502 for details.)

2. *Determine the "a" wave.* This is the definite wave preceding the "c" wave normally by $\frac{1}{5}$ of a second or less.

3. *Determine the "v" waves.* These are normally the only remaining waves of importance and lie approximately midway between the "c" wave and the "a" wave of the following heart cycle.

4. *Determine the nature of extrasystoles* if they occur. (Detailed description to be given later.)

5. *If the "a" wave* is absent look first for the presence of fibrillation and, if found, label the fibrillatory waves "f," "f," "f."

6. *Determine the rhythm.* With the presence of extrasystoles or fibrillation the *arrhythmia* itself is apparent at a glance. To determine sinus arrhythmia measure the distance between the tops of any two radial thrusts and make comparison with several other similar intervals. A variation in these intervals, in a record otherwise normal, indicates *sinus arrhythmia* in its varying degrees. The pulse rate is markedly affected by respiration, being increased by full inspiration, and decreased by full expiration. It is wholly abolished by vigorous exercise.

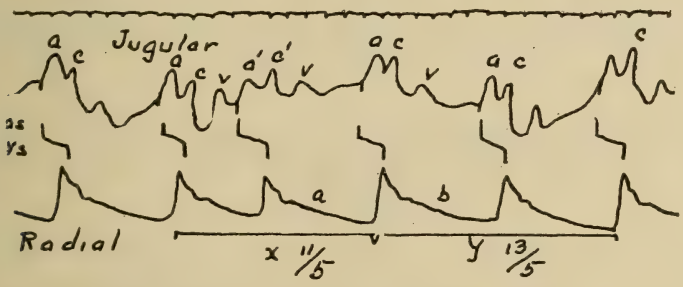


FIG. 197.—This record shows an extrasystole of auricular origin at *a'–c'*. There is no compensatory pause. In this patient the conduction of the premature stimulus is unduly delayed, the *a'–c'* interval being almost two-fifths of a second. The diagram shows the relative incidence of the systoles of the auricles and ventricles. (*After John Hay.*)

7. *Determine the "a–c" interval.* Lay off the distance from the beginning of the "a" wave to the beginning of the "c" wave. Compare this with the time-record. The normal does not exceed 0.20 sec.

8. *Determine the rate.* The time is recorded by the "marker" in fifths of a second. Fifteen spaces on the time-record is equivalent therefore to 3 seconds. From the top of one of the radial thrusts lay off a 3-second interval (15 spaces on the time-record) and count the radial thrusts during that period; then lay off another consecutive distance of 3 seconds and continue the count. The result is the number of heart cycles in a 6-second

interval as recorded by the time-marker. Multiplying this by 10 gives the rate per minute.

Special Conditions.—*Extrasystoles.*—Extrasystoles may be (1) auricular, (2) ventricular, or (3) nodal. Sinus arrhythmia has already been dealt with.

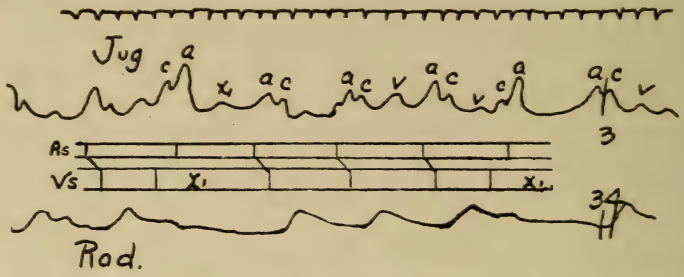


FIG. 198.—In this record an extrasystole, originating in the ventricle has occurred before the normal systole of the auricle a_1. Note that in this case there is no ventricular systole resulting from the auricular systole a_1, the a–v bundle apparently refused to conduct the stimulus. Note absence of "v" wave immediately after extrasystole.

The downstrokes in the upper space "As" of the diagram indicate auricular waves "a" in the jugular pulse; the downstrokes in the lower space "vs" indicate the carotid waves "c" in the jugular tracing, the slanting lines connecting them give the "a–c" interval. (*After John Hay.*)

1. *Auricular extrasystoles* show a premature contraction of the *whole* heart indicated on the record by an abnormally early occurrence in diastole

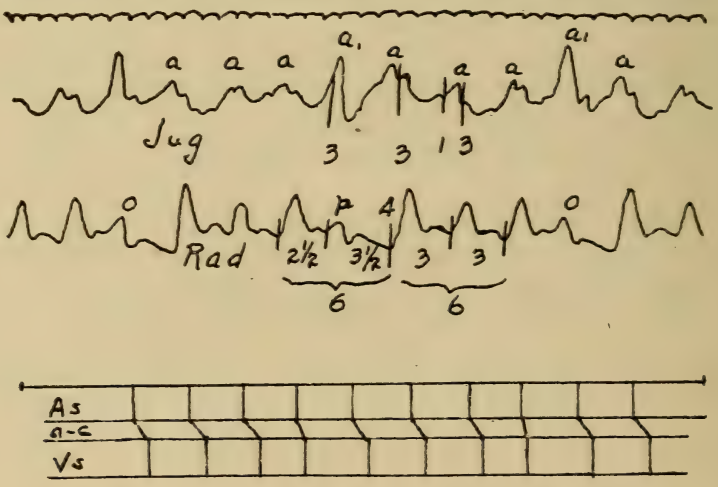


FIG. 199.—Simultaneous records of radial and jugular pulses. Three extrasystoles are recorded at "o," "p" and "o" in the radial tracing. The rhythm of the auricle is regular but the ventricle contracts prematurely as is obvious from the radial tracing. The post-extrasystolic pause is fully compensatory and lasts three and-a-half fifths of a second. "a" = auricular wave. "a_1" = the auricular wave associated with the extrasystole. The diagram represents the events in the latter two-thirds of the above record. (*After John Hay.*)

of the "a" and "c" waves, with a corresponding early appearance of the radial thrust. This is followed by a lengthened diastolic interval representing *less than two heart cycles* in duration as measured on the radial record.

2. *Ventricular extrasystoles* are premature contractions originating in the ventricle and show (*a*) unaltered auricular rhythm, (*b*) a premature systole

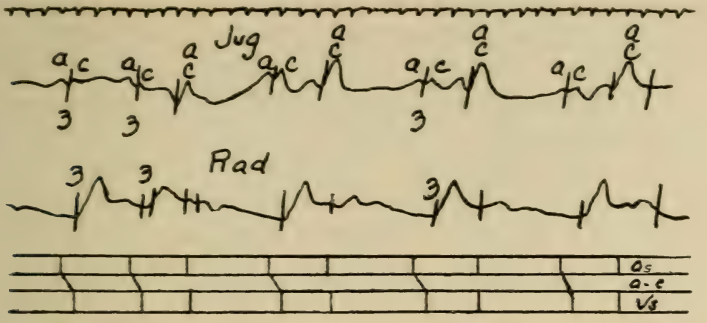


FIG. 200.—Simultaneous records of the jugular and radial pulses. Shows four nodal extrasystoles. The diagram gives the sequence of events as shown in the jugular tracing. Note (1) that both auricle and ventricle contract prematurely; (2) that there is a full compensatory pause; (3) that the wave "v" is greatly diminished after the extrasystoles. (*After John Hay.*)

of the ventricles either preceding that of the auricles or simultaneous with it, and (*c*) complete compensatory pause, *i.e., equal to two heart cycles.*

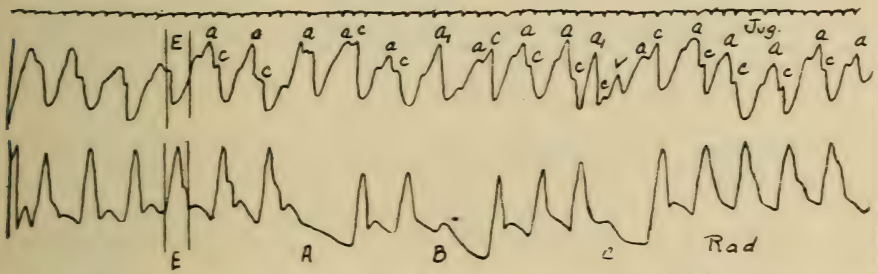


FIG. 201.—Shows three irregular periods due to extrasystoles. At "A" a nodal extra systole; during "B" a ventricular extrasystole; and at "C" an auricular extrasysto le (*After Mackenzie.*)

A record in this case would show the *regular occurrence of "a" waves* and an abnormally early "c" wave appearing either before or coincidently

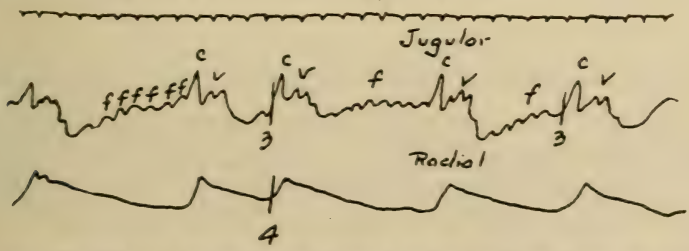


FIG. 202.—Radial and jugular tracing from a patient suffering from auricular fibrillation. Note (1) the absence of "a" waves in jugular tracing; (2) the presence of fibrillatory waves (fff); (3) the lack of rhythm of radial thrusts. The slow radial rate is due to digitalis. (*After John Hay.*)

with the "a" wave. In the latter case the "c" wave cannot be differentiated from the "a" but its location is determined by the usual method.

It is to be noted that in ventricular extrasystoles the "a" waves do not occur prematurely and the so-called "compensatory pause" following *is equal to two complete heart cycles.*

NOTE.—Extrasystoles of *ventricular* origin in rare instances occur as *interpolated* beats between two normally spaced ventricular contractions each of which represents a response to normal auricular stimulation.

3. *Nodal extrasystole* is indicated by the premature and *simultaneous contraction* of both auricles and ventricles. This is followed by a compensatory pause equal to two full heart cycles (called complete compensatory pause). This is indicated on the venous record by an exaggerated wave resulting from and in time measurement representing the simultaneous occurrence and merging of the "a" and "c" waves.

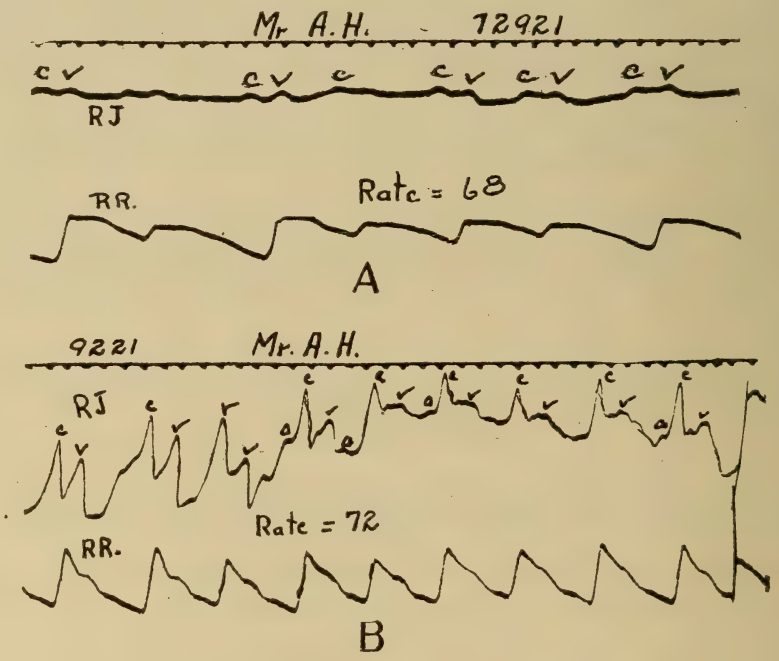


FIG. 203.—Mr. A. H.

(A) Polygraphic record taken in case of fibrillation in a young adult at beginning of hospital regime.

(B) Record in same case 54 days later showing practically normal rhythm at radial, marked improvement in myocardial tone and strength as indicated by the height of the radial. The general absence of "a" waves in the jugular record shows that fibrillation still exists, but an occasional wave in the normal position of the "a" wave is found. R.R. = right radial. R.J. = right jugular.

Fibrillation.—Fibrillation is characterized by a total irregularity in rate, force and rhythm of the radial pulse, the absence of "a" waves in the venous tracings, and the occurrence of multiple coarse or fine undulations (fibrillatory waves). Label fibrillatory waves "f," "f," etc.

In this condition many impulses are so weak as not to be registered on

the radial record and even less would be detected by palpation of pulse. The more rapid the heart the greater the "pulse deficit." (See page 551.)

Block.—Block may be (1) complete or (2) partial. In *complete block* there is complete dissociation between the "a" and "c" waves together with a slow rate in the radial record.

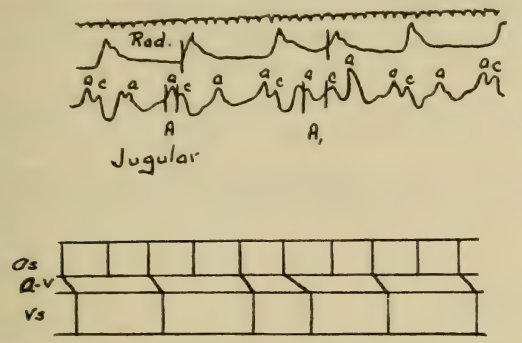


FIG. 204.—Increased depression of conductivity producing an arrhythmia. The "a" wave occurs perfectly regularly. Note that the "a–v" bundle requires th e whole time of two auricular systoles to recover its conducting power. When this length of time is curtailed as in the middle of the record the "a–c" interval A_1 is at once lengthened.

The diagram represents the condition present in the above record. The downstrokes in the upper compartment *As* representing auricular systoles, occur with perfect regularity, with one exception every other systole is blocked. Note the lengthening of the "a–c" interval when two consecutive stimuli pass down the "a–v" bundle. The downstrokes in the compartment *Vs* represent systoles of the ventircles. (*After John Hay.*)

In this condition the complete block existing in the conducting fibres compels the ventricles to adopt their own slow, deliberate rhythm, causing the occurrence in the venous record of correspondingly few "c" waves. The conduction through the auricles being unimpeded, they respond freely to the

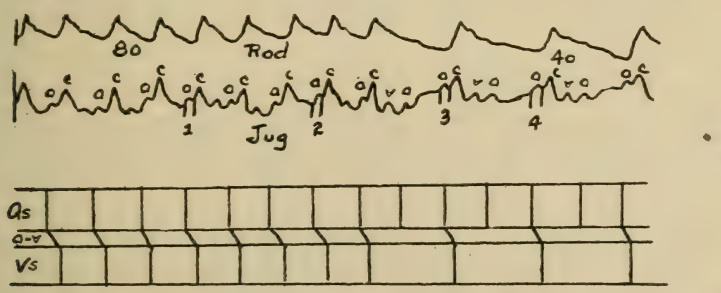


FIG. 205.—The record above shows a sudden halving of the pulse frequency. There is no depression of conductivity. The "a–c" interval is normal throughout. Depression of excitability is shown by the refusal of the ventricle to respond to a stimulus normally conducted. The diagram represents the events shown in the jugular record. (*After John Hay.*)

stimuli arising in the pacemaker (sino-auricular node). The result is a preponderance of "a" waves over "c" waves in the venous record varying in degree with the ventricular rate. An auricular rate of 70 might accompany a ventricular rate of 40, for example. The lowest ventricular rate

reported to date is one beat every 57 seconds, observed shortly before death.

In *partial block* some of the auricular contractions cause corresponding ventricular contractions. There is found a predominance of "a" waves again, but every so often at definite intervals, there is found an "a" wave which has its normal position in relation to the "c" wave. For example, in 2:1 block every other "a" wave is in its proper relation to the "c" wave with the extra "a" wave midway between these two. Here the auricles are

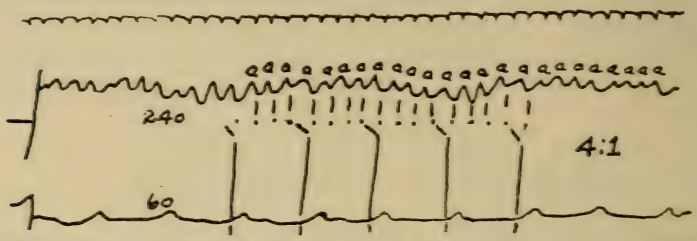


FIG. 206.—Tracing showing auricular flutter "a" wave due to systole of auricle. Auricular rate = 240. Ventricular rate = 60. *As* : *vs* :: 4 : 1. (*After John Hay.*)

beating twice as fast as the ventricles. In 3:1 block there are two extra "a" waves between two normally placed "a" waves, all of the "a" waves being the same distance apart.

Auricular Flutter.—This is a condition of extremely rapid, rhythmic auricular contractions associated usually with a partial block, and occasionally with alternation of the pulse. An associated block may give rise to a 2:1, 3:1, 4:1, or in rare instances, even 6:1 ratio between the recorded auricular

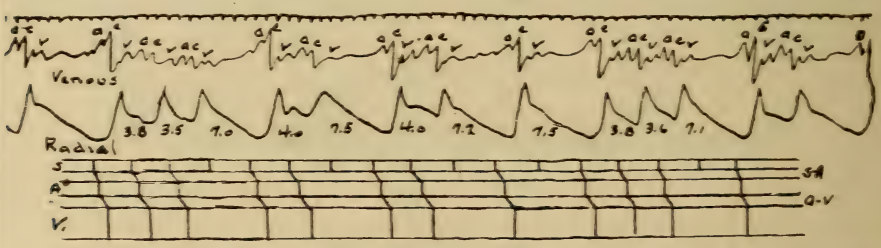


FIG. 207.—Polygraphic curve showing a sinus irregularity during a period of suspended respiration. The arterial curve resembles those found in partial A-V block; the venous curve shows that the auricle participates in the irregularity. The diagram shows the events of the heart upon the hypothesis of "*sino-auricular*" *block*. (See "Sino-auricular Block"—in Electrocardiographic Section.) (*After Thomas Lewis.*)

and ventricular waves. This condition is accurately determinable only by instrumental methods. (See page 555.)

Paroxysmal Tachycardia.—This is a condition characterized by a *sudden* outburst of rapid action of the whole heart and its *abrupt* termination after a duration of minutes, hours or days. It is usually, if not always, initiated by extrasystoles of auricular origin. Rarely, it is said that these cases are ventricular in origin. (See page 559.)

Rhythm.—A record is either rhythmic or arrhythmic. Arrhythmic records are by far the more common and the larger per cent. of these are due to the simple, and relatively unimportant, sinus arrhythmia. Of the arrhythmias due to extrasystoles, the ventricular type constitute the largest per cent.

The Venous Record.—The examination and interpretation of the venous record is very important for it reveals some of the earliest signs of myocardial insufficiency.

General Form and Character.—Normally the venous record is small and narrow, with its various components small but clear-cut and distinct. A wide exaggerated record with large, disproportioned components is significant of an abnormal condition of the cardiovascular system. In order to determine the condition present, the "a," "c" and "v" waves must each be examined as to form, size, and general characteristics.

"*a*" *Wave.*—Normally the "a" wave is a small rounded wave, smaller than the "c" wave and just preceding it. Its absence suggests fibrillation. Note whether the "a" wave is larger than the "c" wave. If not larger all the time, is it larger than the "c" wave part of the time? Is it a wide, broad wave?

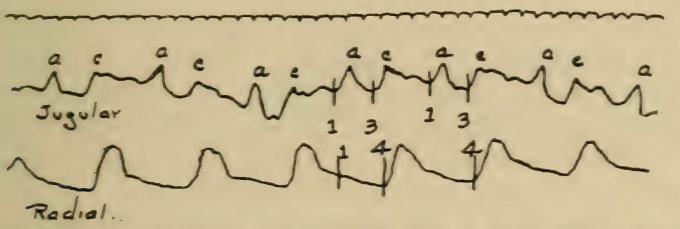


FIG. 208.—Simultaneous records of the jugular and radial pulses. In the jugular pulse there is a marked lengthening in the "a–c" interval. The distance between the downstrokes 1 and 3 represents more than two-fifths of a second. This indicates delay in the passage of the stimulus from the right auricle to the ventricle. (*After Herrirgham.*)

"*a–c*" *Interval.*—Normally the "a–c" interval is 0.20 sec. as measured by the polygraph. Any interval greater than 0.20 sec. indicates delayed conduction. Among the conditions found to be associated with delayed conduction are the following: acute and chronic myocarditis, myocardial degeneration, decided or profound toxemia, myocardial insufficiencies resulting from overexertion, acute infectious diseases, etc.

The determination of the "a–c" interval is important at all times but especially so when digitalis is being administered, for the sudden development of delayed conduction under this circumstance usually means overdigitalization.

The author's experience with the polygraph and electrocardiograph during the past two years have served to convince him that too little stress is laid upon the importance of minor degrees of delayed conduction such as do not reach the grade of even partial block, and believes that it constitutes a finding of definite clinical significance and one that demands

in many instances the most careful and discriminating therapeutic management. This is particularly true of the cases arising in the chronic heart lesions as contrasted with those in which some active and potent infection is demonstrable.

In cases of actual heart block, contrary to the usual belief, digitalis may act most favorably in ameliorating the condition provided that other signs of myocardial insufficiency are present in the case.

In administering digitalis, however, the author exercises the greatest care not to produce the so-called full physiologic effect, finding that in many instances when this is done a definite weakening of the myocardium becomes manifest. In cases of block especial care is needed, but in his opinion the rule applies to nearly all decompensated cases. The object of digitalis administration in such cases is to aid in removing whatever insufficiency may be present inasmuch as this seems to be a decided factor both in persisting delayed conduction, partial block, and complete block alike.

"*c*" *Wave.*—The "c" wave is usually called the carotid wave. It is a venous wave however quite independent of the carotid heat which it precedes by the "presphygmic interval" (see page 505). Normally it is larger than the "a" wave and is followed by a fairly sharp drop and then by the "v" waves.

"*v*" *Wave.*—There is usually one, and sometimes there are two, "v" waves. An exaggerated "v"–1 suggests a tricuspid insufficiency.

The Radial Record.—In general the height of the radial thrusts indicates the condition of the myocardium. A good myocardium gives a good radial thrust. Before the interpretation of the height of the radial thrust can be made, however, one must know that the instrument was working properly and that the adjustment to the radial artery was accurately made. This is especially important in cases of weak myocardial strength which frequently give a practically flat radial record.

One exception to the above statement is in the case of the "drop" type of heart, for, even with a good myocardium, this heart gives a relatively small radial thrust.

Normal tracings show a nearly vertical upstroke of moderate amplitude, moderately sharp apex and a gradual descent, the tidal wave being small, the dicrotic wave low but well marked.

Abnormal Tracings.—(*a*) *Broad Apex.* This indicates sustained tension and a strongly acting deliberate heart, as in interstitial nephritis, aortic stenosis and aneurysm, or, merely too great pressure on the artery by the pad.

(*b*) *An unduly long sharp apex* indicates low or unsustained tension, and is especially marked in aortic regurgitation.

(*c*) *A short upstroke* indicates small volume, as in mitral regurgitation, aneurysm, aortic stenosis, and arteriosclerosis.

(*d*) *A long upstroke* means free peripheral circulation, sharp systole, or, in general, unsustained pressure. Aortic regurgitation furnishes a typical example.

(*e*) *Excessive obliquity of the upstrokes* points to slow filling of the artery and is especially noticed in aortic stenosis, mitral stenosis or regurgitation, arteriosclerosis, aneurysm or a weakened myocardium.

(*f*) *A vertical upstroke* indicates large blood volumes and quick systole whether strong or weak and is exemplified by aortic regurgitation.

(*g*) *A marked tidal wave* suggests high tension or actual obstruction as in aortic stenosis or in arteriosclerosis with a strong cardiac impulse.

(*h*) *A diminished tidal wave* indicates cardiac weakness, or strength with relaxed peripheral circulation, as in mitral or aortic regurgitation.

(*i*) *A marked dicrotic wave* points to low tension or to high tension with cardiac weakness. It is best marked in the pulse of typhoid fever.

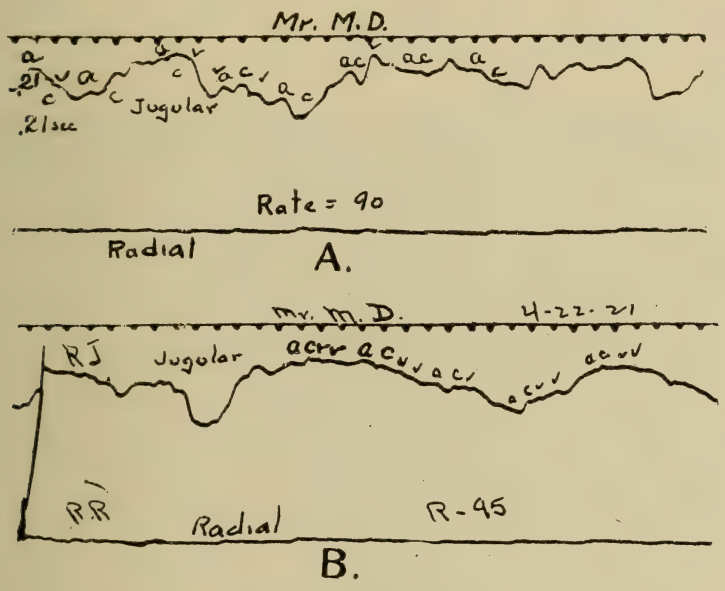


FIG. 209.—Mr. M. D.

A. Case of extreme terminal decompensation of sudden onset. Note flattening of radial record.

B. Record made 3 days before death and just before onset of terminal coma. Flattening of radial tracing still greater than in A. Undulations barely perceptible.

(*j*) *Lessened dicrotic wave.* Aside from aortic regurgitation this points to an obstructed circulation with a strong systole, *i.e.*, aneurysm, arteriosclerosis and aortic stenosis. Aside from these points, *irregularities in the line of descent* are noted in mitral lesions; *irregularities of the base line* corresponding to respiration, in dyspnea or any form of irregular forcible breathing, *and any intermittency or irregularity which can be reflected in the pulse is usually made clearly evident by the tracings.*

In the pulsus celer type the radial has a rapid rise and fall which usually goes below the base line. This type of pulse is due to a weakening of the aortic ring and quickly expanding and relaxing vessels.

The "compensated mitral type" of pulse is a wide, low, flat-topped

type of radial thrust. This is the result of the powerful deliberate action of the left ventricle which has greatly increased its muscle mass. The radial thrust is relatively low and flat-topped for, because of the regurgitation, it can attain only a certain height, but it maintains this height longer than usual as the increase in the muscle mass gives the left ventricle a longer sustained action than it has normally.

The pulsus alternans is the type of record in which we find high radial thrusts alternating with lower ones. This is a very important prognostic sign. (See page 561.)

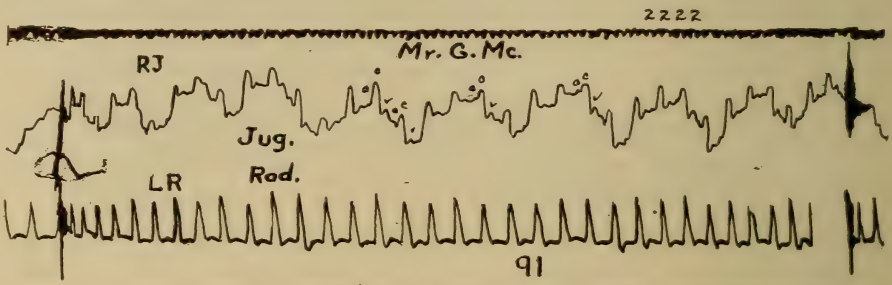


FIG. 210.—Mr. G. McC. Polygraphic record in a case of typical, definitely insufficient, "drop" heart of a 36-year old man. The high, unsustained radial (*pulsus celer* type), a common finding in this type of heart, is shown in the record. See *x*-ray, Fig. 000, page 000, and Electrocardiographic record, Fig. 234, page 540.

By vagus hypertonus (not hypertension) we mean a radial record with a radial thrust in which the fall is more gradual than usual. It is supposedly due to increased resistance in the vessels as a result of overaction of the vagus. This is not hypertension.

Valuable information from use of polygraph.

It must be evident that the polygraph gives much very valuable information about the heart and the circulation. Much of this information cannot be obtained by other than instrumental methods. When used routinely in the first examination of a patient, side by side with the ordinary clinical

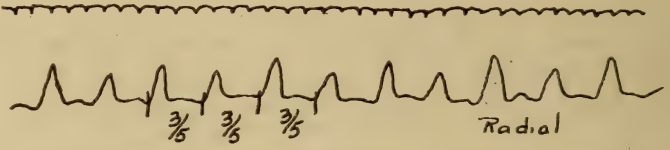


FIG. 211.—Pulsus Alternans. Every alternate wave is small, each pulse period is of full duration. (*After John Hay.*)

methods, it is a great aid in diagnosis. Its value does not stop here, however, for if employed frequently thereafter it gives much valuable information as to the progress of the case and the results of medication. It can be of inestimable value in hospital and house cases where the elaborate and efficient office instruments are not available. When used in this way it very frequently gives the first hint that the patient is not doing as well as he is believed to be doing and gives the opportunity of changing the existing regime of the patient before any real harm is done, hours or even days before the ordinary clinical methods reveal the threatening trouble.

THE ELECTROCARDIOGRAPH

Basic Principles.—Whenever a portion of muscular tissue takes on activity it becomes electrically negative with relation to the resting portion.

The differences of electric potential thus created (electromotive force) can be measured by the "string galvanometer" when one electrode overlies the active portion and the other the passive area.

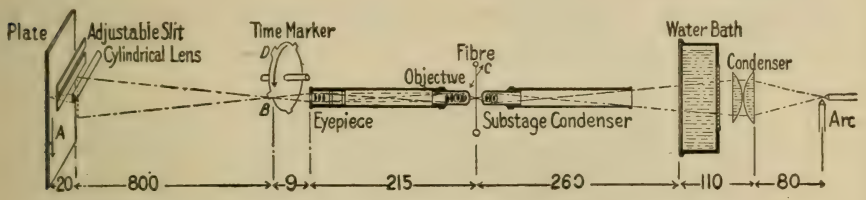


FIG. 212.—Essential parts of electrocardiograph, as shown below.

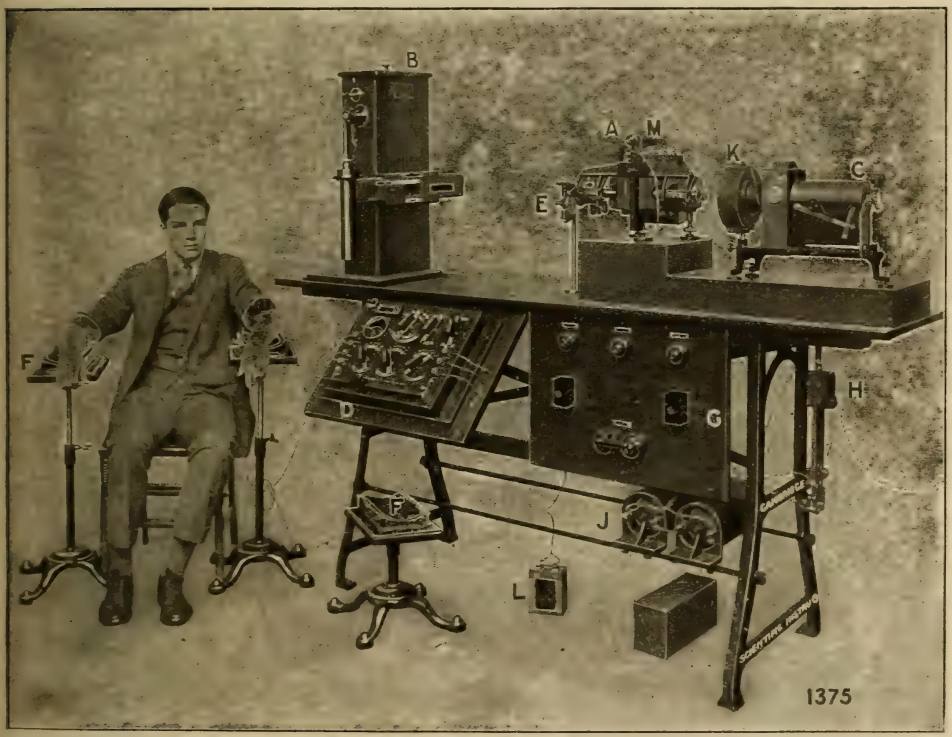


FIG. 213.—Cambridge electrocardiograph. Simpler and less expensive instruments are much to be desired and those shown are undoubtedly satisfactory. (*Courtesy of the Taylor Instrument Co., Rochester, N. Y.*)

In the heart we deal with an aggregation of muscular units which become intermittently active, not as a whole, but in a coördinated sequence which especially sets apart in greater or less degree its different chambers and the threshold formed by the junctional tissues of primary conduction.

There is created a recurrent flow and ebb of electric potential from one portion to another, each beat of the heart representing a tidal cycle of "measurable action current" which is described technically as a "*diphasic*" current.

Waller first obtained registrations of such human heart currents in 1887, using a capillary electrometer and in 1903 Einthoven devised the string galvanometer which forms the basis of the modern electrocardiograph.

Essential Features.—As now made, the electrocardiograph is merely a galvanometer to which a photomicrographic projection apparatus with an arc lamp is attached, which registers the deflection shadows of an inter-

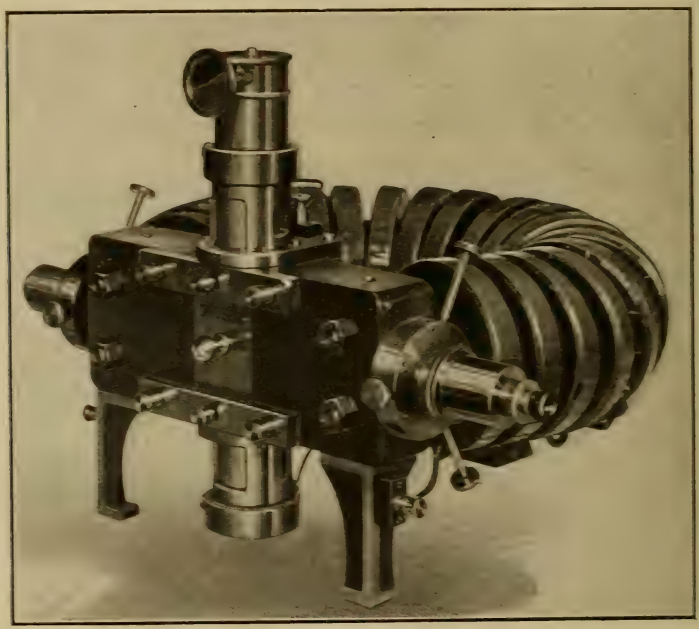


FIG. 214.—New simplified and relatively inexpensive electrocardiograph manufactured by C. F. Hindle, New York City. This is the instrument used by the author and is most satisfactory. Mr. Hindle now puts out a still simpler and less expensive instrument.

posed current-conducting filament, of platinum or of silvered quartz, upon a screen opposed to a photographic film moving at any required rate.

Sensitivity and Responsiveness.—To secure sensitivity and quickness of movement the filament which conducts the action currents of the heart must be extremely fine (0.002 to 0.003 mm.) and the strength of the magnetic field in which it lies must be maximal.

Any electric current generates a magnetic field acting at right angles to the course of the current and exerting attraction and repulsion upon any second adjacent magnetic field. If, therefore, a conducting filament is interposed as in this instance and made to conduct a current it will move at right angles to itself and to the magnetic field, reversing its direction with reversed flow of current.

The movements of the conducting filament are directly proportional to

the strength of the magnetic field and the current passing through the string itself, and, inversely, to the tension of the latter.

Extracardial Sources of Deflection.—One must obtain the heart-action current separate and apart from any others, such as skeletal muscle movements and the skin current, generated in the body of the patient who, therefore, must be absolutely still, muscularly relaxed, and breathing quietly.

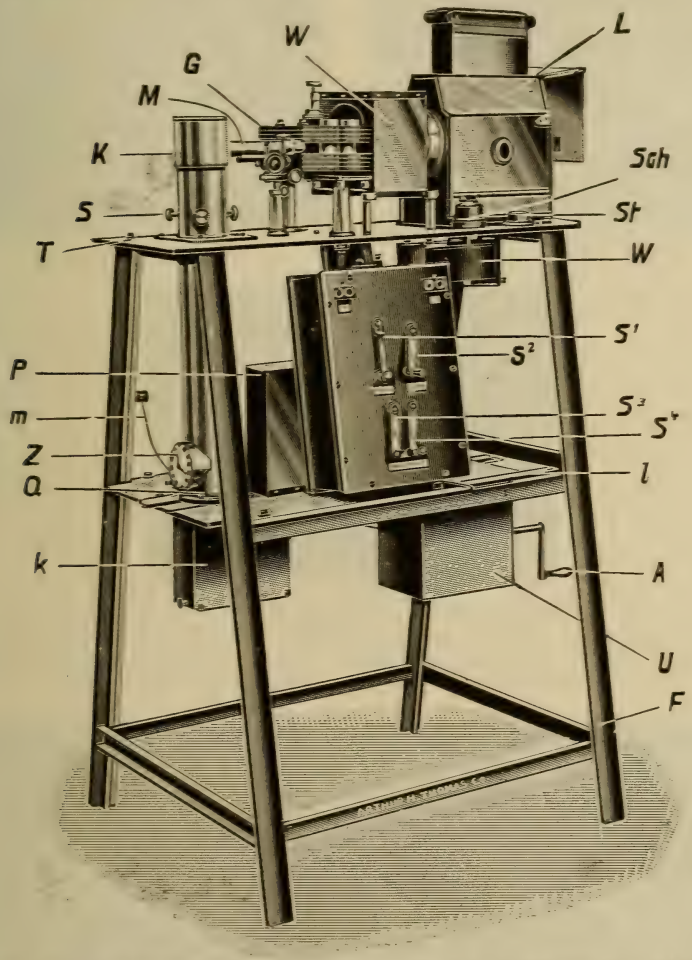


FIG. 215.—New simplified electrocardiograph. (*A. H. Thomas Co., Phila.*)

The rest "current" representing the difference in potential between the two parts of the body in contact with the electrodes, and quite distinct from the "action current" of the heart, is neutralized by passing through the galvanometer, in an opposite direction, a current derived from an accessory battery.

Skeletal muscle movements create a slow deflection easily recognized as distinct from that of the heart action and, in the case of mere muscular tension, one meets with a characteristic rapid vibration.

Accidental Vibrations.—Irregular rapid vibrations may arise from the passage of electric cars, defective insulation, improperly applied electrodes, loose connections, wireless waves and the induction due to an imperfect arc lamp, but are easily identified.

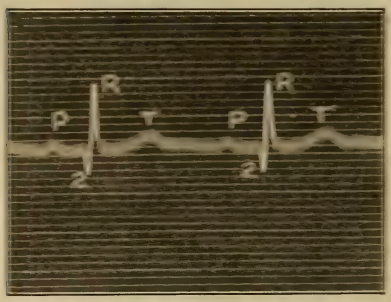


FIG. 216.—Lead I.

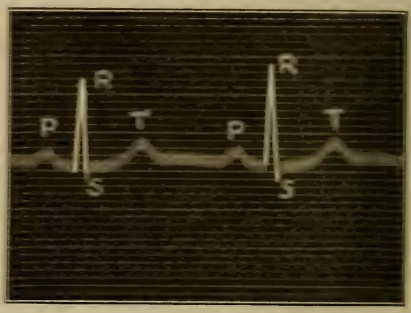


FIG. 217.—Lead II.

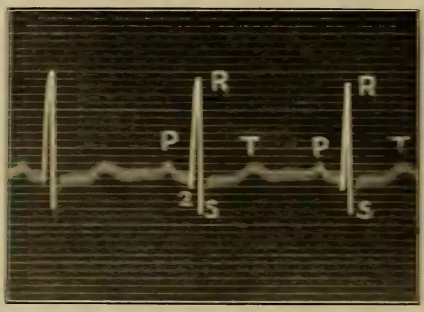


FIG. 218.—Lead III.

FIGS. 214, 215, 216.—Normal and typical electrocardiogram in each of the three leads. Lead II shows largest deflections in all phases. (Some or all R summits touched up with white for contrast throughout section. All are of equal height in original of "lead" II.)

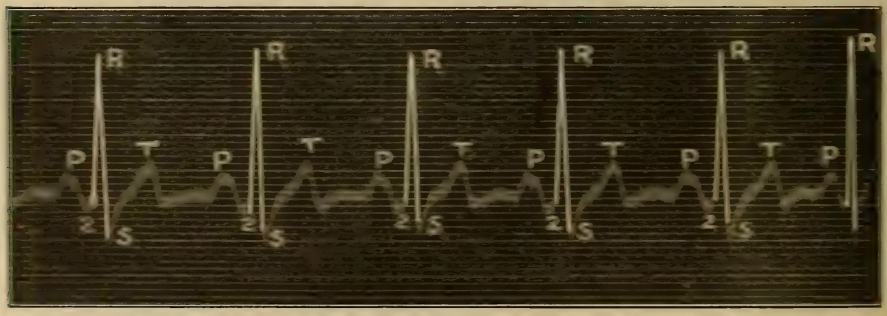


FIG. 219.—Effect of a loose string in exaggerating events of cycle. Mitral stenosis is evident in the split or truncate "P" summits. (*R. Edwin Morris.*)

Means of differentiation.

All non-muscular vibrations persist even when contact with the patient is absent. The coarse slow waves of active muscle movement are distinctive and the finer ones, though simulating auricular fibrillation, lack the distinctive absence of the "P" wave.

The Electrocardiographic Leads.—The action of the heart produces

changes of electric potential throughout the entire body and for convenience one uses the extremities for making the connections necessary to tap the cardiac action currents.

In securing the records three so-called "leads" are usually adopted:

1. *Right arm and left arm.*

2. *The right arm and left leg, the most important lead, but all three are necessary to accurate differentiation and interpretation.*

3. *Left arm and left leg.*

Choice of leads.

The electrocardiogram obtained varies slightly with the lead, one often yielding information not given by the others (see plates). The second lead is the one most generally useful, but in many instances the other leads are of especial value.

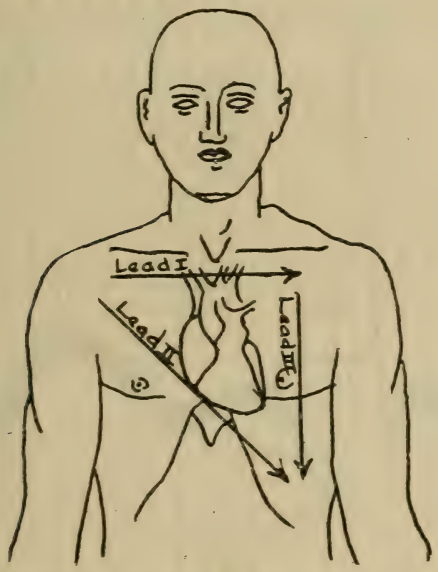


FIG. 220.—A diagram representing the relationship of the heart to the three Leads. Lead II approximates most closely the long axis of the heart, and is the lead most commonly used. (*After John Hay. Modified.*)

The Auricular Complex.—In interpreting the electrocardiogram one must remember that it normally represents practically the current generated by a contraction starting in the auricle at or near the sino-auricular node (junction of right auricle with the superior vena cava).

Starting point of contraction.

This auricular contraction is registered by the electrocardiogram as the summit "P" representing a base negative effect. This is followed by a short "no-current" (isoelectric) phase which may be represented by a horizontal line, or by a slight (base positive) downward dip.

The "P" wave probably represents the phase of sinus activity and the spread of the wave of contraction through the auricle.

The Ventricular Complex.—The ventricular complex immediately follows and is characterized:

1. *By the sharp base-negative upstroke instantly followed by a base-positive downstroke, these together forming "spiked" summit "R," which probably corresponds practically to the contraction of the papillary muscles, as it just precedes the heart sound.*

2. *By a rapidly succeeding descent ("S") which may dip below the line and finally by a slower phase ("T") which presumably represents the terminal phase of ventricular contraction and ends just before the second sound of the heart.*

Thus it appears that auricular contraction is represented by the summit "P," and that *ventricular contraction* is expressed by the phases represented by "R" and "T" or to be more exact by "Q," "R," "S" and "T."

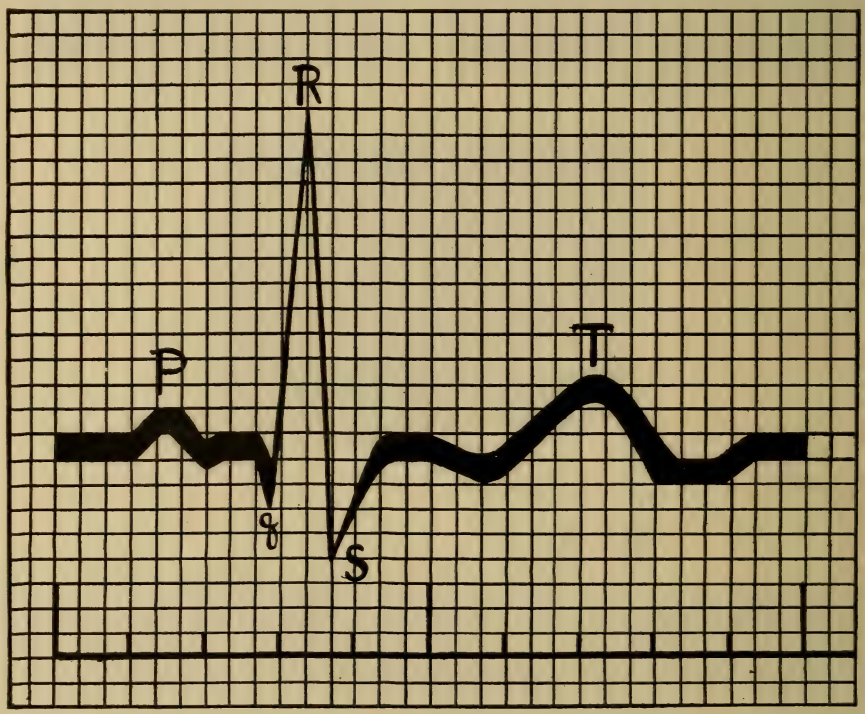


FIG. 221.—The normal and typical electrocardiogram. Showing nomenclature of Einthoven, in common use. (*After Müller.*)

VARIOUS INTERPRETATIONS OF THE ELECTROCARDIOGRAM.—Among the many conflicting interpretations of the electrocardiogram that of Hoffman is the simplest and clearest, and best lends itself to the student's memory and comprehension of the complicated and obscure phenomena. His hypothesis assumes that the electrocardiogram represents merely the variations in electric potential accompanying *impulse conduction* rather than the *act* of contraction.

The "P" wave represents auricular conduction; the "P-R" interval covers the crossing of the auriculo-ventricular bridge ("His bundle"); depression of the intervening ("Q") wave marks the phase at which the papillary muscles first

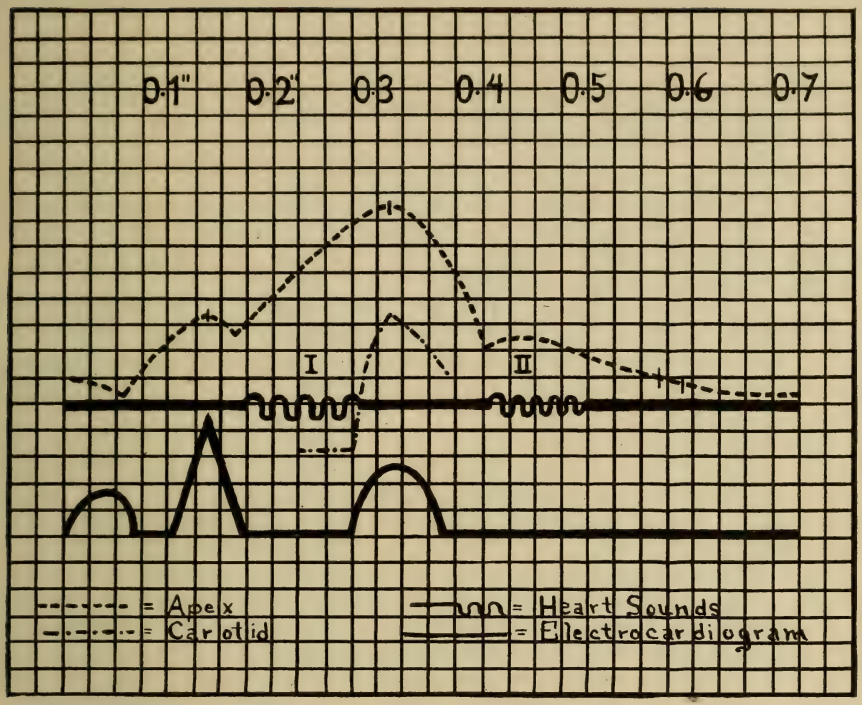


FIG. 222.—Time relationship of the apex and carotid beats, heart sounds, and events of the electrocardiogram. (*Müller, slightly modified.*)

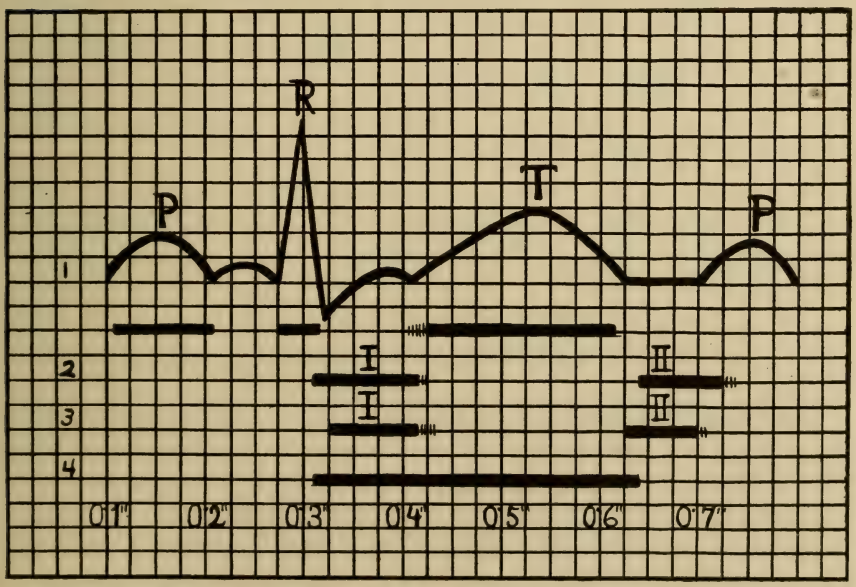


FIG. 223.—Time relationships of the electrocardiographic phases. 1. Electrocardiogram. 2. Heart tones at apex of heart. 3. Heart tones at base of heart. 4. Ventricular pressure. (*Müller. slightly modified.*)

receive the excitation; the rise of the "R" peak denotes excitation of the bases of the ventricles; the "T" wave denotes the initiation of muscular relaxation after contraction.

Wiggers thus summarizes with admirable brevity the various views now held with respect to the matter.*

SUMMARY.—The following summary expresses the different interpretations that have been given to the different waves:

The "P" wave *accompanies (a) contraction of auricle, (b) conduction through auricle, (c) activity of sinus region plus conduction through auricle.*

The "R" wave (rise) *accompanies (a) predominant contraction of right ventricle, (b) contraction of basal portions of both ventricles, (c) impulse conduction from base to apex of ventricles.*

The "R" wave (drop) *accompanies (a) predominant contraction of left ventricle, (b) contraction of apical portion, (c) conduction from apex to base.*

The "S-T" interval *results from (a) a balance of potential between left and right hearts, (b) a balance of potential between base and apex, (c) a balance between different layers of the heart, (d) an absence of further conduction because all muscle is already excited and contracting.*

The "T" wave *is due to (a) a change in the position of the heart (Uskoff), (b) a return of the contraction wave to fibers around the aorta (Gotch, Nicolai), (c) a continued negativity of base outlasting that of apex (Bayliss and Starling, and Einthoven), (d) the contraction of no particular part but the entire expression of the electrical wave accompanying the contraction of the ventricle (Samojloff, Straub, Hoffman), (e) a diminution of the intracardial short-circuiting at the end of systole when the ventricle is empty.*

The "U" wave *(not lettered) is a diastolic event, due to (a) the last relaxation of the fibers of the ventricle (Einthoven), (b) the electrical variation of the arteries (Hering).*

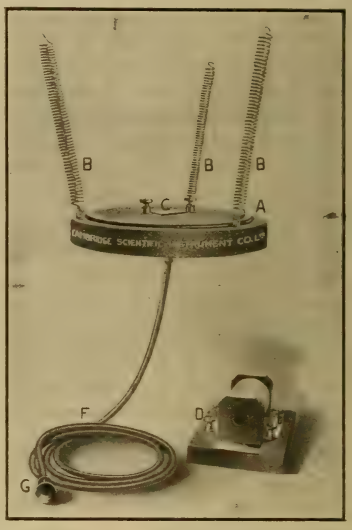


FIG. 224.—Simple apparatus for registration of heart sounds and murmurs when attached to the electrocardiograph. It consists merely in the bringing of a microphone into circuit with the string galvanometer.

Registers time pitch and volume of murmurs.

The Phonocardiograph.—By a relatively simple combination of a second galvanometer, microphone, stethoscope, rheostat, accumulator, and a transformer, simultaneous electrocardiograms and phonocardiograms are obtainable which are capable of showing both the volume and pitch of the murmur together with its exact place in the cardiac cycle and relation to the events of the electrocardiogram.

* "Modern Aspects of the Circulation in Health and Disease," Carl J. Wiggers, 1915. An admirable exposition of the most recent work in this field.

MARKING AND INTERPRETATION OF ELECTROCARDIOGRAPHIC RECORDS

Systematic Procedure Indispensable.—The absolute necessity for proceeding in a systematic and orderly manner in the marking and interpretation of the polygram was emphasized in that chapter.

While it is important there, it is even more important in regard to the marking and interpretation of electrocardiographic records.

Time Record.—In all instruments the time is indicated in hundredths of a second. In the Hindle instrument, used by the author, it is indicated by main divisions of 0.20 seconds with sub-divisions of 0.04 seconds.

General Description of Waves.—The important deflections in the electrocardiographic record have been arbitrarily designated as P, Q, R, S, and T, waves as shown on page 522. Usually the P-wave is the first upward deflection of the heart cycle. The Q-wave, which is frequently absent, is the first downward deflection immediately following the P-wave and occurring just before the tall R-wave, which is the second upward and the most prominent deflection. The S-wave is the second downward deflection and occurs immediately after the R-wave. The T-wave is the third upward deflection, occurs after the S-wave and indicates the end of the cardiac cycle. Following this is the diastolic period of rest, indicated on the record by a flat line extending to the P-wave of the next heart cycle. (See pages 522–524 inclusive and accompanying illustrations.) The P-wave normally is a small wave 1.5 to 3.5 mm. in height.

The R-wave is the tall narrow peak 5 to 25 mm. high. The width of the R-wave or the Q-R-S- interval is about 0.03 sec.

The T-wave is normally a low broad wave 2 to 4 mm. high occurring from 0.30 to 0.40 sec. after the S-wave.

The length of the diastolic period of the heart following the T-wave is inversely proportional to the rapidity of the heart beat.

Technic of Marking.—The only instrument necessary to the marking is a pair of needle-pointed dividers.

1. Indicate the first lead by the numeral I, and follow this by the date, the patient's name, and any special circumstances surrounding the taking of the record, inasmuch as these are permanent records. Designate also by numerals Leads II and III.

2. Identify and label in capitals the P, R and T waves, and the Q and S waves if they be present. Label the positive waves above the line and the negative waves below. (A positive wave is directed upward and a negative wave downward.) In this manner label three or four heart cycles in each lead.

3. Determine the P-R interval. This observation is made first on Lead II and later, if necessary, on Leads I and III merely to corroborate the first finding. To determine the P-R interval place one point of the dividers on the base line at the first point of rise of the P-wave. The other divider point is placed on the first point of rise of the R-wave. The distance between

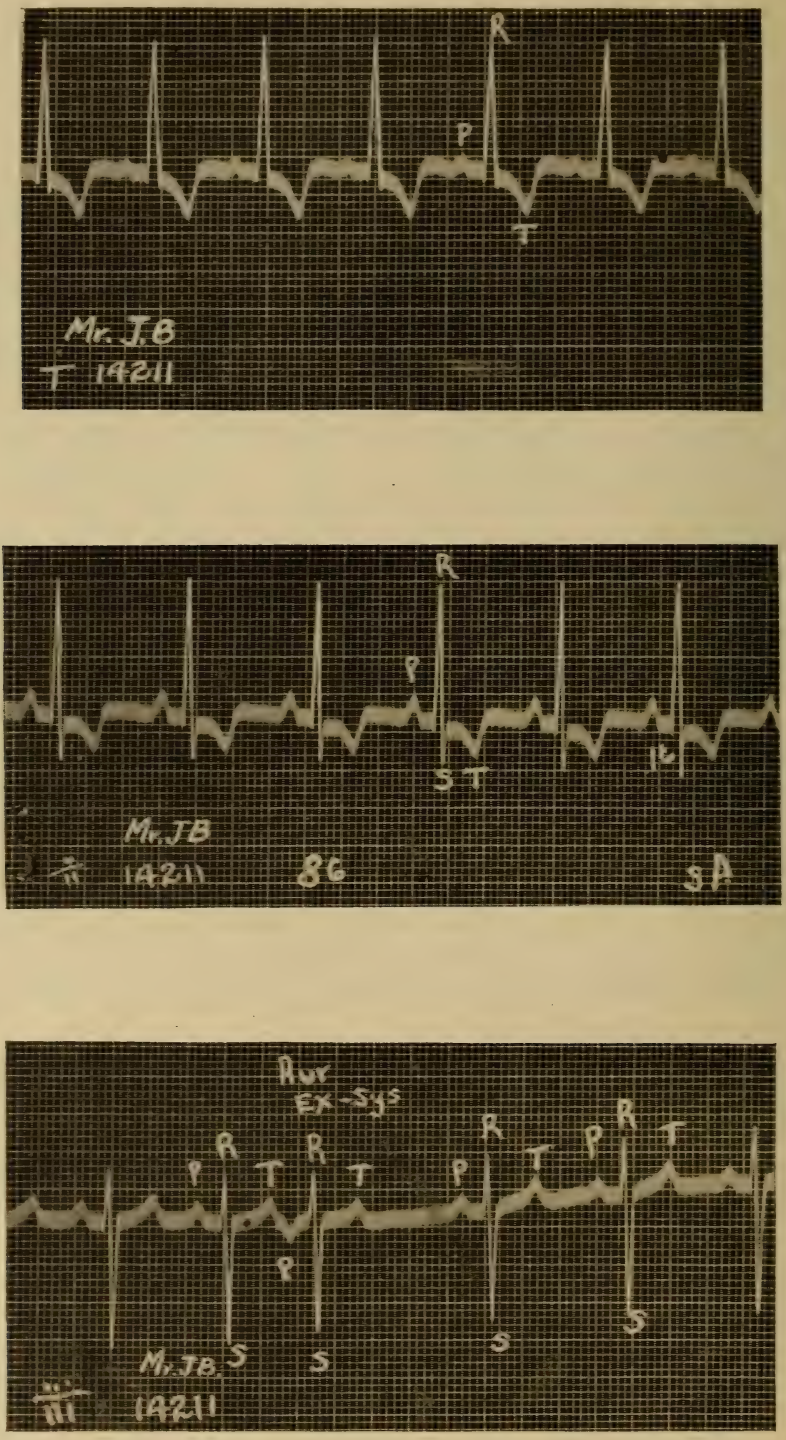


FIG. 225.—Mr. J. B. Electrocardiographic record showing right branch block and an auricular extrasystole. The extrasystole occurs in Lead III.

the two is the P-R interval of that heart cycle. This distance laid off on the time record will give its equivalent in hundredths of a second. The normal P-R interval is 0.12–0.18 sec. Indicate the time length on the record in the proper heart cycle just below the base line.

In a like manner determine the P-R interval of the remaining heart cycles of Lead II and label those showing a variation from the first reading if any be present. It is important to measure many P-R intervals, for a record may show considerable variation in them. This variability of the P-R interval in a record is of distinct clinical significance.

4. Determine the rate of the record. With the dividers lay off on the time record a distance equal to 3 seconds (15 main divisions on time record). Along the tops of the R-waves lay off a 3-second interval from the exact top of any *one* and count the R-waves occurring in that period. Then lay off another consecutive distance of 3 seconds and continue the count. The result is the number of heart cycles in a 6-second interval as recorded by the time-marker. Multiplying the figure obtained by 10 gives the rate per minute.

5. Determine the rhythm. With the presence of extrasystoles or fibrillation, the arrhythmia is readily apparent. To determine sinus arrhythmia measure the distance between the tops of any two R-waves and make comparison with several other similar intervals. The variation in these intervals in a record otherwise normal indicates sinus arrhythmia in its varying degrees.

Extrasystoles.—Extrasystoles may be auricular, ventricular, or nodal.

1. *Auricular extrasystoles* show a premature contraction of the whole heart indicated on the record by a heart cycle occurring *abnormally early in diastole*, the only abnormality evident in the appearance of this heart cycle being some change in the P-wave, which is usually of the inverted type. On the other hand the P-wave may be upright and show a narrowing or flattening, or may be diphasic; that is, showing both an upward and a downward deflection.

The diastolic period of rest following the abnormally early heart cycle is lengthened, but is *less* than two heart cycles in duration (incomplete compensatory pause).

2. The *ventricular type of extrasystole* is characterized on the record by an abnormally appearing exaggerated *diphasic* wave; that is, extending both above and below the base line. It stands out in striking contrast to the rest of the record. Bearing no relation to the stimulation or contraction of the auricles, the ventricular extrasystole occurs with no relation to the P-wave of auricular contraction. It is a premature heart cycle in which the impulse to contraction originates in the ventricles, not in the auricle. This ectopic impulse causes a contraction of (1st) the ventricle in which it originates, and (2nd) a contraction of the opposite ventricle. This asynchronism of the ventricles, while *very slight* is sufficient to cause an absolutely characteristic electrocardiographic picture, and determine the ventricle, right or left, in which the aberrant impulse originates. One is able, therefore, by means

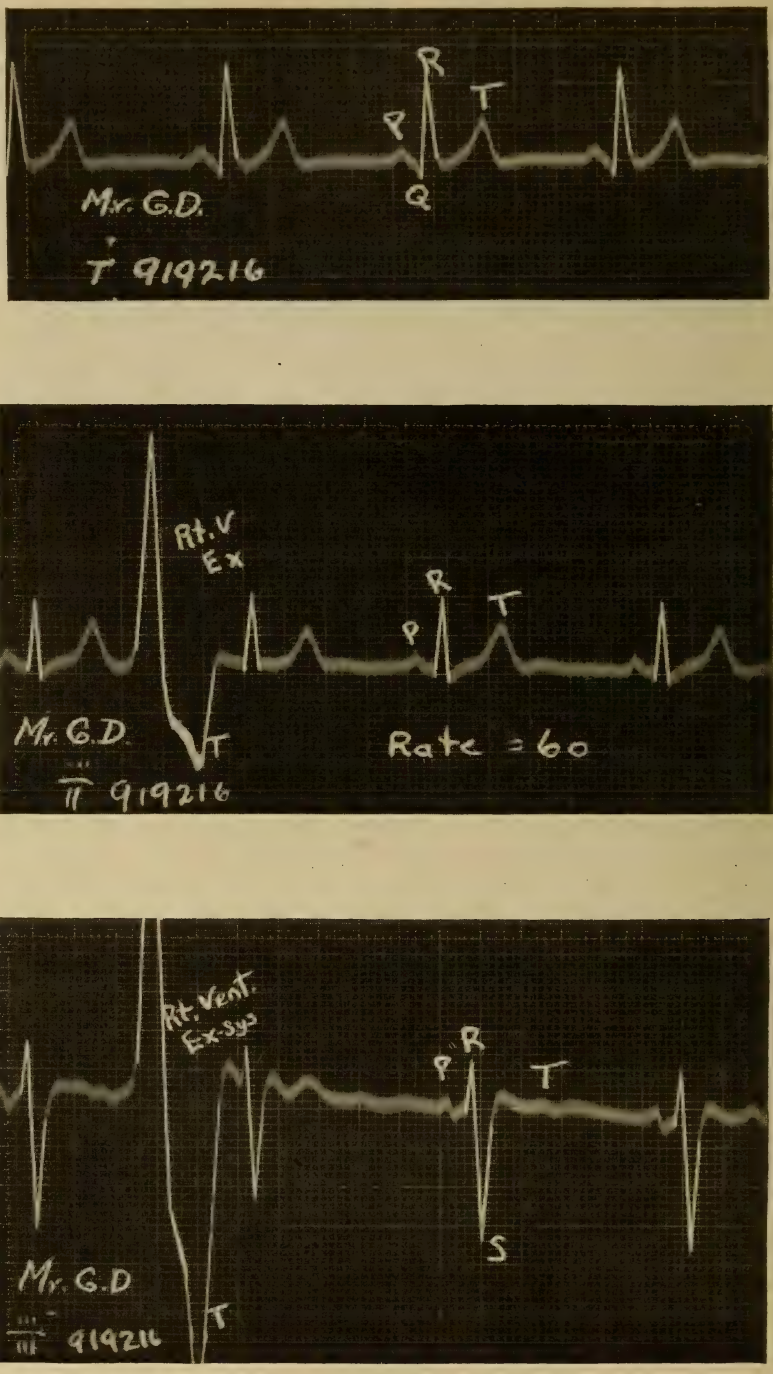


FIG. 226.—Mr. G. D. Electrocardiographic record showing right ventricular extrasystoles and overaction of the ventricles in a slowly beating heart. Left ventricular preponderance indicated in high R in Lead I and deep S in Lead III. Conduction time normal. Slowing of heart due to digitalis. Exaggeration of T-wave shows overaction of ventricles.

of the electrocardiograph to differentiate the side in which the ventricular extrasystole occurs, by the following characteristics.

In Lead II a right ventricular extrasystole is indicated by an initial upward deflection, while the direction of the T-wave following is downward. A left ventricular extrasystole is indicated in Lead II by an initial downward deflection, while the direction of the T-wave following is upward. In other words the one is a mirror image of the other. The direction of all waves of the extrasystole occurring in Lead I may be opposite in direction to those occurring in Leads II and III. For example, a right ventricular extrasystole in Leads II and III would, in Lead I have the appearance of a left ventricular extrasystole and vice versa.

The auricular contraction being unaffected as has been said before, the P-wave may be evident after the extrasystole or occur during the extrasystole, malforming its contour.

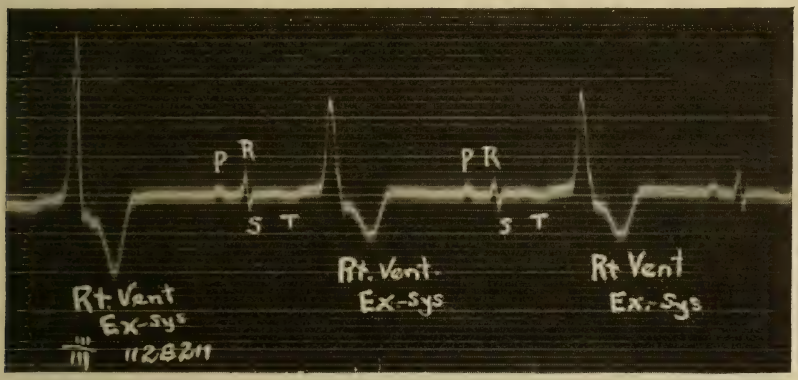


FIG. 227.—Lead III. Three examples of right ventricular extrasystole as shown by the electrocardiograph.

The pause following the ventricular extrasystole before the occurrence of the next heart cycle is equivalent to two heart cycles, spoken of as a complete compensatory pause.

3. *Nodal extrasystoles* are premature heart cycles in which the impulse for contraction is supposed to arise in the auriculo-ventricular node instead of in the pacemaker, (sino-auricular node). These are indicated on the record by an abnormally early heart cycle normal in appearance with the exception that the P-wave is absent or merely suggested. The compensatory pause following is equivalent to two heart cycles.

A short run of nodal extrasystoles is spoken of as nodal rhythm.

Fibrillation is characterized in the electrocardiographic record by the absence of P-waves, the occurrence of multiple coarse or fine undulations (fibrillatory waves), and an utterly disorderly rhythm in the occurrence of the R-waves. The latter show also considerable variation in height.

Fine fibrillation is usually associated with the rapid rates while with coarse fibrillation we have usually a slower rate. The P-wave being absent, of course there is no P-R interval.

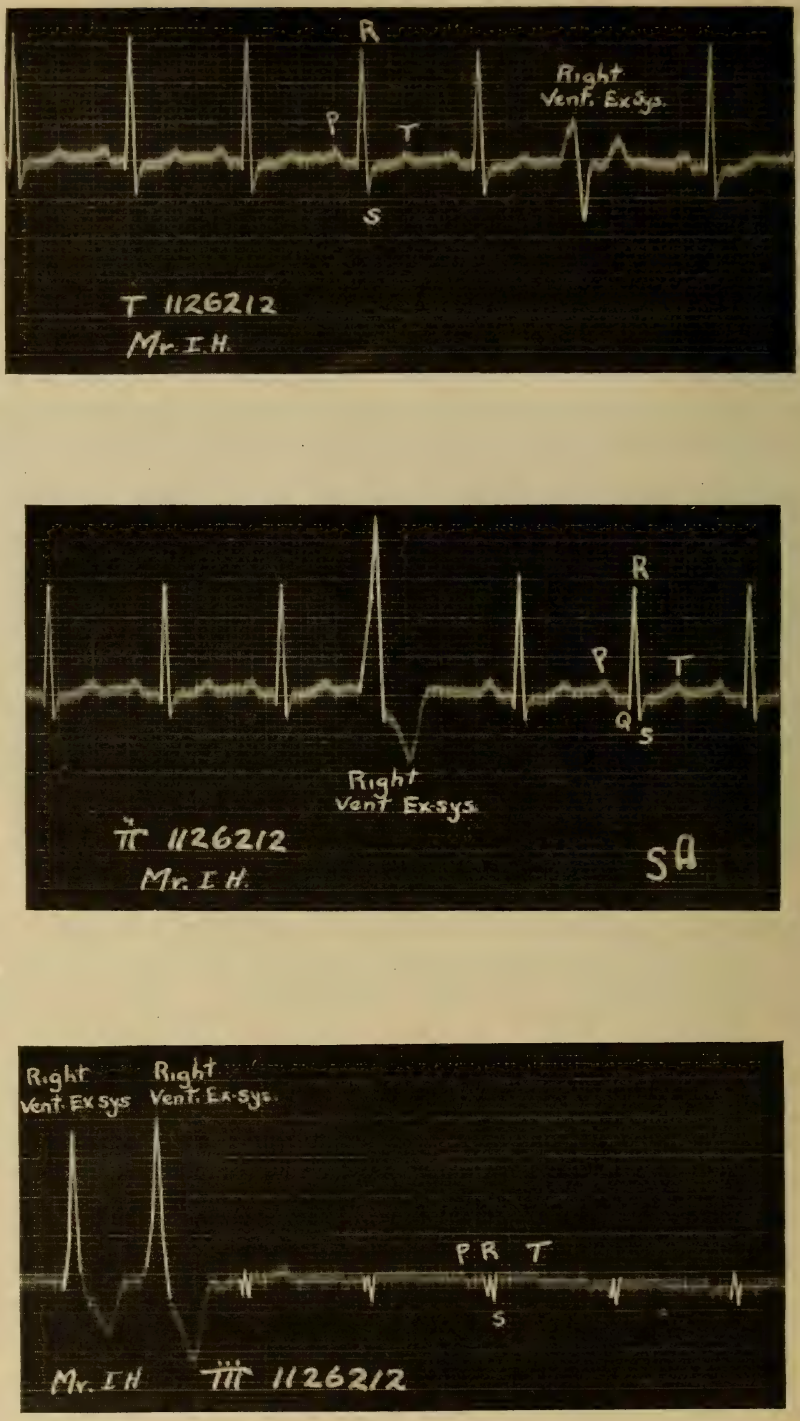


FIG. 228.—Mr. I. H. Electrocardiograph record showing a right ventricular extrasystole in each Lead. The direction of the deflections of ventricular extrasystoles in Lead I is opposite to that of the deflections in the other two leads.

Block.—Heart block may be either complete or partial. In complete block there is an entire dissociation between the P-waves and the R (or S) waves, together with a slow ventricular rate, as indicated by the number of R-waves occurring in the 6-second interval.

In this condition the complete block existing in the conducting fibres compels the ventricles to adopt their own, inherent, slow deliberate rhythm causing an occurrence of correspondingly few R (or S) waves. The con-

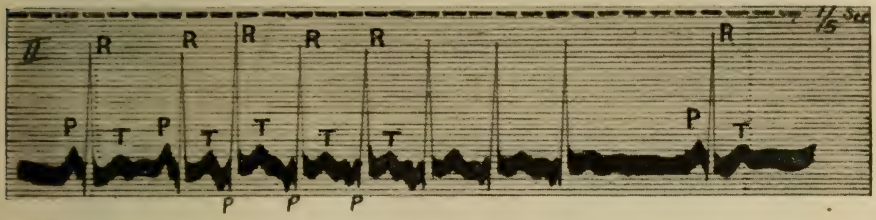


FIG. 229.—Lead II. A short paroxysm of six beats shown electrocardiographically to be of A–V nodal origin. P-wave is inverted during the progress of the paroxysm, and the P–R intervals are shortened. Time in one-fifth of a second. (*After John Parkinson.*)

duction through the auricles being unimpeded, they respond freely to the stimulation arising in the pacemaker (sino-auricular node). The result is a preponderance of P-waves over R-waves varying in degree with the ventricular rate. An auricular rate of 75 may accompany a ventricular rate of 40 to 20 or much less, for example.

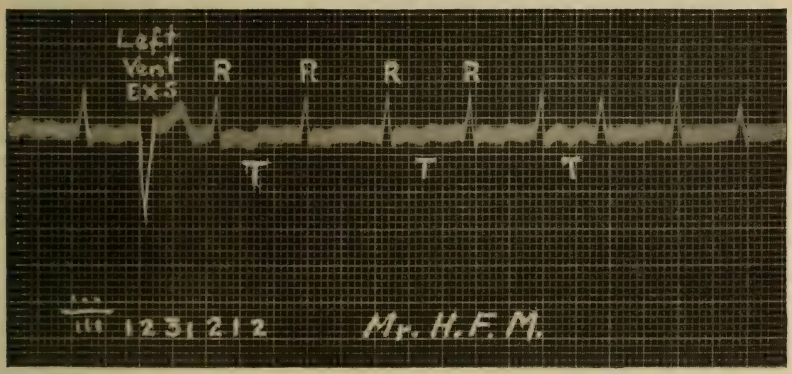


FIG. 230.—Mr. H. F. M. Lead III in a case of auricular fibrillation with a rapid ventricular rate. Left ventricular extrasystole is present. Note absence of P-waves, and inversion of T-wave.

In partial block some of the auricular contractions get through and cause corresponding ventricular contractions. There is found a preponderance of the P-waves again but at definite intervals there is found a P-wave which has its normal relation to the R-wave. For example in 2:1 block every other P-wave is in its proper relation to the R-wave with an extra P-wave midway between these two. Here the auricles are beating twice as fast as the ventricles. In 3:1 block there are two extra P-waves between two normally placed P-waves, all of them being the same distance apart.

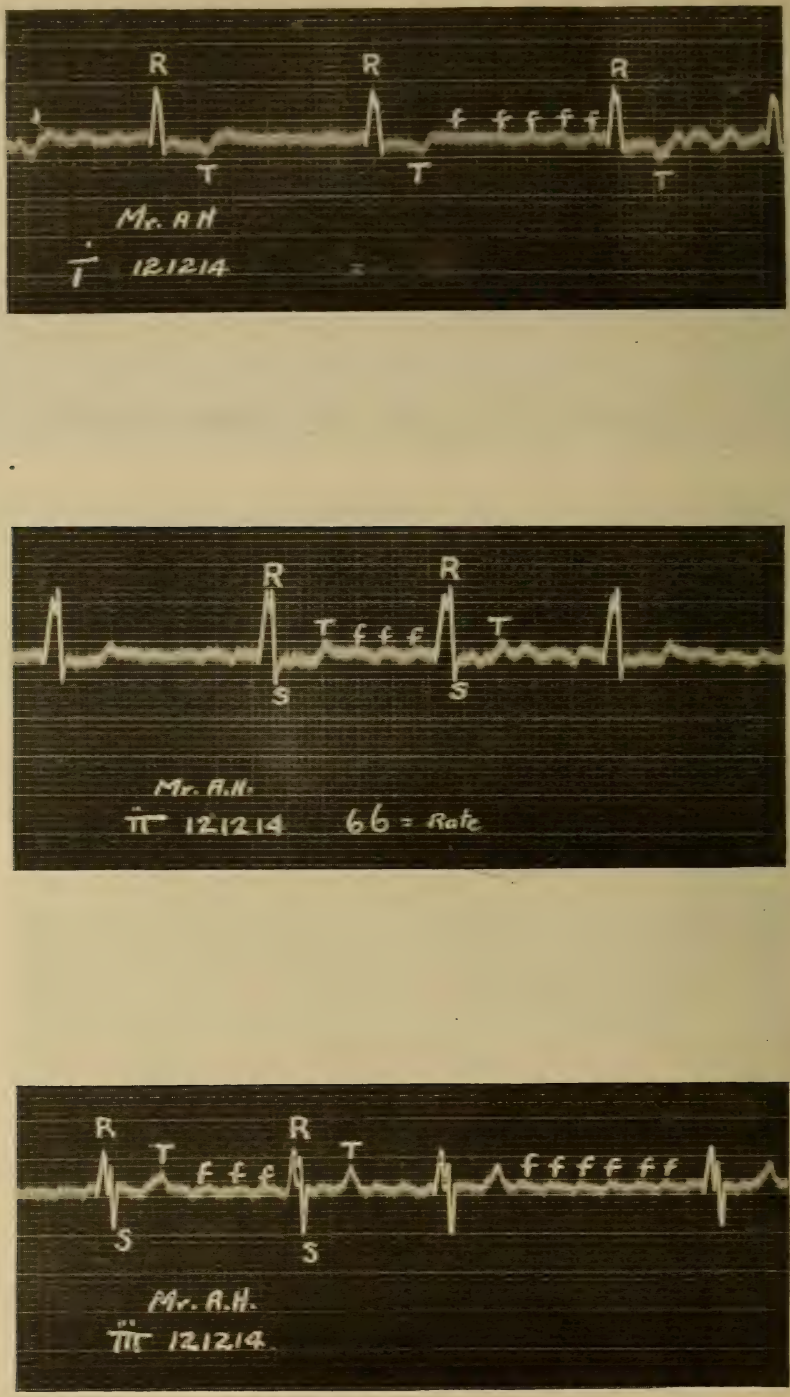


FIG. 231.—Mr. A. H. Electrocardiographic record of auricular fibrillation under the influence of digitalis. Note the complete absence of P-waves and the presence of coarse fibrillatory waves (fff). The slow rate of 66 is due to the administration of digitalis. Arborization defect is indicated by the notching and widening of the R-waves.

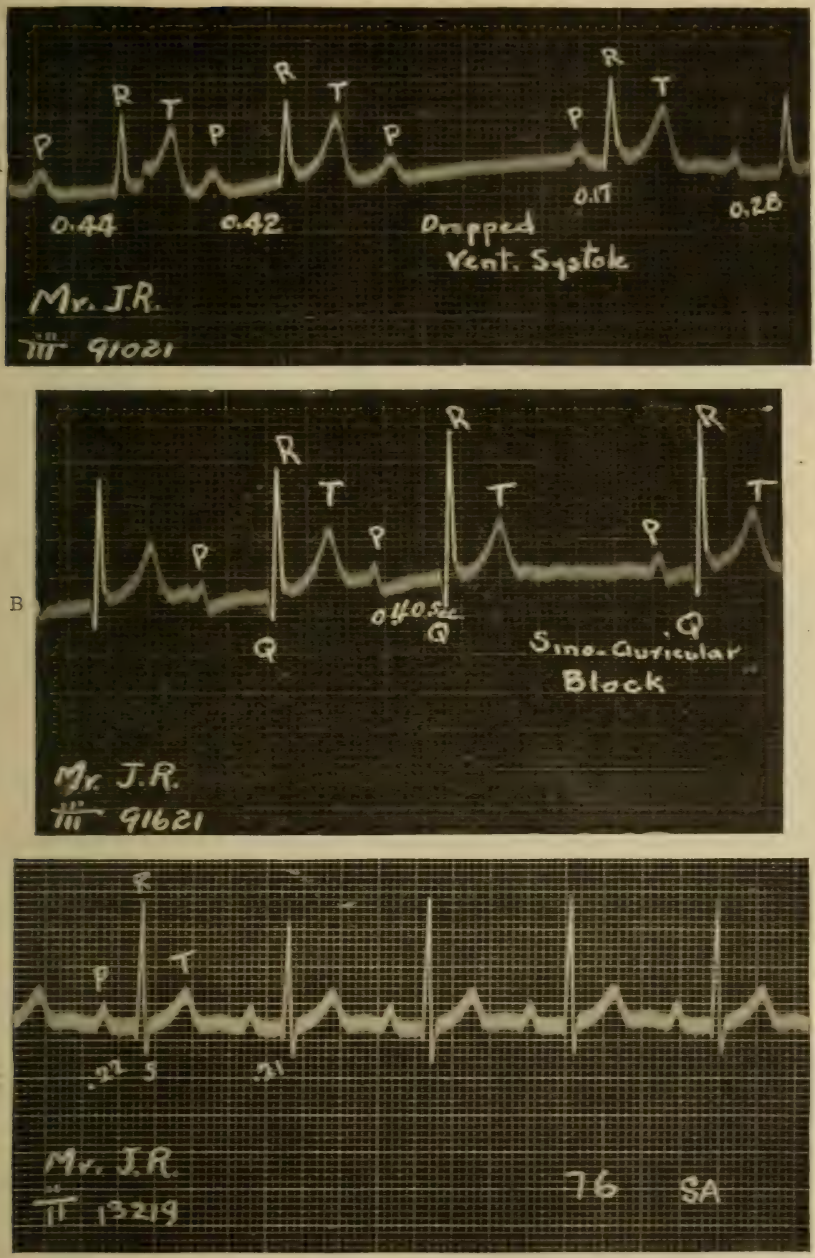


FIG. 232.—Mr. J. R. Electrocardiographic records on a 16-year old boy with mitral and aortic insufficiency and decided decompensation.

(A) Shows a dropped ventricular systole. The conduction time before the period of block is 0.44 and 0.42 sec. and immediately after the rest-period of the Bundle of His is 0.17 sec. and then immediately lengthens to 0.28 sec. This shows the presence of extreme toxemia and the marked fatigability of the bundle.

(B) Taken 6 days later still shows a markedly delayed conduction (0.40 sec.) and sino-auricular block. This is simply an arrest of the whole heart and comparison with (A) will show that the period of rest is not preceded by a "lone" P-wave, as occurs in partial block.

(C) Taken 4 months later after hospital regime, showing absence of all forms of block and reduction of conduction time to 0.22 sec.

In the less severe grades fewer and fewer ventricular contractions are missed until there may be a ventricular beat dropped only rarely.

Sino-auricular block is indicated on the record by a temporarily arrested action, that is, the diastolic period is unusually long but the heart cycles preceding and following are both absolutely normal.

It is customary to indicate the occurrence of events such as described above on the face of the record in the following manner: Right ventricular extrasystole—R.V.Ex., left ventricular extrasystole—L.V.Ex., total block—T.B., partial block—P.B., fibrillation—f, f, f, sino-auricular block—S.A.B., etc.

The reader will recall that the Bundle of His (conducting bundle) divides into two main branches, one for each ventricle, and that each of these in turn subdivides into many smaller branches, ending finally in specialized cells, called the Purkinje cells which are situated in the outermost portion of the ventricular musculature. Block may occur in any branch or subdivision, and is determinable by the electrocardiograph alone.

In *branch block* the conduction of the impulse to contraction is interfered with in one of the main *branches* of the bundle of His. The electrocardiograph indicates this condition by the following findings:

1. An accentuation of the first and third leads; that is the deflections are wider here than in the second lead.
2. The R-wave is widened (normally it is 0.03 sec.).
3. There is a notching of the R or S waves.
4. The T-wave in the first and third leads is opposite in direction to that of the dominant wave of those leads.
5. The general appearance of the second lead is that of a continuous ventricular extrasystole of the side opposite to that on which the block occurs. This has been taken by some as indicative of the side on which the block occurs, but this finding has been found not to be constant. The branch blocked is now more accurately indicated by the following findings.

In a block of the right branch, the first lead shows a high R-wave, with an inverted T-wave, while the third lead shows a deep S-wave with the following T-wave upright.

In a *block of the left branch*, Lead I shows a deep S-wave, followed by an upright T-wave, while lead three shows a high R-wave with the T-wave following being inverted.

In arborization block, one of the lesser branches is affected. The electrocardiographic picture has the same essential features as that of branch block, but all are exaggerated. The difference is one of degree only.

Arborization defect is a condition in which the terminal fibers of the "His" conduction system are affected. It is a much less important condition than either branch block or arborization block, and is indicated on the records by a notching of the R-waves or the S-waves, the rest being unaffected. It is usually interpreted as indicative of some impairment of the intrinsic circulation of the heart. Some writers believe it is due to local pathology. Robinson in a recent article assumes that it is due to the

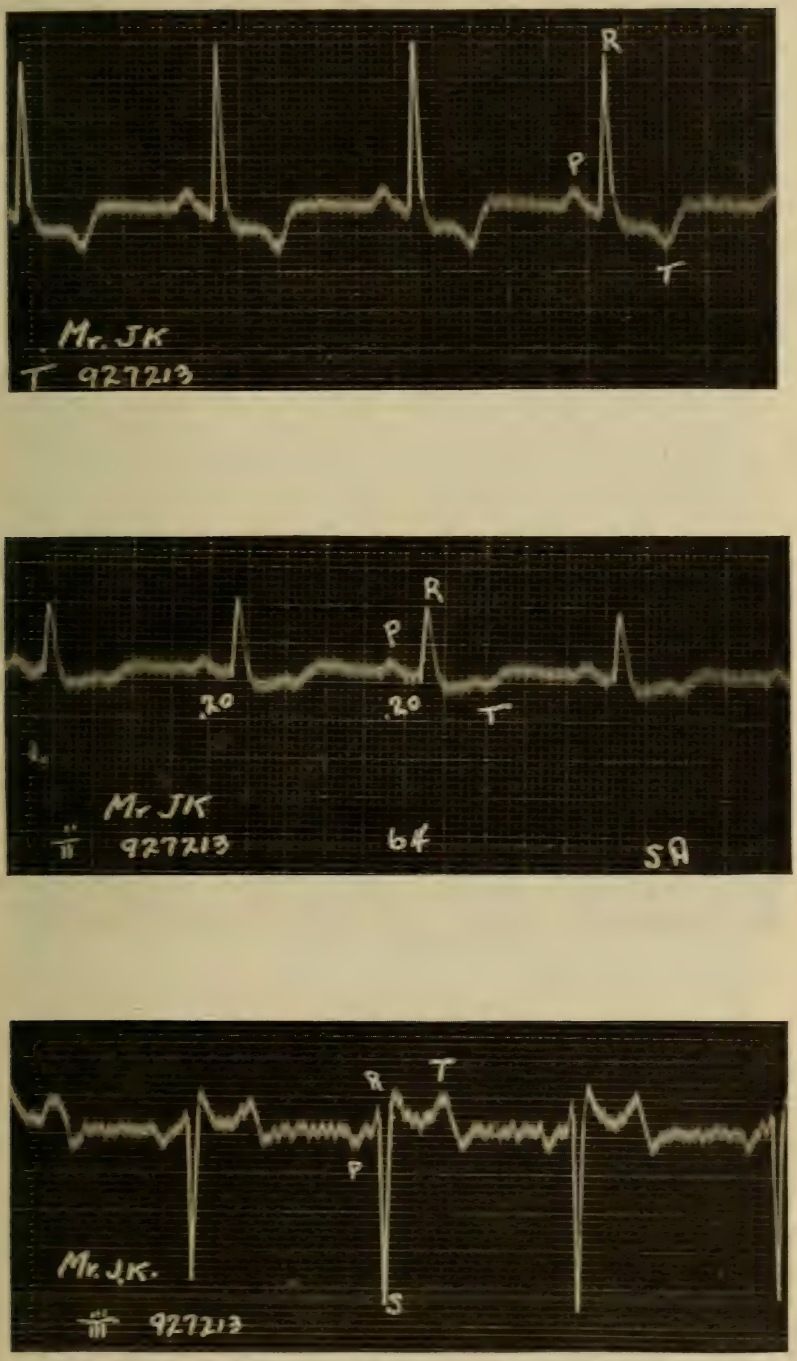


FIG. 233.—Mr. J. K. Electrocardiographic record of right branch block. Right branch block is indicated (1) by a vertical accentuation of Leads I and III; (2) by a widening of the V–R–S interval; and (3) by the inversion of *T–i*, its direction being opposite to that of the dominant *R–i;* and *T–iii* upright in direction and opposite to the dominant *S–iii*.

presence of acid metabolites which are the result of anything that interferes with nutrition of these tissues, be it fatigue, over-strain, sclerosed coronaries, or what not.

Abnormalities in the P-wave.—Normally the P-wave is a small rounded wave, 1.5 to 3.5 mm. high. A P-wave higher than this is spoken of as an exaggerated P-wave. A wide and exaggerated P-wave is usually interpreted as indicating auricular hypertrophy, while a tall narrow one means usually, over-acting auricles. A definite notching of the P-wave is taken to indicate an asynchronism of the action of the auricles. This is a common finding in mitral stenosis.

Immediately after the P-wave and before the R-wave, is an isoelectric (no-action) flat line. Frequently there is found here, however, fine oscillations. These may stop just before the R-wave, or they may continue on past the R-wave and be evident up to the T-wave. This condition is spoken of as continued action of the auricle.

Auricular disturbance is a term applied to the finding of indefinite undulations in the diastolic period of rest on the electrocardiogram, between the T and the following P-wave.

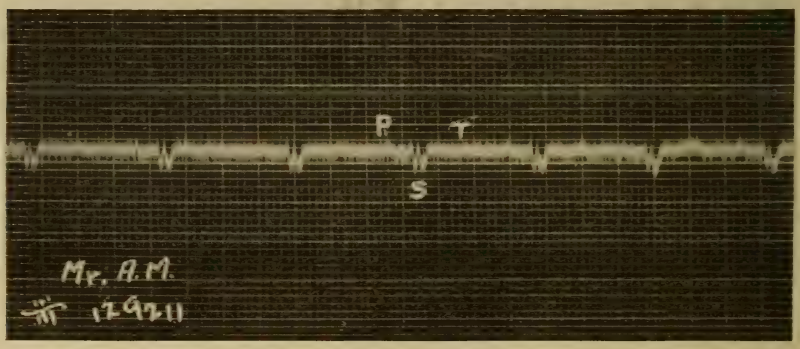


FIG. 234.—Mr. A. M. Lead III of electrocardiographic record showing arborization defect as indicated by the notching and widening of the S-waves.

The P-R Interval.—As has been said before, the normal P–R interval is 0.12 to 0.18 sec. A lengthening of the P-R interval is spoken of as delayed conduction and this finding is of great clinical significance. The usual delayed conduction finding is 0.19 to 0.24 sec. The longest conduction period noted by the author is 0.42 sec. without actual block.

Frequently there is found variation of the conduction period within the normal limits. This means that the impulse to contraction does not always start from exactly the same point.

The T-wave.—This is the last wave of the heart cycle and usually is a positive wave, from 2 to 4 mm. high. The inversion of the T-wave in the first lead, the first and second, or the first second and third leads is very important from the standpoint of prognosis, and is thought to indicate the presence of one of the graver heart conditions, making the prognosis less favorable. Inversion of the T-wave in the third lead alone is of no practical

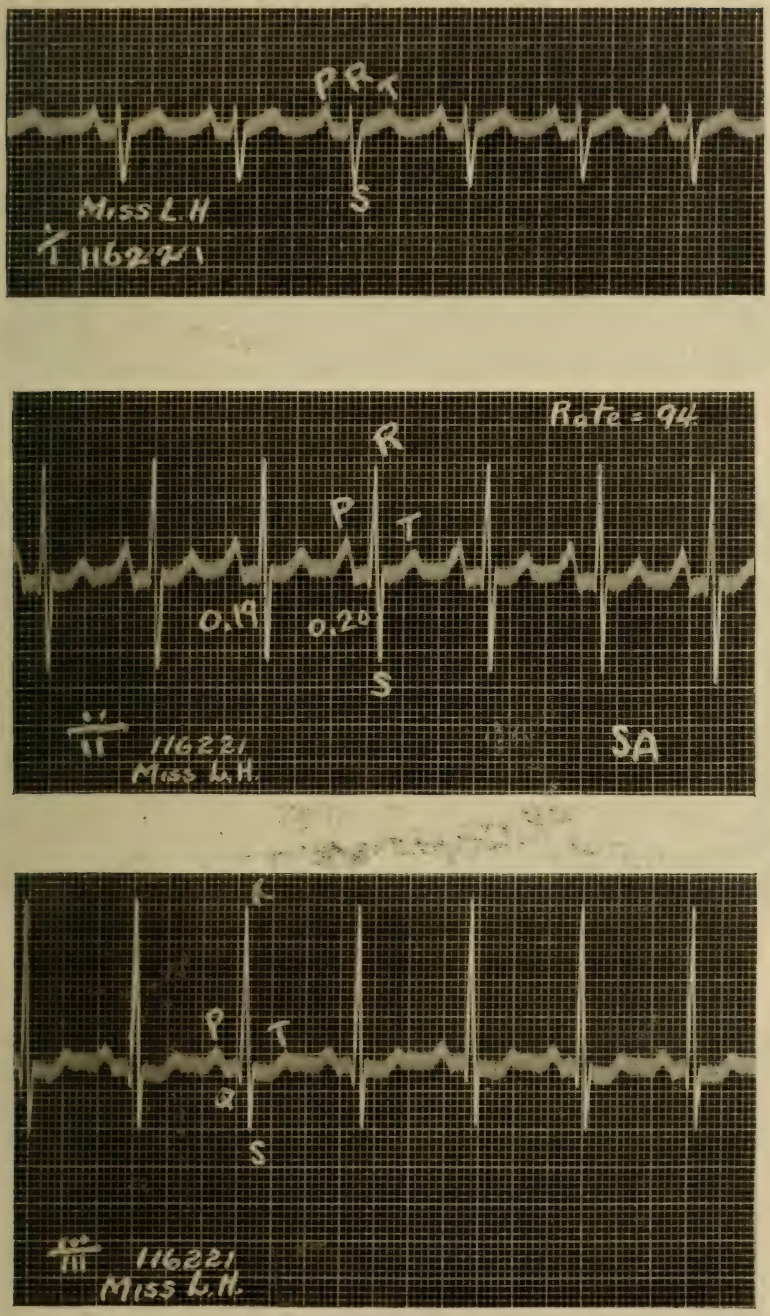


FIG. 235.—Miss L. H. Atypical electrocardiographic record in a 14-year old girl with mitral stenosis. *P–ii* is exaggerated and wide showing the auricular hypertrophy. The *typical* notched P-wave of mitral stenosis is flat-topped. Slight notching of the P-wave, indicative of asynchronism of the auricles, can be made out here and there. The conduction time is delayed, being 0.18–0.20 sec. Right ventricular preponderance is indicated by the deep *S–i* and the high *R–iii*. The deep *S–ii* shows the asynchronism of the ventricles.

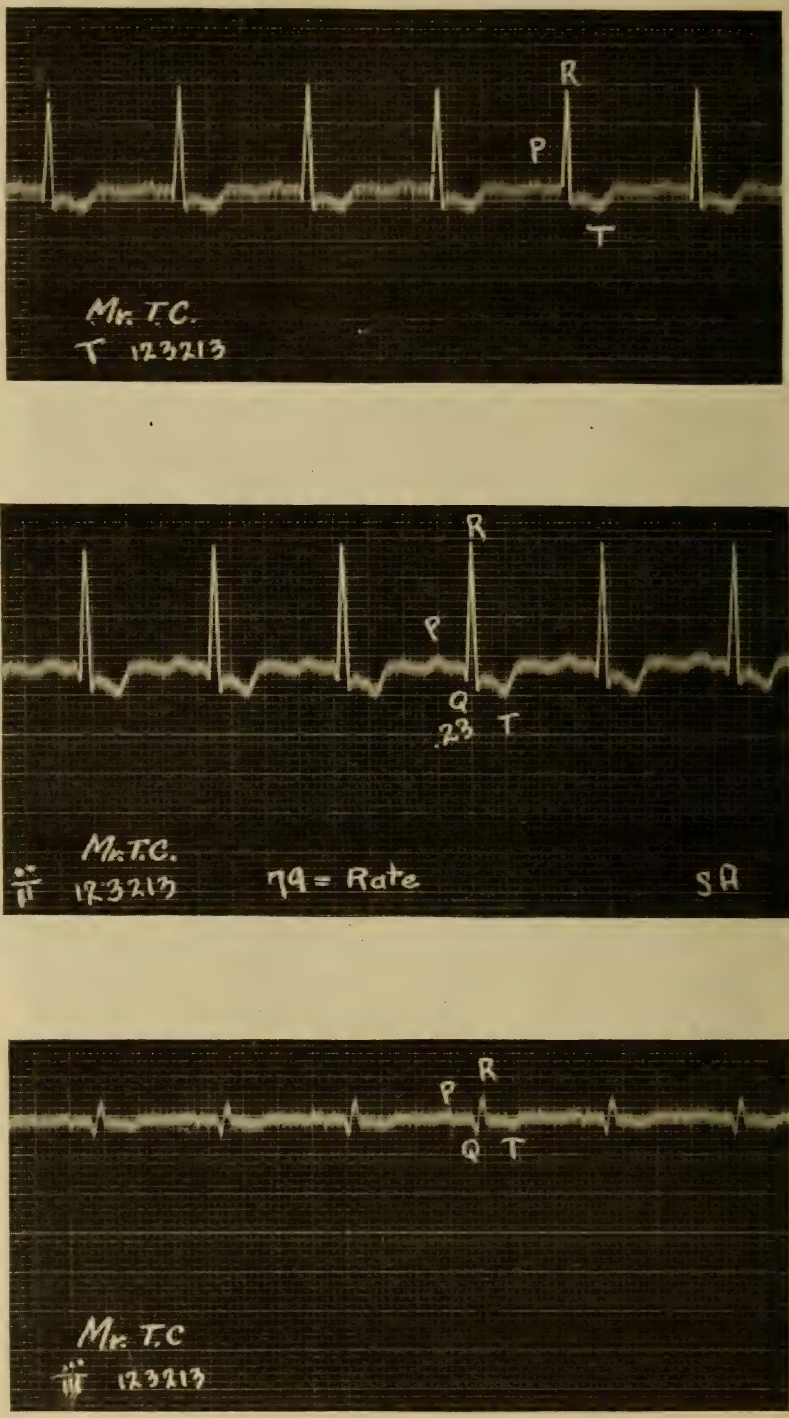


FIG. 236.—Mr. T. C. Electrocardiographic record of over-digitalization. This condition is characterized by an inversion of the T-wave in all Leads together with a delayed conduction.

significance. The inversion of the T-waves due to digitalis must not be lost sight of however, and it is usually readily distinguishable from the other types. It follows the administration of digitalis, and besides the inversion of the T-wave in all leads, there is also a delayed conduction. The sudden development of this picture in an electrocardiogram, should call for immediate cessation of all digitalis administration. Over-digitalization.

Inverted T-waves are of two types. (1) That type which is the mirror image of an upright T-wave, and (2) that type in which there is a negative wave which begins earlier than the rise of the usual upright T-wave. This negative phase then rises, and becomes actually a positive phase, small but definite, ending at the usual time for termination of the T-wave.

Ventricular Preponderance.—In ventricular preponderance, there is increased muscle mass, in one or the other ventricle, over that normally found. This increases the conduction time through the affected side, the result being the loss of the normal unison of action of the two ventricles and a characteristic electrocardiographic picture, depending upon the side involved. In preponderance of the right ventricle, there is found a deep S-wave in the first lead, while the third lead shows a dominant R-wave. In preponderance of the left ventricle, the picture is the mirror image of this and shows a dominant R-wave in the first lead with a deep S-wave in the third lead.

Asynchronism of the ventricles is indicated by an S-wave in the second lead.

Auricular Flutter.—This is a condition of extremely rapid, rhythmic auricular contractions associated with a partial block and occasionally with alternation (alternately higher and lower R-waves). An associated block may give rise to a 2:1, 3:1, 4:1, or in rare instances even 6:1 ratio between the recorded auricular and ventricular waves (auricular = P, ventricular = R and T, or S and T-waves). This condition is accurately determinable only by instrumental methods. (See page 555 and 556.)

Paroxysmal Tachycardia.—This condition is characterized by a *sudden outburst* of rapid action of the whole heart and its *abrupt termination* after a period of minutes, hours or days duration. It is usually, if not always, initiated by extrasystoles of auricular origin. Rarely it is said to be ventricular in origin. (See page 559.)

Special Conditions.—The infantile type of heart is a centrally placed, low-lying heart. The electrocardiographic record in these cases is said to be characterized by a minute first lead, the other two leads being of the usual height.

Hearts having a congenital defect are said to give characteristic electrocardiographic records, and the unusually wide R-waves, together with evidences of hypertrophy in one of the chambers are taken as the indications of this condition.

Conditions of hypotonus of the myocardium are indicated on the electrocardiographic record by unusually low P and T-waves throughout the entire record.

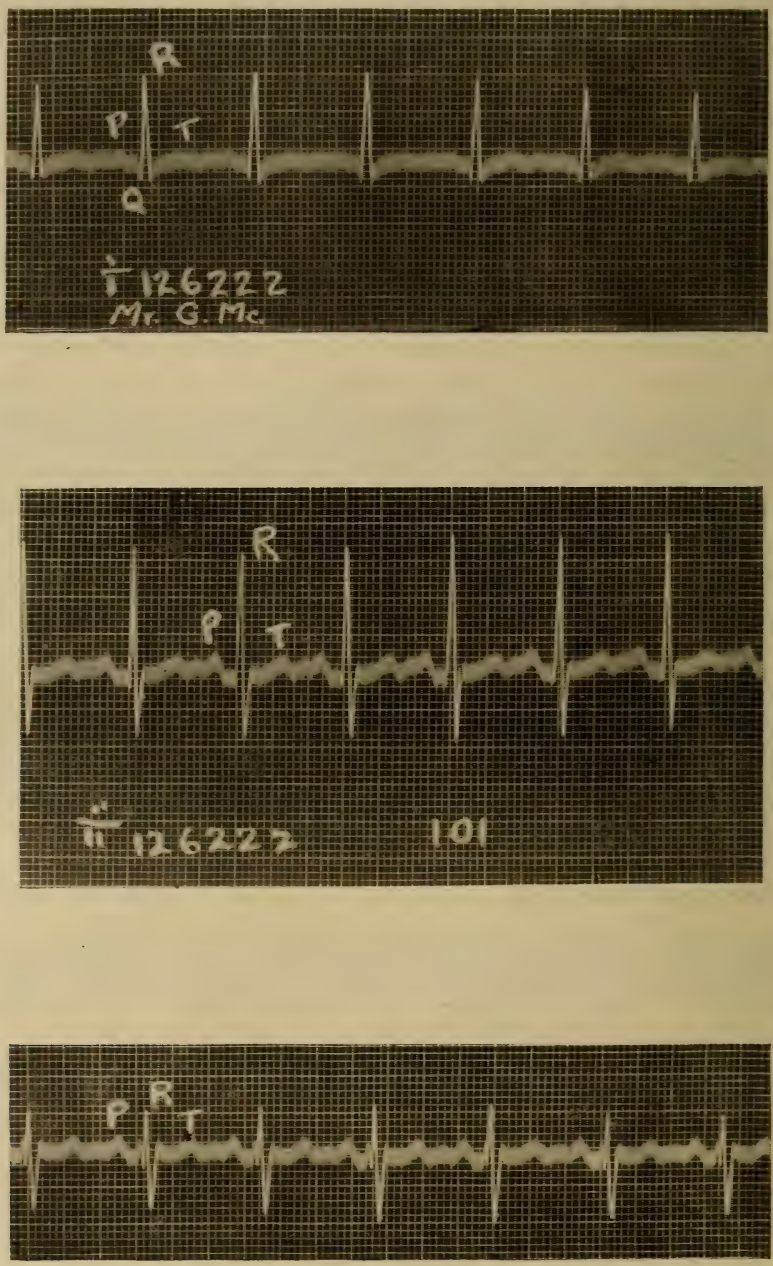


Fig. 237.—Mr. G. Mc. Electrocardiographic record showing left ventricular preponderance in a typical "drop" heart of a 36-year old male. The preponderance of the left ventrical is indicated by the high *R–i* and the deep *S–iii*.

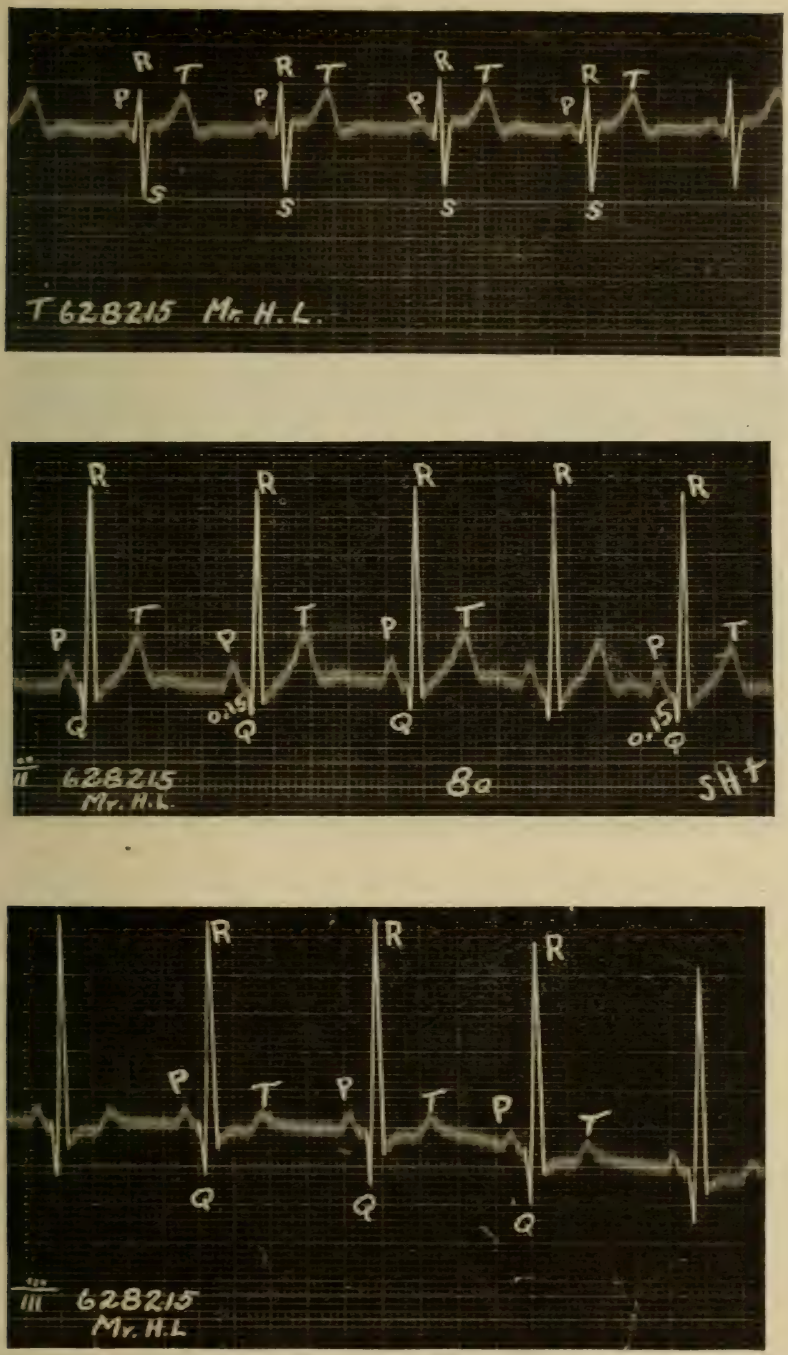


FIG. 238.—Mr. H. L. Electrocardiographic record in a case of typical "soldier's heart" (exhausted insufficient "drop" heart) occurring in a 16-year old male. V-wave in Leads II and III shows marked continued action of the auricle. Conduction time is normal (0.15 sec.). Right ventricular preponderance is indicated by a deep *S–i* and a high *R–iii*.

An arching of the T–P interval, which is usually flat, is said to indicate a hyperthyroid condition. It is spoken of as the hyperthyroid type of record.

Myocarditis is said to be indicated by an abnormally low lead **II**, the first and third leads being normal in height.

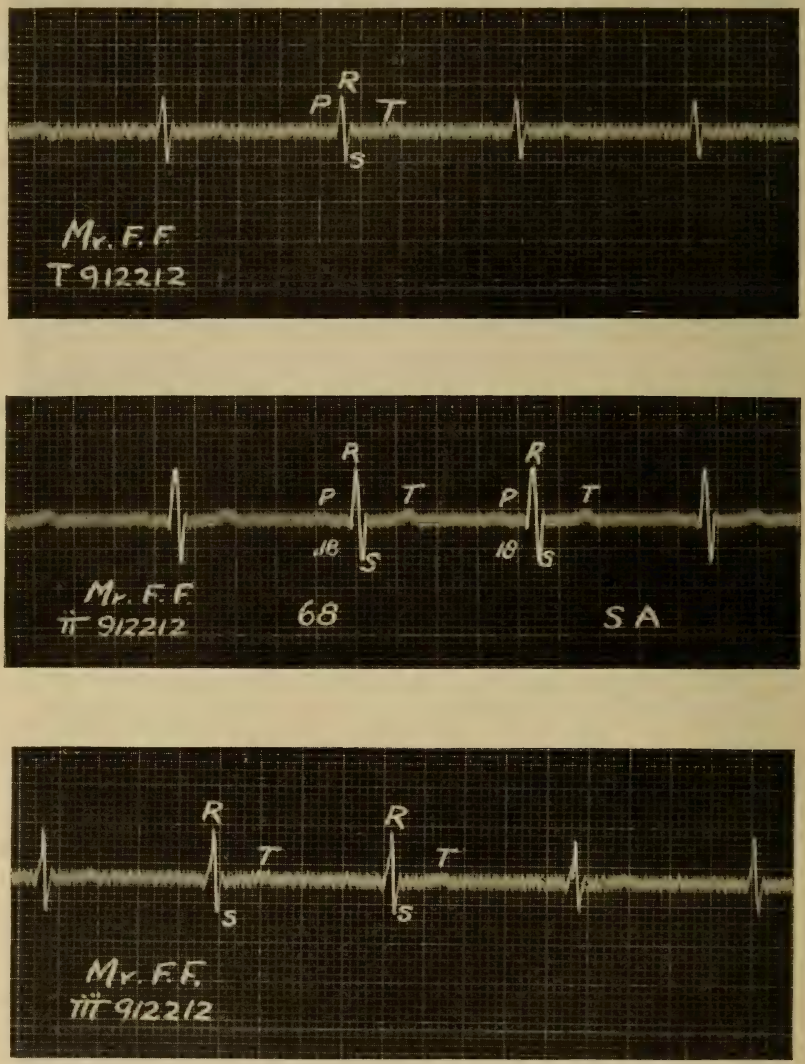


FIG. 239.—Mr. F. F. Electrocardiographic record showing hypotonus. This is indicated by the marked flattening of both the P- and T-waves throughout the entire record.

Alternation, a condition very important from the standpoint of prognosis, may be indicated in the electrocardiographic record, by alternating inequality in the height of the R-waves. This finding is not constant, the polygram being much more dependable, but when it is shown, it is clearly shown. (See pages 516 and 561.)

CARDIAC ARRHYTHMIAS

Classification of Arrhythmias.—The simplest is that of von Tabora, who divides them as follows:

1. *Arrhythmias usually associated with retained fundamental rhythm (pararrhythmias), viz.:* (*a*) **Extrasystoles** (*premature contractions*). (*b*) **Impaired conduction** (*heart block*).

2. *Arrhythmias resulting from causes which abolish the fundamental rhythm (true arrhythmias), viz.:* (*a*) **Extracardial (sinus) arrhythmias** (characterized chiefly by equality of force co-existing with variations in the length of the diastolic periods). (*b*) **Intracardial arrhythmia,** viz.: auricular fibrillation (**"arrhythmia perpetua," "delirium cordis"**).

Paroxysmal tachycardia and alternation are not properly arrhythmias but represents respectively: the one, abnormal acceleration; the other, rhythmic variations in the force of the contraction.

ANALYSIS OF THE ARRHYTHMIAS.—Clinical Importance.—In most cases the *direct* clinical information afforded is not great save in the case of auricular fibrillation, yet certain important deductions may be drawn and at times accurate and more timely diagnosis or prognosis made possible, if one can readily differentiate the arrhythmias, with the various ingenious instruments now available for research and teaching.

Occasiona great value.

EXTRASYSTOLIC ARRHYTHMIA (*Premature Contraction*).—*The extrasystole is an abnormally hasty, inefficient contraction initiated by misplaced stimuli which may be generated at any point in the heart muscle or its conduction system. It introduces not an additional beat, but merely a premature one without vitally altering the fundamental rhythm, as may be determined instrumentally or, usually, by auscultation of the heart.*

A common arrhythmia.

The extrasystole may or may not carry its wave into the radial artery. The untimely precocious beat is not an *extrasystole* actually for it merely replaces the one which should come at the normal time and is followed usually by a long pause which is practically equivalent to the displaced full cycle plus the shortened diastole of the premature systole. If the latter does not carry to the radial the pulse intermission equals two complete cycles.

Compensatory pause.

Frequency of Extrasystolic Contractions and Their Direct Cause.—These and the auricular fibrillations are the commonest irregularities associated with cardiac disease and result from irritability or an extreme stimulation of the whole, or some portion of the myocardium or conduction system which releases the hasty aberrant and ineffectual contractions before the proper filling of the heart chambers is completed and causes an elision of the normal systoles which these premature contractions anticipate and replace.

Abnormal *loci*.

One encounters extrasystoles originating in the sinus, auricle, auriculoventricular node, ventricle, or bundle of His. Each may be differentiated as to its point of origin by modern instrumental methods and the commonest (*ventricular form*) at times, by the unaided sight.

Points of origin.

Extrasystoles may be rhythmic and persistent in their appearance or come at longer or shorter intervals between which the heart maintains a normal rhythm.

Means of Recognition.—They may be palpable at the wrist, absent at the wrist and present in a tracing, or detected only by the stethoscope, polygraph, or electrocardiograph.

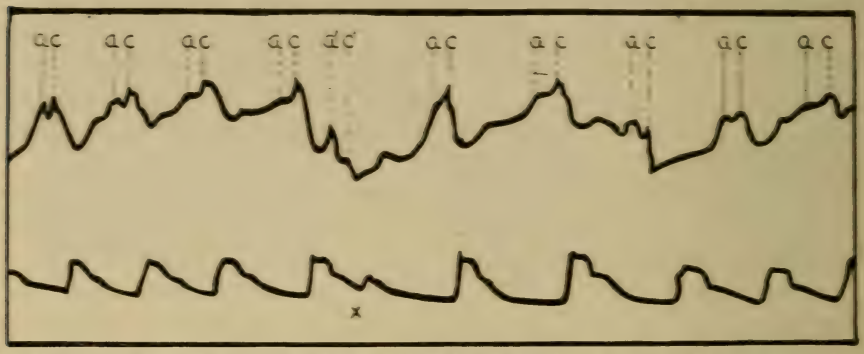


FIG. 240.—Simultaneous jugular and radial polygraphic tracing. At *x* a premature contraction indicated in the phlebogram above by the premature "a" and "c" waves is reflected in the radial tracing below. The auricular origin of the extrasystole is indicated by the presence of the "a" wave in the jugular tracing. (*After Parsons-Smith.*)

Auscultation.

Frustrated Contraction.—In most instances the first and second sounds associated with the premature contraction are both audible but in many the extrasystole is too weak to open the aortic and pulmonary valves and only the first element of such a "frustrated" heart contraction is heard. In such cases the pulse intermits and the single sound audible may usually be identified by the ear alone as a frustrated premature contraction. Only in

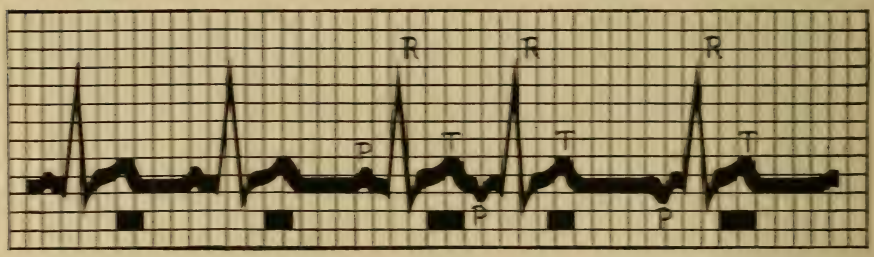


FIG. 241.—Single auricular extrasystole. Note that the pause following the premature contraction is less than two full cycles. This is characteristic of extrasystoles of the *auricular* type. Note also the inverted "P" both of the extrasystolic cycle and the one of normal *time incidence* immediately following. *The significance of the inverted* "P" lies in the proof it furnishes of an abnormal initial point of stimulation to auricular contraction. The auricular phase is not the result of a stimulus proceeding normally from the sinus node ("pacemaker," "node of Keith and Flack"), but is ectopic (displaced) in origin in both cycles showing the "P" inversion. An inverted "P" signifies an ectopic stimulus to auricular contraction. (*After Lewis, slightly modified and with lines of original deepened.*)

mitral stenosis with a protodiastolic third heart tone is a similar sound audible at the apex.

It is obvious that in certain instances of valvular insufficiency or stenosis

either sound may be lacking over its area of normal maximal audibility even though contractions are registered graphically.*

Intermittent Pulse.—It is evident that dropped pulse beats due to extrasystoles constitute a form of intermittent pulse.

Ventricular Extrasystolic Intermittent Pulse.—When occurring regularly after each full beat and followed by a full compensatory pause (paired beats with pause) and when strong enough to raise the aortic valve and become palpable at the wrist, they cause the full *pulsus bigeminus*. Extrasystoles of auricular origin show a pause slightly less than that of ventricular

Bigeminal pulse.

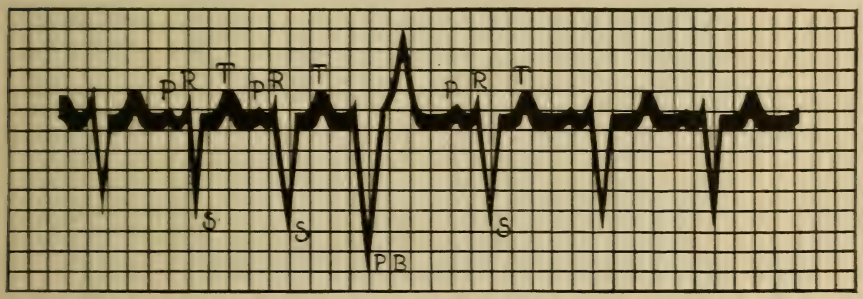


FIG. 242.—Extrasystole (P. R.) originating in left ventricle (lead III). In ventricular extrasystolic ("premature," "ectopic," "heterogenous") contraction, the wave of contraction spreads from the *abnormal* point of initial stimulus production to the other chambers of the heart, not from the sinus node and auricles. This results in a wide departure from the normal electrocardiographic complex representing the heart cycle or cycles affected. Obviously (a) the entire diphasic wave will be large and protracted, as compared with the normal and (b) the anticipated, rhythmically recurrent "P" wave of auricular activation is submerged or buried. As between extrasystoles of right and left ventricular origin respectively *it is known that an initial ascent of the "R" wave represents initial right ventricular stimulation; an initial descent, left ventricular initial stimulation.* In both instances the diphasic "R" wave is of exaggerated dimensions. The characteristic features then are: (1) The absence of the "P" wave from its normal position. (2) An exaggerated broad based "R" summit. (3) An "R" *peak* (upward deflection) in right ventricular extrasystoles; an "R" *cleft* (downward deflection) in left ventricular extrasystoles. (4) A *pause* equal to two complete normal heart cycles, by reason of the substitution of the extrasystole for one normal heart cycle and its interruption of the rest period of the preceding cycle. There are many variants in the form of double consecutive extrasystoles and very rarely two such successive premature contractions may arise in the right and left ventricles respectively as shown in one of the electrocardiograms taken by Dr. R. Edwin Morris (Figure 243). If ventricular extrasystoles are double, or if a premature contraction is auricular in origin, the compensatory pause is less than two full heart cycles.

form. It is obvious that the beat following the prolonged pause will be oftentimes of unusual amplitude because of the prolonged resting period.

Underlying Factors.—Aside from experimental stimulation, the extrasystole is a common event in cardiac disease, when the walls are dilated or the heart is working against high pressure.

Effect of overload.

A dilated and usually degenerated ventricle, in the presence of an excess of residual blood (imperfect emptying, overdistention), failing reserve power, and, perhaps, a coincident increased peripheral resistance, may become highly excitable and generate its own impulses independent of the pacemaker.

Toxemia.

* In a case of combined congenital and acquired heart disease examined by the author just before writing this note both the pulmonary and aortic tones were wholly lacking.

So also with an auricle overdistended by reason of an obstructed or insufficient mitral or tricuspid valve.

Yet again, toxic influences may similarly affect temporarily even the otherwise normal heart (digitalis, gastrointestinal subinfections, the causative agents of acute infectious disease and their toxins). It is also said to occur in the wholly normal heart, but unless it be due to excessive distention of the stomach or colon or in advanced pregnancy such a statement would seem at least debatable.

Clinical Significance of Extrasystoles.—*That these not infrequently occur in persons in whom no heart lesion can be* **demonstrated** *at the time admits of no doubt, nor can their presence and persistence be considered a grave symptom in the absence of other signs of serious cardiac impairment. They may exist for years*

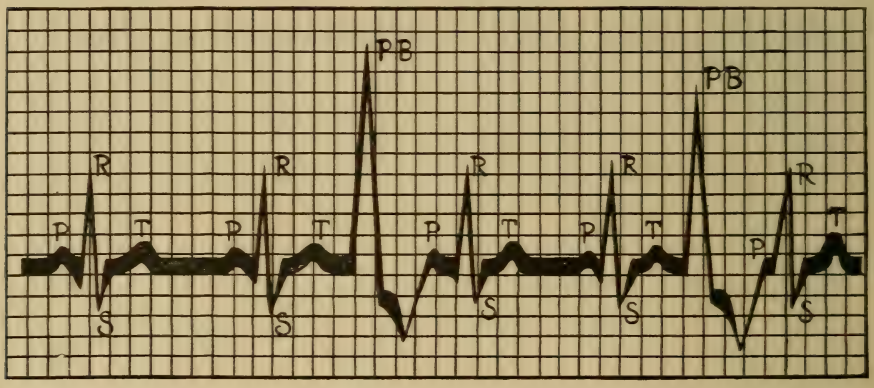


FIG. 244.—"Interpolated" right ventricular extrasystole. Compare with left ventricular systole (Fig. 242). The "P" of the perverted cycle is submerged in the exaggerated "R" wave in both instances. Note that the extrasystolic contraction of abnormal origin happens to fall in with the rhythm of the normal cycles. Hence, the absence of the usual compensatory pause. (*After Thos. Lewis. Lines of original deepened.*)

unattended by demonstrable gross myocardial disease, **but so may the commonest forms of myocardial degeneration.**

Miniature Murmurs.—If lesions of the valves exist and the strength of the extrasystoles is sufficient, murmurs in miniature may occur with premature contractions, save in the case of mitral stenosis.

Characteristic features.

Extrasystoles of Ventricular Origin.—These are most common and are characterized (*a*) by a remarkably constant time interval representing the period between the beat just preceding the substituted premature contraction and that regularly following the one which the extrasystole has anticipated, (*b*) by a large, single, jerky, venous wave which is the auricular wave of the anticipated systole acting against auriculo-ventricular valves closed by the extrasystolic contraction. This when present sweeps from the jugular fossa over the sterno-mastoid muscle.

Exception.—In bradycardia (slow pulse) if the premature contraction is very early the compensatory pause may be absent because the regular beat escapes full elision and takes its proper place in the cycle, time enough having elapsed between the ventricular extrasystole and the period of normal

nterpolated extrasystoles.

auricular excitation of the true contraction to enable the muscle fibers to partially recover from the "refractory stage." Such imperfect normally timed contractions are called "interpolated" extrasystoles but the term "interpolated ventricular contractions" is preferable *as they are not premature but correctly timed.*

Electrocardiographic Differentiation of Extrasystoles.—The electrocardiogram is characteristically altered in event of the presence of premature

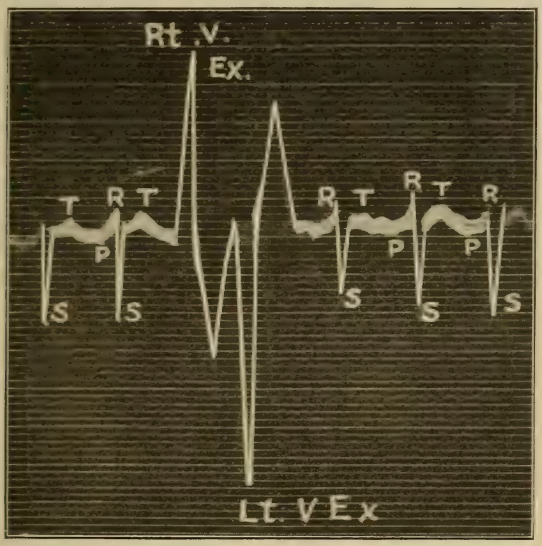


FIG. 245.—Extrasystolic tachycardia. Remarkable occurrence of one right and one left ventricular extrasystole in direct succession. (*R. Edwin Morris.*)

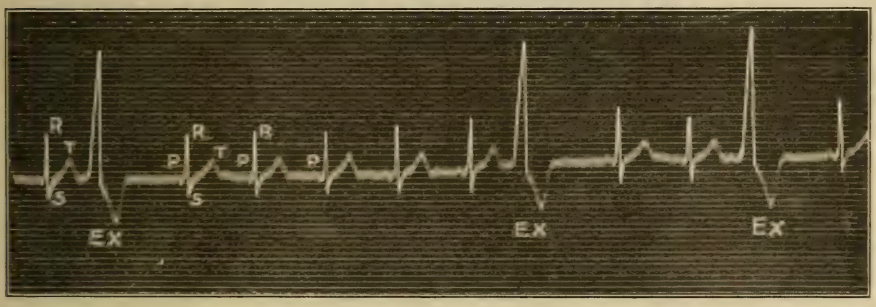


FIG. 246.—Right ventricular extrasystoles (ex). "R" summit high; sharp drop downward. Giant "*peak.*" Lead II.

Electrocardiogram, and polygram.

contractions and shows decided differences not only as to their auricular or ventricular origin, but also as between those originating in the right and left ventricle respectively.

Thus with lead II right ventricular extrasystoles are indicated by an extremely high "R" summit followed by a deep "S" descent.

Left ventricular extrasystoles are indicated by an extreme dip "Q" preceding "R" which is also decidedly higher than normal. In lead I the

characteristic deflections, with rare exceptions, are reversed, *i.e.*, they are mirror images of the deflections encountered in leads II and III. (See Electrocardiographic section for further discussion).

The Auricular Extrasystoles.—These invariably produce ventricular response in the form of a premature contraction and show usually "shortened bigemini" in the pulse waves if the extrasystoles are carried through to the wrist, inasmuch as the so-called "compensatory" pause rarely or never is

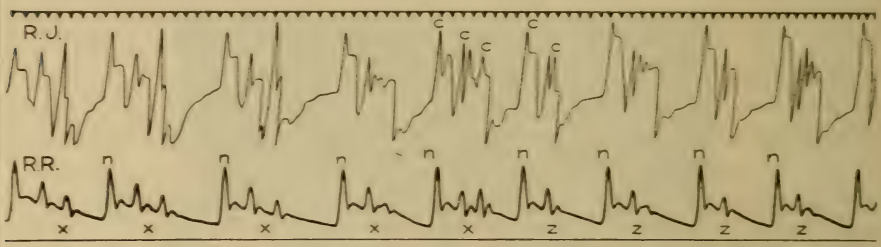


FIG. 247.—A beautiful example of ventricular extrasystolic arrhythmia showing pulsus bigeminus (z) and trigeminus (x) together with the typical corresponding jugular tracing. (Right jugular above; right radial below.) "n" indicates the normal radial beats. (*R. Edwin Morris.*)

quite complete.* If they originate in the neighborhood of the great veins or sinus node, the pause is that of the normal rhythm. They are associated with a *double* jugular wave in the polygraphic tracing and the electrocardiogram shows an inverted imperfect or superimposed auricular complex ("P" wave), the last being due to so premature an impulse as meets and coincides with the "T" of ventricular systole in the electrocardiogram.

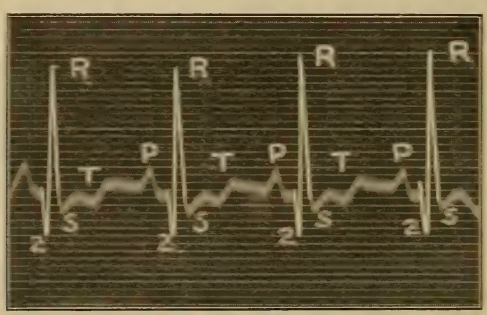


FIG. 248.—Inverted "T" in an *apparently* normal heart. (*R. Edwin Morris.*)

It is obvious that extrasystoles of shortened pause are of auricular origin and that a pause reduced to that of the normal rhythm indicates the sinus region as the point of origin.

Extrasystoles from Bundle of His.—In this type the auricular and ventricular systoles may or may not fall absolutely together. These are recog-

* Lewis believes that they are in some instances fully compensatory but he says that "if in the case of any premature contraction the pause fails to be compensatory then such a (premature) beat has arisen in the auricle" adding in a footnote that the interpolation cases are excepted.

nized with difficulty in polygraphic tracings by their prematurity and the wholly absent or very short conduction time as shown by the reduced or absent "*a-c*" interval. In the electrocardiogram they show, when typical, an undisturbed ventricular complex and a buried auricular complex. The typical electrocardiogram is by no means constant.

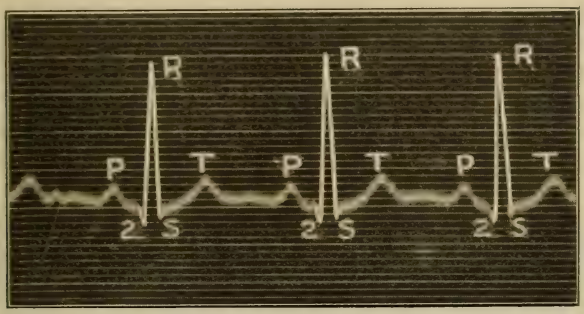


FIG. 249.—Normal electrocardiogram inserted here for contrast and comparison.

Clinical Significance of the Auricular and Ventricular Extrasystole.—*Premature* **auricular** *contractions* are most frequently encountered in primary myocardial degeneration and in mitral stenosis.

Premature **ventricular** *contractions* also occur most frequently in myocardial degeneration alone or as associated with aortic and mitral lesions.

Common associations.

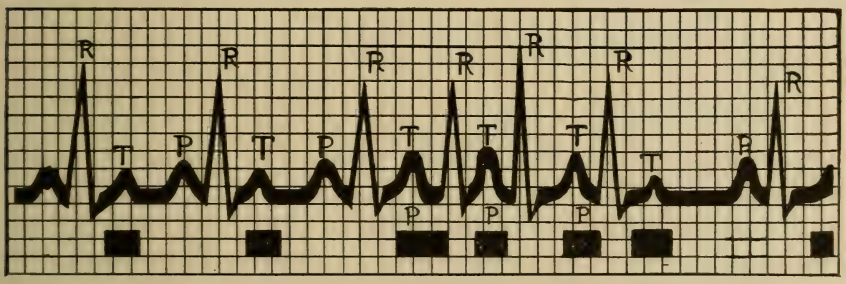


FIG. 250.—Auricular extrasystoles Note three successive premature contractions In each case the "P" phase is buried in the "T" of the preceding cycle. This is indicated by the combination of extrasystolic cycles with a marked increase in the amplitude of the "T" summits of the abnormal cycles. In the original plate of Thos. Lewis *the jagged "P" apex characteristic of mitral stenosis* is present, not alone in the cycles of normal rhythm, but carried also into the "T" summits which bury the "P"s of the extrasystoles. This variant is known as the split "P" or mitral "P" and is beautifully shown in some of the actual electrocardiograms reproduced elsewhere in this section.

Many instances are seen in marked coronary sclerosis, Bright's disease and arteriosclerosis in general. As regards the ventricle affected, it is obvious that in most instances the lesions primarily and chiefly tending to structurally cripple, overwork and exhaust a particular heart chamber will be associated with extrasystoles originating in that division of the heart, if such premature contractions exist at all.

Proper viewpoint.

As previously stated, persistent extrasystolic irregularities may occur in persons apparently healthy, but the student should not lose sight of the fact that Thomas Lewis has reported but three such instances out of a group of ninety-seven cases investigated, and furthermore, that of the remainder seventy-one cases were definitely those of cardiovascular disease.

Sign of overstrain.

*Extrasystoles are probably to be regarded precisely as one should view heart lesions in general, viz., as conditions not of themselves and of necessity proving the presence of a dangerous or threatening condition but as morbid phenomena strongly suggesting toxemia or structural deterioration and overload and as such always demanding a thorough and painstaking investigation of conditions.**

Certainly premature contractions do not often occur *persistently* in a heart which is sound, fully competent, and well nourished and the author

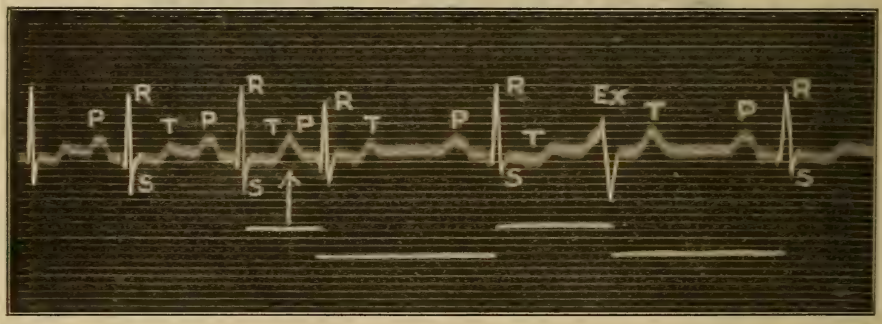


FIG. 251.—Auriculo-ventricular (junctional) extrasystole; originating in junctional tissues. A long pause follows, then a normal cycle, then a right ventricular extrasystole. Mitral stenosis is indicated by the "P" summits (truncate or split) and the effect of digitalis administration by the constantly inverted T.

has rarely encountered it save in decided toxemias and intoxications, frank cardiac or renal disease, or the overstrained, undersized, flabby, and insidiously dilating hearts of congenitally asthenic and nutritionally depressed or unstable individuals.

Prognostic value.

Furthermore, the onset of marked extrasystolic irregularity in known cardiopaths, formerly free from it, may be an ominous symptom and its disappearance under treatment a favorable sign.

Useful Maneuvers.—It should be remembered that holding the breath, manual or postural increase of intra-abdominal pressure, and the erect posture will often bring out any latent tendency to extrasystolic beats. On the other hand, the *abrupt* assumption of the erect posture, exercise and even excitement or fear may temporarily abolish such an arrhythmia even if present primarily.

A pulsus alternans is occasionally present in connection with them, and irregular extrasystolic beats, or in fact any of the extrasystolic phenomena

* The author has seen many cases of long-enduring extrasystolic irregularity, but rarely or never in persons presenting a perfectly clean bill of health. It is quite possible that in Great Britain toxic cases, of gouty origin, are especially frequent, which assumption may account for the optimism of so distinguished an authority as Mackenzie.

are usually associated with a relatively slow or bigeminal *radial pulse* by reason of the frustrated beats.

Their abrupt disappearance when instrumental recording is attempted is one of the trials often encountered by the medical investigator.

AURICULAR FIBRILLATION (*Delirium Cordis*).—In this important condition there is a veritable riot of abnormal futile and abortive contraction

Fibrillary flickering.

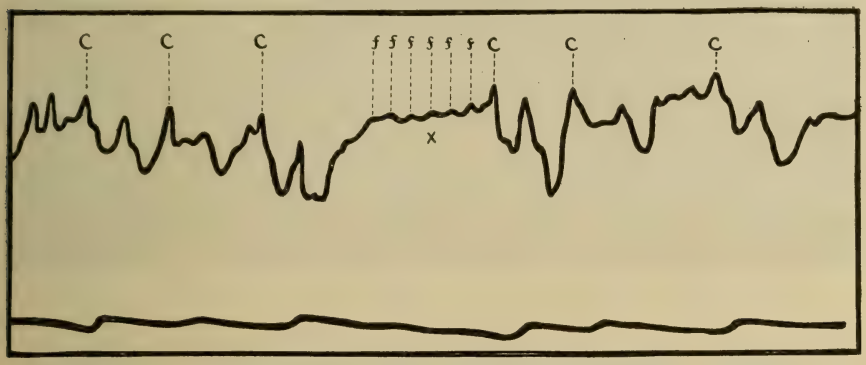


FIG. 252.—An extremely well-marked case of auricular fibrillation. The normal "a" wave of auricular systole is wholly lacking, and replaced by small diastolic undulations (f f f).

waves in the auricle, the result being a continual fibrillary flickering of the muscle and an entire loss both of effective rhythmic transmission and coördinated muscular contraction.

Delirium cordis.

Myriad impulses of the most feeble, irregular, hasty, and imperfect sort, crowd upon the ventricles at a rate far exceeding the possibilities of complete transmission, and such as can achieve transmission excite these chambers to

Blocking and unregulated escape.

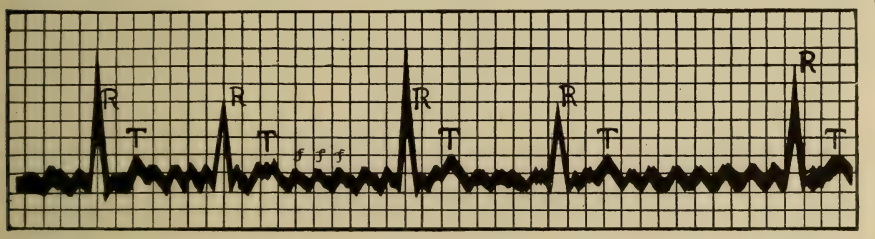


FIG. 253.—Auricular fibrillation. Note very decided oscillations (f f f) due to fibrillation; evidence of absolute irregularity and inequality shown in the varying height of "R' summits and spacing of cycles; absence of "P" in every cycle, indicating lack of efficient auricular impulses to contraction. *Pulsus irregularis perpetuus or delirium cordis* evident. Record taken over auricles. (*After Thos. Lewis.*)

rapid, irregular and unequal contractions which are nevertheless, *in toto*, sufficient in many instances to maintain a circulation of some effectiveness over *long* periods.

The auricle itself is meanwhile dilated in the diastolic position and is as utterly unable to maintain an efficient contraction of its own musculature, as it would be in a state of actual paralysis.

Polygram and electrocardiogram.

As a result the normal presystolic venous pulse (represented by the "a" wave of the polygram or "P" wave of the normal electrocardiogram) is faint, lacking, or substituted by a *systolic* wave in the jugulars, the feeble auricular flickering may be revealed as an undulating line in the polygraphic tracing, and in the electrocardiogram by a complex in which the auricular

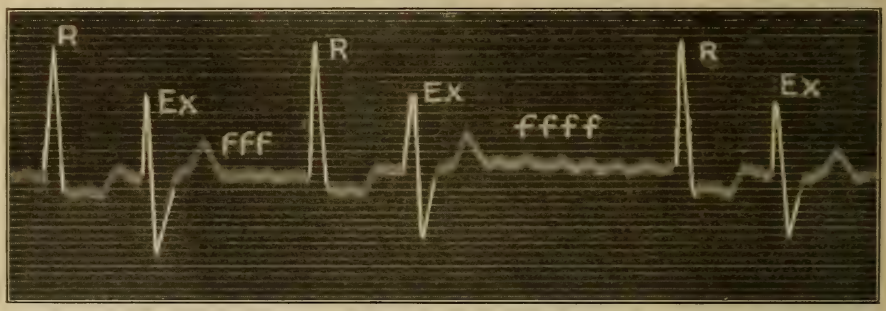


Fig. 254.—Auricular fibrillation; right ventricular extrasystoles. Auricular fibrillation (f). Right ventricular extrasystoles (Ex). Pulsus bigeminus was evident in the rhythmic pairing of a normal beat with an extrasystole. Lead I. Time ⅕ sec. (*R. Edwin Morris.*)

(presystolic) "P" wave is absent and represented only by this rippling undulation and a hurried, irregularly spaced and unequal ventricular complex ("QRS").*

A clinical paradox.

If in a case of auricular fibrillation the bundle of His (*atrioventricular conduction bundle*) be normal, the heart rate is doubled or trebled, though the ventricular contractions are haphazard, the pauses of varying length and

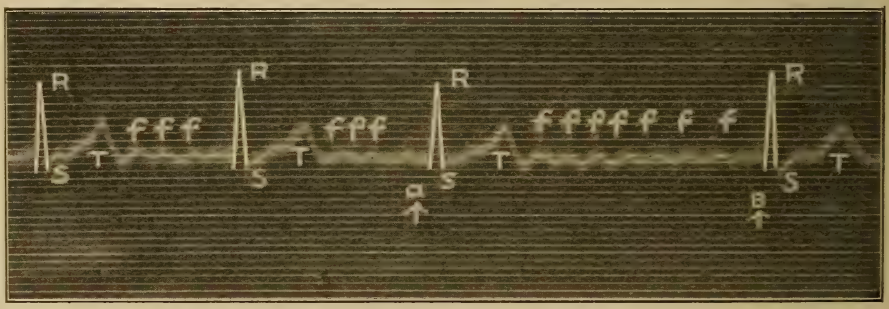


Fig. 255.—Auricular fibrillation. (*R. Edwin Morris.*)

the beats unequal. If, however, the conduction bundle is diseased, a block may be evident and the pulse may drop to 40 or less per minute.

In all pulses of less than double the normal rate and showing the other characteristics of fibrillation, pronounced digitalis effect being excluded, heart block from other causes may be assumed as a factor.

The commoner pulse rate in auricular fibrillation is about 120 *to* 140, *and nearly all markedly irregular pulses of this rate result from this condition.*

* Unless heart block is present.

The efficacy of digitalis is peculiarly marked in most but not all such cases in which the pulse rate equals or exceeds 120.

A careless observer may overlook fibrillation with slowed pulse; indeed, in some cases its presence cannot be positively affirmed without instrumental aid.

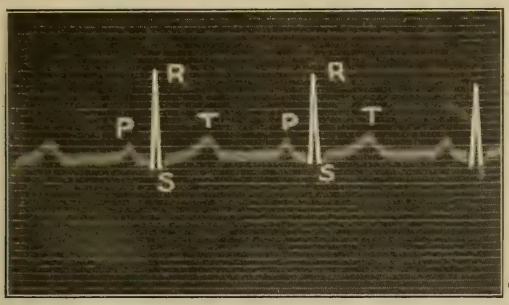


FIG. 256.—Normal electrocardiogram inserted here for purposes of contrast and comparison.

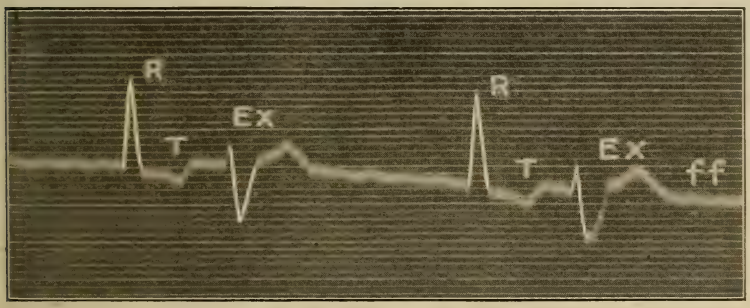


FIG. 257.—Lead I.

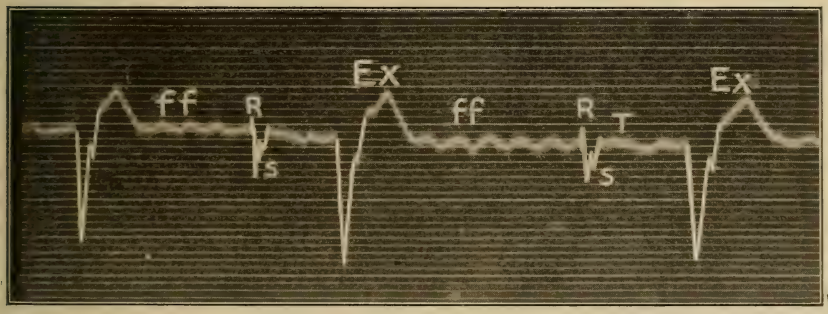


FIG. 258.—Lead III.

FIGS. 257 AND 258.—Auricular fibrillation with left ventricular extrasystoles. Left ventricular hypertrophy is indicated by large "R" and absent or small "S" in lead I, and small "R" and large "S" in lead III. Fibrillation is evident (fff); "P" (of auricular contraction) is absent; T is inverted from digitalis effect.

It has been said that angina pectoris and fibrillation cannot coexist and the onset of the former condition and its persistence completely removes the tendency to anginal attacks.

In the light afforded by recently reported cases, one must assume at least this rule is subject to exceptions.

Important fact.

Any considerable exertion or even quick change of posture such as so often suffice to temporarily remove or to bring out extrasystolic irregularity represent maneuvers which are not without risk in severe incompensation, and this is especially true of cases showing the *delirium cordis** of auricular fibrillation.

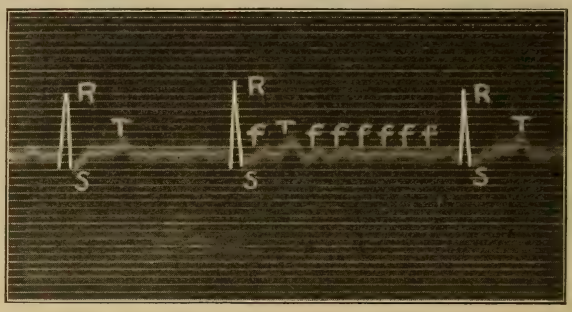


FIG. 259.—Auricular fibrillation. The absence of all evidence of auricular contraction is manifest. Fibrillary wavelets are shown (f f f+).

Frequency of occurrence.

Cases of auricular fibrillation constitute over 40 per cent. of the total arrhythmias encountered in a general hospital and it is most commonly associated (50 per cent. of cases) with mitral stenosis. It is common in other conditions in which *myocardial degeneration*, cardiac decompensation and prolonged chronic overstrain are marked features, *i.e.*, advanced myocardial degeneration alone (20 per cent.), aortic lesions, chronic diffuse nephritis with arterial hypertension or arteriosclerosis.

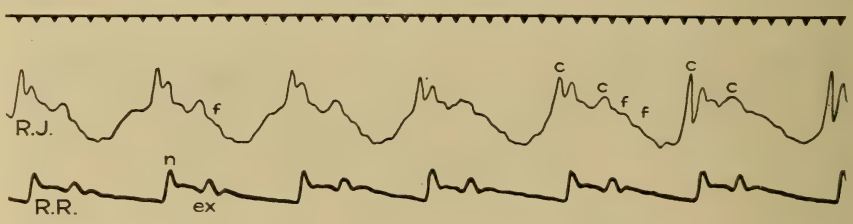


FIG. 260.—Arrhythmia, *pulsus bigeminus* of extrasystolic type; fibrillation. n, Normal beat; Ex, l.v. extrasystole producing bigeminal pulse; f, fibrillation waves from auricular fibrillation; RJ, jugular pulse; R, radial. Time 1/5 sec.

In all of these the myocardial change is doubtless the chief factor and in a public clinic nearly three-fourths of those specifically cardiovascular indoor cases admitted who show severe incompensation and associated arrhythmia will

* The secondary slowing of the pulse which follows the primary acceleration induced by exertion is usually lacking in this condition but, if present, *decreases* fibrillation, though it usually increases other arrhythmias (Lewis).

show auricular fibrillation. (In the author's clinic at the University Hospitals nearly 90 per cent. were of this type.)*

Unquestionably a great number of unrecognized temporary and even fleeting attacks of fibrillation occur and, no doubt, there is a tendency to more frequent recurrence and longer persistence of the individual attacks prior to its appearance in an established form.

Once fully developed, it tends to persist throughout the after-lifetime of the victim but this rule also is subject to exceptions.

As in the case of auricular flutter, embolic manifestations occasionally follow the resumption of normal rhythm.

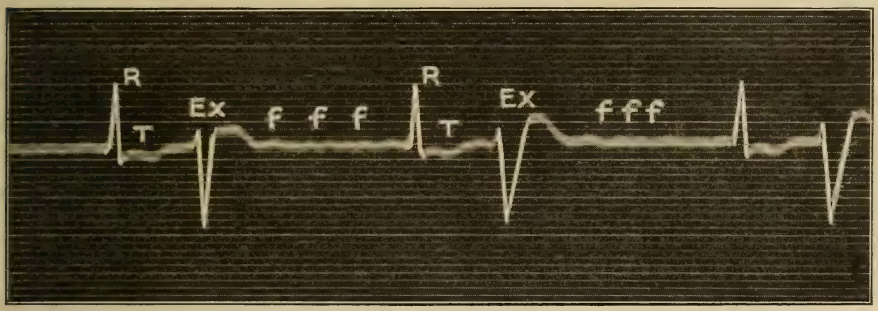


FIG. 261.—Electrocardiogram. Same case as Fig. 254. R, normal "R" wave; Ex, extrasystole, ventricular type; f, fibrillation; time $\frac{1}{10}$ sec. showing exact nature of extrasystole in Fig. 254 and absence of "P" wave and presence of fibrillation "f".

AURICULAR FLUTTER.—*This term is of recent introduction and covers cases of suddenly initiated, excessively rapid auricular contraction exceeding* 200 *to the minute and reported as high as* 420.

It may continue for months or years only to cease as abruptly as it came or occur in brief attacks lasting for a few hours. The pulse rate is usually one-half that of the auricular contractions but various degrees of block are

Duration.

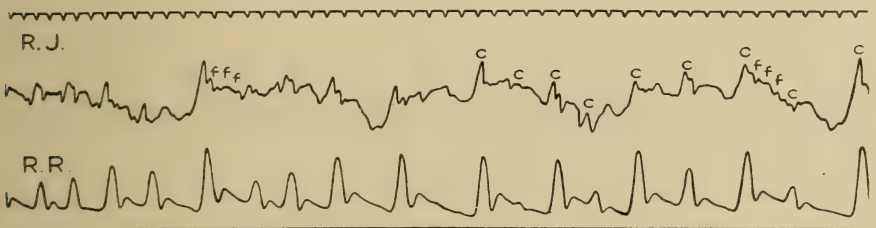


FIG. 262.—Auricular arrhythmia due to auricular fibrillation. The cause of the greater proportion of cardiac arrhythmias. "f f" indicates auricular waves of fibrillation. Upper line jugular, lower line radial. Time $\frac{1}{5}$ sec. The "*Pulsus irregularis perpetuus*" and "*delirium cordis*," are beautifully shown in the radial.

manifest and the ventricle may respond to only the third, fifth or eighth auricular contraction.

It not infrequently passes into auricular fibrillation spontaneously or as a result of digitalis administration (Lewis) under which the existing block is

*The most responsive cases of fibrillation from the therapeutic standpoint seem to be those in which it is initiated by unusual effort or acute toxemia. (*R. E. Morris.*)

Digitalis phases.

heightened purposely, the ventricular rate falling first with irregularity, then into a regular rhythm of lower rate; then into fibrillation. At this point the drug is discontinued and in "a large proportion of cases" the regular heart action returns and any preëxisting cyanosis, engorgement or dropsy "vanish quickly" (Lewis).*

The occasional occurrence of embolic manifestations following the resumption of a normal rhythm is a fact of clinical interest and importance.

Suggestive resemblances.

Apparently it is closely allied to paroxysmal tachycardia. It is frequently associated with alternation. It is next neighbor to auricular fibrillation and may pass into that condition spontaneously, but nevertheless is still dealt with as a distinct clinical phenomenon.

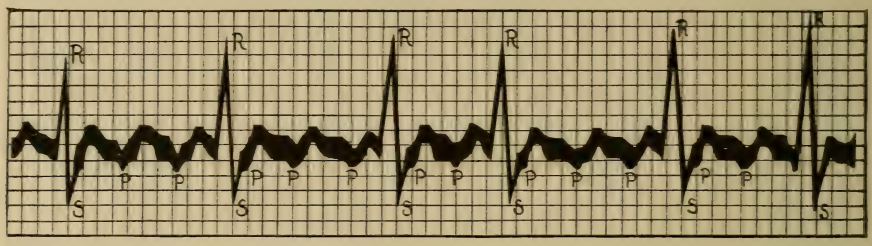


FIG. 263.—Case of auricular flutter. Auricular contractions were 310 per minute; ventricular rate, 120.

Relatively uncommon.

Frequency and Causation.—It is a relatively uncommon condition† and its causes are essentially those described under auricular fibrillation save that it more distinctly affects the middle-aged or elderly and seems especially common in cardiosclerotic degeneration.

Diagnosis of Auricular Flutter.—*The electrocardiograph alone furnishes constant and positive diagnostic results, yet one may in some instances make a correct diagnosis by other means.*

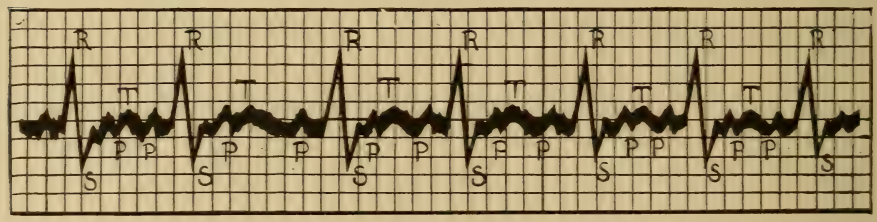


FIG. 264.—Auricular flutter. The ventricle is responding only to alternate auricular stimuli. Auricular rate, 324 per minute. (*After Thos. Lewis.*)

The Venous Pulse in Auricular Flutter.—In some cases the condition is clearly indicated by visible excessive rapidity of the venous pulsations or the multiplicity and rapidity of "a" waves in the polygraph. The auricu-

* The frequency of failure of such medication to produce this result would seem to have been somewhat underestimated. Certainly many cases are wholly unaffected.

† Thos. Lewis and Mackenize find it common, but in the author's clinic it was encountered very rarely unless one followed Satterthwaite's dictum, which declares all countable auricular contraction rates above 200 to be "flutter."

FIG. 265.—Passage from flutter and state of general arterial anemia and venous stasis or fibrillation. Profound cardiac decompensation. (R.J. = right jugular. R.R. = right radial.

1. "Running" pulse barely palpable; rate 168 per minute; jugular waves enormous.

2. After 24 hours treatment with digitalis by Eggleston's method. Radial now presents a definite pulse of 156 beats per minute. Jugular less intense but lacking "a" waves.

3. Twenty-four hours later. Radial shows clearly the characteristics of *fibrillation* ("*pulsus irregularis perpetuus*"); "a" waves wholly absent from venous tracing. Pulse rate 132.

4. Twenty-four hours later. Pulse rate 108; less arrhythmic; fibrillation clearly shown by minute waves (f f+) in jugular tracing.

Functional block.

lar pulsations are so rapid as to surpass the conduction capacity of the His bundle or the possibilities of recovery and response on the part of the ventricles, and, therefore, save in the rarest instances, such cases show a relatively low pulse rate; 240 or even 400 aurciular contractions to but 60 or even 30 ventricular beats being present. If the block, or as one might say, the inadequacy of the conducting bundle, is less marked, the rhythm may be and usually is definitely 2–1, in which case, of course, a rapid radial pulse is present.

The polygraphic tracing may show clearly the excessively rapid production of distinct or even large "a" waves occurring in the various auricular-ventricular ratios common to the "block" usually present in this condition.

This statement applies chiefly to cases in which the arterial pulse is distinctly slow, and the veins of the neck are yielding good auricular pulsations.

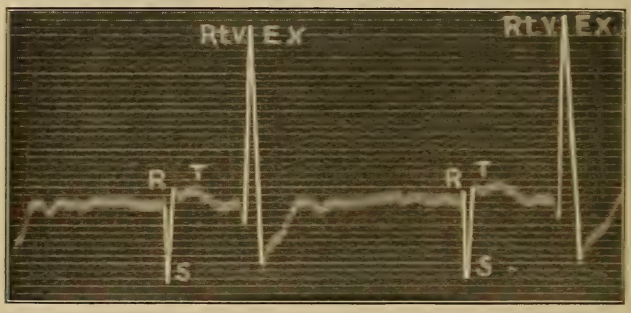


FIG. 266.—Same case three months before. Electrocardiogram shows fibrillation and right ventricular extrasystoles.

Occasional excessive ventricular rapidity.

If, as occasionally happens, the auricular rate is under 300 and the ventricle responds to every auricular contraction, an uncountable, excessively weak radial pulse results, together with symptoms of profound incompensation, syncopal attacks, or coma and death.

The electrodiagrams show the same characteristics and it is usually clearly distinguished from auricular fibrillation by its lack of the extreme erratic variability both in the pauses and in the heights of the "R" summits characteristic of its congener.

Paroxysmal tachycardia and alternation.

The equality in time, large and uniform size and regular occurrence of the "flutter" waves are points of importance in differentiation.

Under such conditions the pulse characteristics of paroxysmal tachycardia are actually present and, very frequently, the excessively rapid beat often shows distinctly an alternating strength and weakness (pulsus alternans) if instrumentally recorded.

Distinguished from fibrillation.

Symptoms.—It will be noted that the extreme irregularity of interval so marked in fibrillation is ordinarily absent in these cases, the ventricular response being usually, but by no means always, rhythmic. As in paroxysmal tachycardia these cases may show arterial anemia and venous stasis.

Recurring periods of high pulse rate of which the patient is conscious,

lasting for days or weeks, attended by distinct evidences of cardiac decompensation, yet unlike the wholly delirious arrhythmia of fibrillation, vertigo or actual unconsciousness attending the onset and the frequent initiation of the attacks by slight exertion, are among the clinical evidences of the condition given by Thomas Lewis and Mackenzie.

It is obvious that they are merely suggestive, and unfortunately true that pulsus alternans and irregularity may render the pulse most puzzling.

The cases of regular 2–1 block would be the ones most likely to be guessed correctly, but rational non-instrumental diagnosis would seem to be dependent chiefly upon the pulse phases induced by digitalis therapy as previously described.

Duration.—Auricular flutter like auricular fibrillation may occur in short paroxysms or endure for months, but during any decided seizure, marked and progressive symptoms of decompensation become manifest.

Decompensation produced.

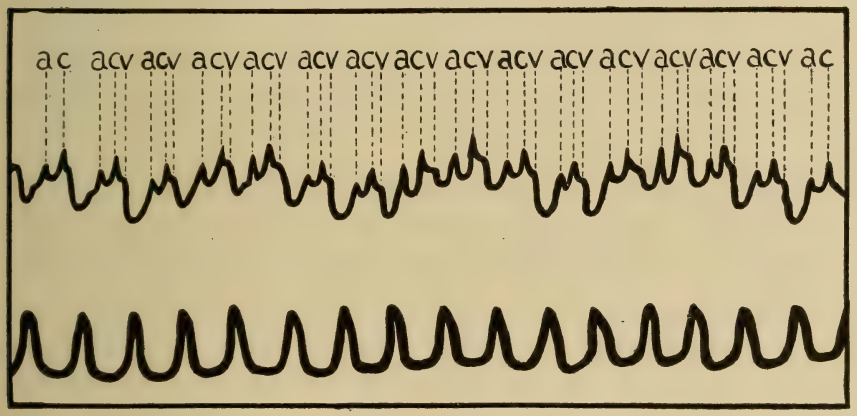


FIG. 267.—Mere febrile tachycardia. Normal cycle throughout in venous tracing. Rapidity only.

Physiologic Heart Block.—Inasmuch as the normal conduction time is nearly one-fifth of a second (0.12–0.18 sec.), it is evident, as previously suggested, that a certain degree of functional heart block must occur in this lesion when the auricular flutterings exceed 300 although it has no such significance as has the heart block due to organically impaired conduction.

PAROXYSMAL TACHYCARDIA.—This curious speeding up of the whole heart apparently represents the *sudden* appearance of a few auricular extrasystoles initiating excessively rapid *rhythmic*, but inefficient heart beats of auricular origin in place of the slower rhythmic stimuli normally arising from the pacemaker (sino-auricular node).*

Excessive rapidity and weakness.

Arise in auricles.

In rare instances the impulse to contraction seems to arise in the ventricles.

The rate is usually about double that of the normal heart.

Essential Features.—*It consists clinically of the abrupt onset of a series of excessively rapid beats*, their equally abrupt cessation after a period varying

* Paroxysmal trachycardia of ventricular origin is extremely rare. In some instances it would appear unlikely that any extrasystolic phenomena initiate the attacks.

Abrupt onset and ending.

from a few seconds to several hours or weeks, a somewhat prolonged pause, and the resumption of a normal rate.

The subjective cardiac distress is primarily slight though curiously alarming to the patient. Weakness or oftener a sense of profound exhaustion adds to his apprehension.

In a case observed with especial minuteness by the author the attack is not initiated by obtrusive or subjective recognizable extrasystoles although the onset is sudden and definite. An immediate limitation of effort is obligatory. If this cannot be secured anginal pain of moderate or even somewhat severe grade results.

This is manifested first in the precordium, then the left arm, and finally by radiation to the left side of the neck and jaw.

Until habituated through the frequent recurrence of attacks a "sense of impending death" was experienced in the painful seizures.

The patient feels convinced that the heart would dilate rapidly under any decided physical exertion, *i.e.*, he "senses" the extreme overload present as a result of the quickened rate.

The tendency to attacks may pass into auricular flutter or fibrillation, wholly cease for long periods or occur persistently several times a day or at any other interval.

In many cases an acute cardiac insufficiency is developed in a few days or even hours. The heart dilates widely, general edema appears, orthopnea is present, the liver enlarges and decided passive congestion of the kidneys is manifest.

Vagus inhibition lost.

Vagus control is almost or quite removed throughout the period of tachycardia. (Vagus pressure slows the ventricular beat in these cases by creating a partial vagus block, but without affecting the abnormal auricular contractions in the slightest degree.)

Sometimes beyond counting

The beats may show decided inequality and the rate varies from 120 to 200, the average rate being somewhat more than double the normal.

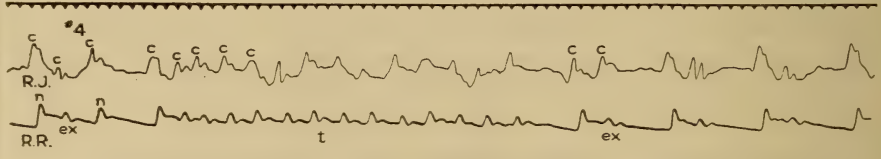


FIG. 268.—Paroxysmal tachycardia. The abrupt onset and cessation of the brief paroxysm and its equally sudden relapse into extrasystolic irregularity (Ex) and "*pulsus bigeminus*" are clearly shown. In the full-sized tracing, alternation seems to be present during the paroxysm. (*R. Edwin Morris.*)

The lack of the subjective sense of violent palpitation and the fact that exercise during the attack does not increase the rate (Mackenzie-Lewis) stand in sharp contrast to the symptoms of simple acceleration (palpitation) or a simple tachycardia of exertion or excitement.

Electrocardiogram.

The electrocardiogram demonstrates clearly in most instances the auricular origin of the aberrant impulses, the "P" wave of the paroxysmal

cycles and that of the introductory auricular extrasystoles being identical in form. Rarely it is initiated by a ventricular extrasystole.

In cases of excessive rapidity the "P" wave may be merged in the "T" wave or wholly obscured by it.

It occurs at any age save the first decade but with significant frequency between the ages of twenty and forty, the selective rheumatic period, and even more frequently between forty and sixty, the preferential period of the myocardial degenerations. Age groups.

It is most common in myocardial degeneration, is often present in mitral stenosis, and has a decided pathologic significance though occasionally present without other *distinct* signs of cardiac disease. In severe cases the rhythm is essentially fetal (the "*tic tac*" rhythm). "Tic-tac" rhythm.

To the author it would seem wiser to consider this condition clinically as one standing wholly apart from "flutter and fibrillation."

ALTERNATING PULSE.—This represents usually very rapid rhythmic alternation of stronger with weaker beats, the variation and force being often imperceptible to the finger. It is best shown by the radial and carotid tracings, and is sometimes beautifully clear in the electrocardiogram. Impaired contractility

It will be noted that the beats fall at regular intervals, the abnormality being one of force only.

It is especially common in cardiosclerosis, the terminal cardiac insufficiency of interstitial nephritis and coronary sclerosis, and is frequently associated with *paroxysmal tachycardia* and with *auricular fluter*.

In practice its immediate seriousness is largely dependent upon the question of attendant pulse rate. Prognostic factor.

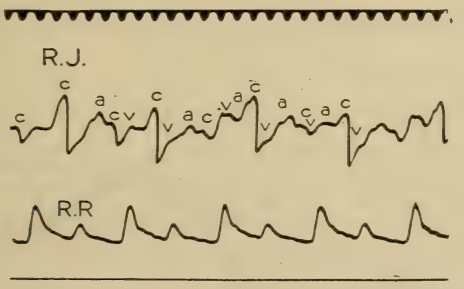


Fig. 269.—Pulsus alternans. Rhythmic pulse, a.c.v. waves present in jugular tracing. Upper tracing jugular, lower tracing radial; time ⅕ sec.

Significance of Associations.—If the alternation occurs in mere rapid heart (simple tachycardia) such as may arise temporarily from trivial causes and subside upon their removal, it may or may not indicate myocardial disease or overstrain. If associated with paroxysmal tachycardia the significance and associations of that form of pulse acceleration, which is probably a manifestation of "auricular flutter" dominate the clinical picture. Rapid hearts.

If it occurs in a heart beating at, or but slightly above, the normal rate, and especially in persons of middle or advanced age, or in connection with known cardiac disease, it is of serious significance. Moderate rate.

Induced by exercise.

The same is true of cases in which alternation occurs under exercise or sudden changes in position with but moderate resulting acceleration of the heart beat.

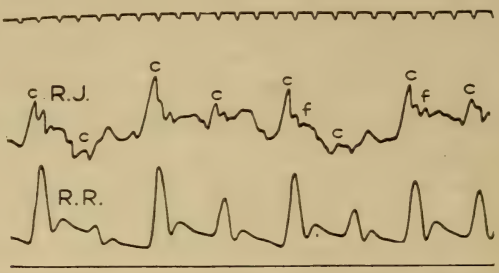


FIG. 270.—Pulse resembling pulsus alternans, though smaller pulsations are due to extrasystoles. Auricular fibrillation shown by waves f f. Upper tracing jugular, lower tracing radial. Time 1/5 sec. (*R. Edwin Morris.*)

It may be confined to but a few cycles, especially in the presence of occasional extrasystoles.

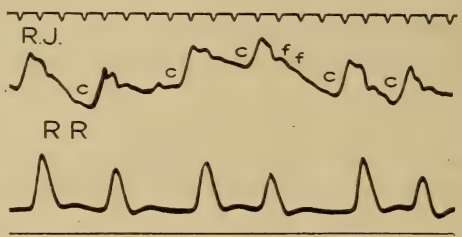


FIG. 271.—Auricular fibrillation. Pulse simulates alternans on this half of original tracing, although the spacing is markedly irregular when instrumentally demonstrated. Left one-fifth of tracing, not reproduced here, showed a bigeminal pulse due to extrasystolic arrhythmia. Fibrillation was manifest in both phases (f f++). (*R. Edwin Morris.*)

A "herald of death."

Prognostic Significance.—*Occurring either under the conditions of moderate rate, on the one hand, or excessive paroxysmal rapidity, on the other, as the*

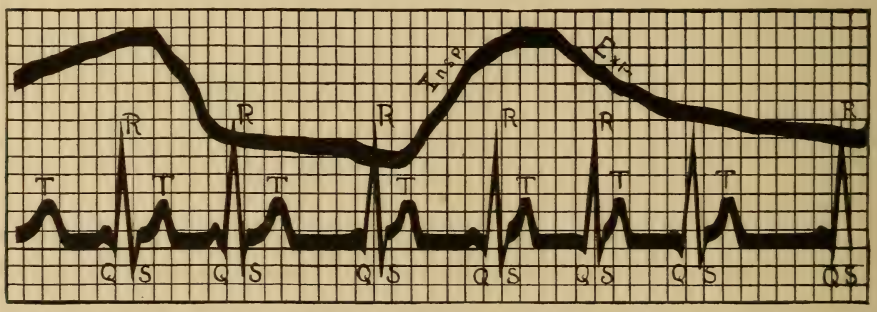


FIG. 272.—*Sinus arrhythmia.* Electrocardiogram and respiratory curve. (*After Thos. Lewis.*)

result of exercise, or in connection with extrasystoles, the alternating pulse is a serious condition representing extreme exhaustion of reserve and impaired effective response, and ranking, as Lewis says, with "risus sardonicus," "subsultus tendinum" and optic neuritis as heralds of a death at best not many

months distant. In fact it is often actually associated with such urgent and ill-omened conditions as "Cheyne-Stokes breathing," cardiac asthma, and severe angina pectoris, though as in these conditions the possessor may sometimes live many months or even years.

SINUS IRREGULARITIES.—The vagus nerve controls the "pacemaker" of the heart (*sino-auricular node*), normally reduces by over one-half the rate of the unrestrained-heart stimuli, and probably determines their point of departure from the sinus area.

Vagus influence.

Partial inhibition of the vagus influence is evident in the quickened heart (tachycardia) associated with fear, joy, pain and a host of other emotions, no less than from the action of many well-known drugs. The more unstable the nervous system or psyche of the individual, the more readily are trivial sights, sounds, odors, aches and trials magnified and made effective inhibitors of vagus control. So also may the inspiratory quickening of rate be exaggerated under conditions or toxemia, poor nutrition or psychasthenia.

The known independence and automatic irresponsibility and lawlessness of the heart muscle under certain conditions of overstrain and valvular and myocardial disease largely vitiates the vagus control.

Characteristic Features of Sinus Irregularities.—*These are characterized by a varying length of cardiac cycle represented by a diastolic variation but unassociated with dropped beats, alternation, delirium cordis or extrasystoles, though peculiarly unstable in rhythm and often responsive to and modified by, the phases of respiration.* Merely taking one's pulse during forced inspiration, a long pause and forced expiration demonstrates this common "juvenile type of irregularity." The rate of the beats tends to wax and wane, to speed up and gradually slow down in series.

A juvenile type.

A simple demonstration.

In many the respiratory response is absent but all show the tendency to complete orderly systole, varying diastole, equality of force in the beats, and the serial waxing and waning of the rate.

Significance.—*Sinus irregularities* are usually of slight significance and in the respiratory form constitute the great majority of the arrhythmias of childhood and youth and of neurotic, congenitally asthenic, unstable, undernourished or mildly toxic individuals.

Furthermore, if the pulse is sufficiently quickened by exercise, excitement, or more radically, by full doses of atropin, which usually lifts the vagus influence to a marked degree within an hour, arrhythmia is often modified or wholly set aside.

Tests of exercise and atropin.

Instrumental methods at once define them clearly for, whatever the rate of any series, the graphic auricular and ventricular complexes are wholly normal and the variable diastolic phases are obvious.

It is an arrhythmia of perfect physiological complexes occurring in normal sequence, abnormal only in the spacing of the rest periods.

It is obviously a sinus effect in every instance for it is *space* only that is affected save in the dropped beat type of "sinus block" and here the defect lies in the failure of the *sinus* to release its stimulus.

Always a sinus effect.

Simple Bradycardia.—If, on the other hand, vagus inhibition be excessive, a slow pulse develops (50 to 55 beats per minute) which in itself lacks

Import ant possibilities.

serious significance though it may occur in paralysis of the sympathetic, direct vagus pressure from any cause, from poisoning by adrenalin, digitalis, or lead, or in association with intracranial pressure, acute infections, asphyxia, icterus, convalescence from acute infections, profound toxemias, arterial

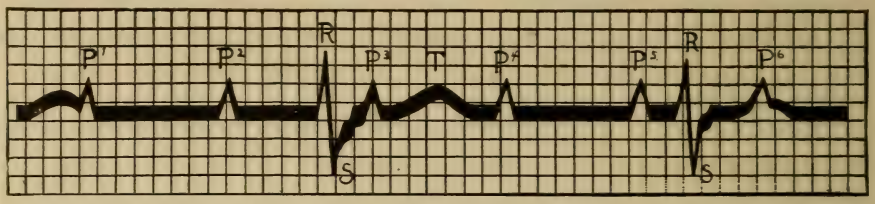
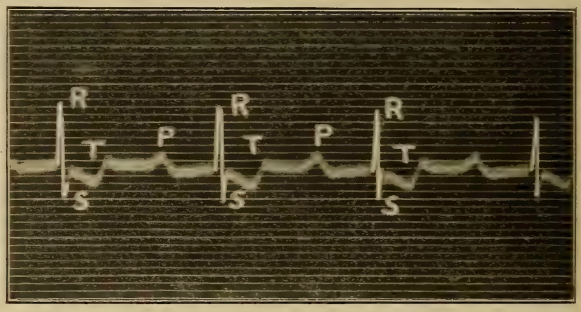


FIG. 273.—Complete heart block. Note regularity of auricular and ventricular complexes and wholly independent rhythm. P^6 has fallen accurately upon a ventricular T. (*After Thos. Lewis.*)



Lead II.

FIG. 274.—Digitalis heart block (partial). Inverted "T" and lengthened "P-R" interval (delayed conduction characteristic of digitalis block). "P-R" normally (conduction time) is 0.12–0.18 sec. In this case 0.32 sec. Split "P" of mitral stenosis was present in lead I. In this case left ventricular hypertrophy was indicated by prominent "R" and absent "S" in lead I, contrasted with its opposite, a submerged "R" and relatively prominent "S" in lead III. Only lead II illustrating the block is shown here. (*R. Edwin Morris.*)

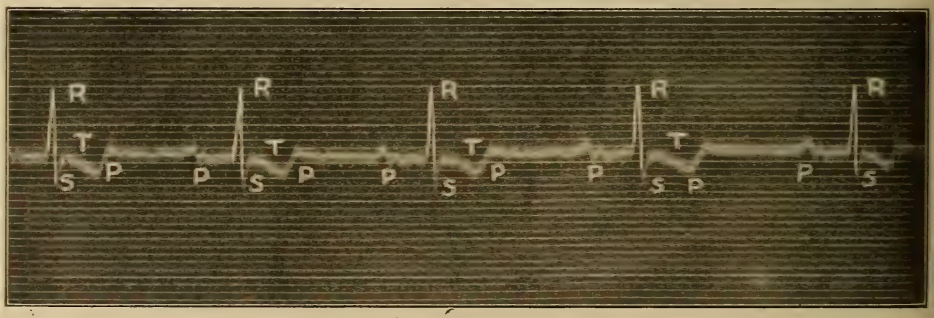


FIG. 275.—Digitalis heart-2 : 1 block. Pulse 60; auricular contractions 120 per minute. Inverted T throughout all three leads (digitalis). Lead II here shown. Dissociation does not exist here, but one-half of the auricular contractions fail to cross the Bridge of Gaskell ("*Bundle of His.*")

hypertensions and the like, in connection with which heart block is found to be very common if tested by instrumental methods.

"Vagus heart-block."

In sino-auricular heart block the complexes are normal but certain *entire beats* may be wholly absent or the heart may slow down and beat at

about one-half the original rate, whether or not this is always a vagus effect is not determined, but it is frequently seen in digitalis toxemia.

HEART BLOCK.—*As previously stated, true heart block results from an impaired conduction of stimuli to contraction passing from auricle to ventricle through the bundle of His ("Gaskell's bridge").*

Partial block.

The normal conduction time is one-fifth of a second and the "block" may represent a mere lengthening of this interval (the a-c interval of the polygram or "P-R" of the electrocardiogram) or there may be complete dissociation, in which case the auricle beats independently at about the normal rate while the ventricle originates its own contractions and beats at a rate approximately one-half the normal.

Complete block.

Acute Cerebral Anemia.—If the ventricular contractions become too slow and feeble to supply the brain, syncopal attacks, convulsive seizures, vertigo, or headache may occur which with the evidence of auriculo-ventricular block constitute the Adams-Stokes syndrome.

Adams-Stokes syndrome.

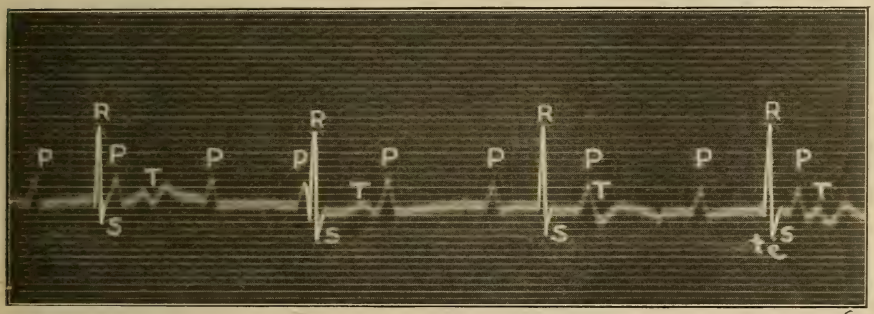


FIG. 276.—Heart block. Dissociation of auricle and ventricle is shown. Ventricle has adopted its independent intrinsic rhythm and the auricle is responding faithfully to the impulses from the pace maker.

Death may occur in these attacks or recovery may take place despite a complete cessation of ventricular systoles extending over a brief period. The radial pulse may drop to 5 or 6 beats to the minute just before such an attack, and in one such case of the author's in which recovery from the attack occurred the ventricular beat was wholly inaudible for a period of over twenty seconds.*

Asystole.

Apparent death.

Age Periods.—Heart block is far more frequent in the male (75 per cent.) than in the female, may occur at any age and will fall into two general groups, *i.e.*, those of the younger ages (ten to forty) in which past rheumatism, chorea and acute infections generally play a chief part, and the older group in which the primary cardiovascular degenerations, aortic lesions and syphilis are the main factors.

Rheumatic.

Luetic and degenerative.

* One of Mackenzie's cases had 50 attacks of syncope and 15 slight epileptiform seizures during a period of ninety minutes, these attacks recurring every few moments for ten days.

Odriozola recently reported a case showing shortly before death intervals of from 50 to 58 seconds between the heart beats (practically one to the minute).

Acute Infections.—The heart block so frequently present in the acute infections though occasionally complete is usually partial and apparently transient, being indicated by a mere increase of the a-c or "P-R" interval, detected by the polygraph or electrocardiograph.

Transient block.

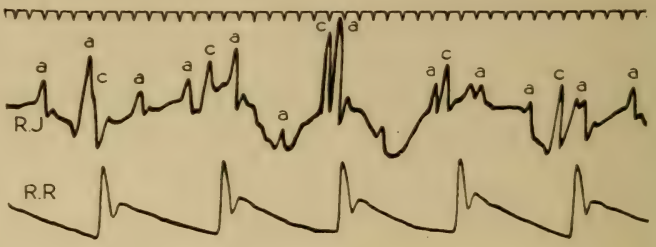


Fig. 277.—Heart block and dissociation in child aged twelve years. Auricular waves are marked "a," carotid waves, "c." Auricular rate 38 per minute. Ventricular rate 88 per minute (almost a 2 : 1 block.

Fig. 278.—Same case recovering from pneumonia. Auriculo-ventricular dissociation. Upper tracing, jugular. Lower tracing, radial. The auricle beats at the rate of 108 per minute (a.a.a., etc.). The ventricle takes its own rhythm from the junctional tissue and beats at a rate of 41 per minute. Auricular rate has increased one-fifth; ventricular rhythm remains practically unchanged).

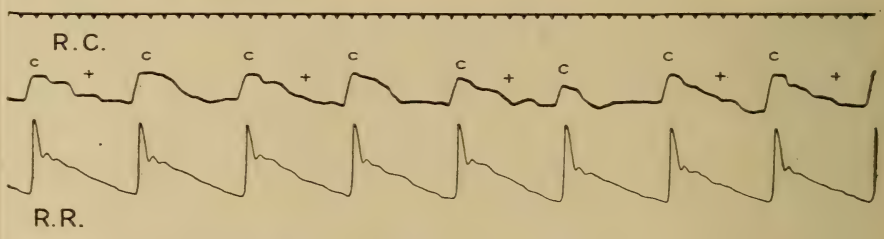


Fig 279.—*Carotid and radial* tracing of same case. Carotid tracing shows slight eminences indirectly due to the auricular contractions. See beats marked +.

Heart block of this type is especially common as a sequence of or in direct association with, acute rheumatism, secondary syphilis, influenza, diphtheria, pneumonia, typhoid fever and profound sepsis.

As in the case of the extrasystoles, its practical value in this connection lies chiefly in the fact that it emphasizes the importance of myocardial toxemia in the commonest of acute infections, which are doubtless the starting point of chronic degenerative processes far more often than has been believed hitherto.

Indicates myocardial toxemia.

Changes in the Bundle of His.—Aside from the transient acute infectious types of which little is known, these are of the most varied description, viz. —acute myocarditis, ulceration, chronic myocarditis, gummata, fibrosis,

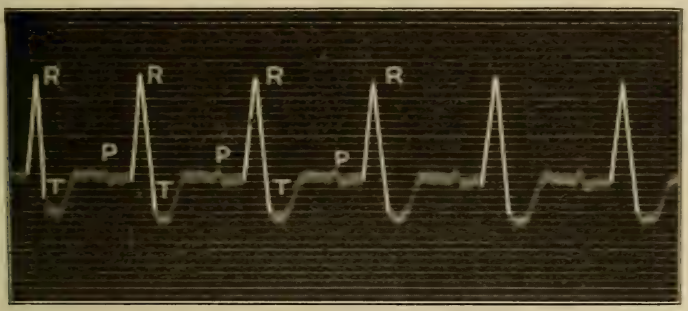


FIG. 280.—Lead I.

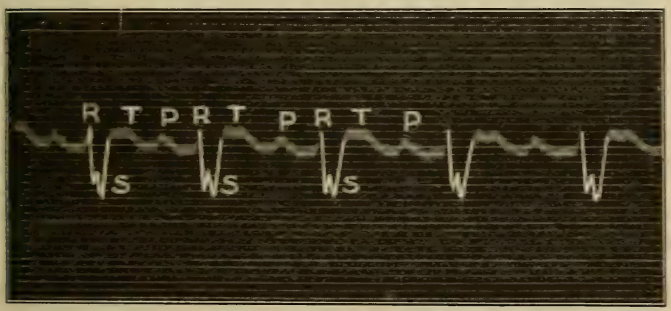


FIG. 281.—Lead II.

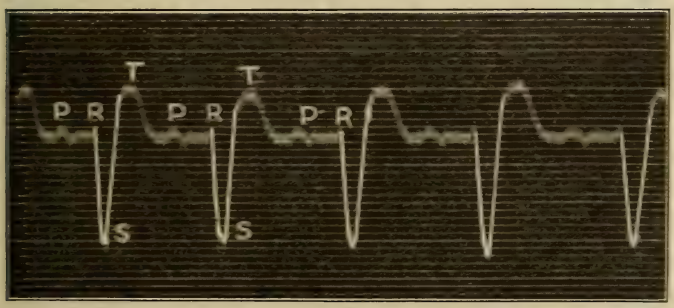


FIG. 282.—Lead III.

FIGS. 280, 281 AND 282.—Intraventricular heart block. Impaired conduction in right branch of "Bundle of His." The "R" "S" "T" complex is characteristically broadened and split. The appearance of the electrocardiogram resembling broadly extrasystolic deflections of the opposite (unaffected) ventricle. If left branch were affected the picture would be reversed. (*R. Edwin Morris.*)

tumors, etc. Cryptogenetic focal sepsis and syphilis probably play a prominent part in the heart block of the older-age group.

Recognition of Heart Block by Simple Means.—The lesser grades associated only with a lengthened auriculo-ventricular conduction-interval

(a-c of the polygram, "P-R" of the electrocardiogram) cannot be detected by mere observation.

Suspicious bradycardia.

One should suspect block (a) when the pulse is below 50; (b) whenever the presystolic murmur and thrill of mitral stenosis is maximal in early or mid-diastole of the ventricles; (c) in cases of fibrillation with a pulse rate less than

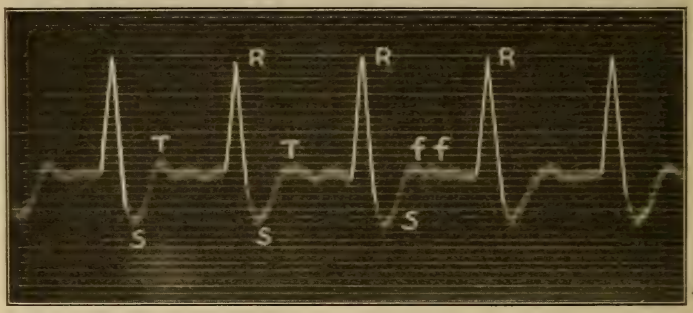


FIG. 283.—Lead I.

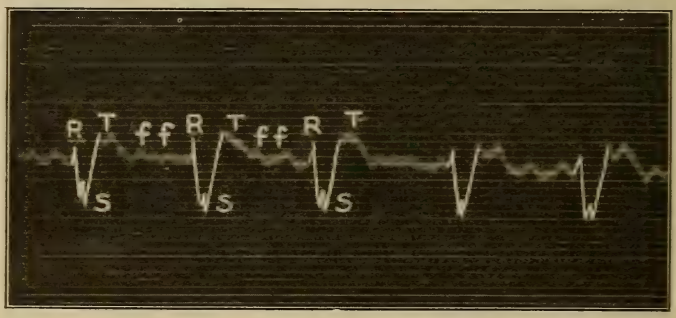


FIG. 284.—Lead II. Note fibrillation in all leads.

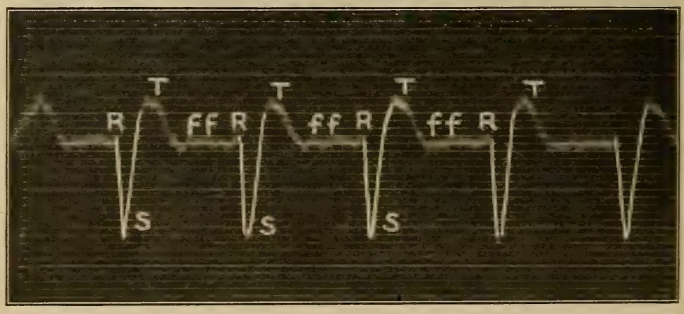


FIG. 285.—Lead III.

FIGS. 283, 284 AND 285.—Same case three weeks later.

Suggestive conditions.

twice the normal; or (d) in cases of any kind associated with clearly reduplicated first or seconds at the apex.

Auricular "ticking."

Turning the patient on the right side one rarely may hear over the auricles tiny clicks which resemble the ticking of a watch and represent

auricular contractions made audible by their distinct separation from the ventricular systole. This, of course, only in cases of complete dissociation or very decided block.

Large positive jugular pulse.

Furthermore, the normal visible jugular pulse may be increased and, perhaps, additional undulations single or multiple which are suggestive of a lack of orderly sequence in the auriculo-ventricular events.

Dropped systoles.

Single systoles may be dropped even when others pass through the conducting bundle and these elisions may be regular or irregular in occurrence. The silent pause with pulse intermission thus induced is in sharp contrast to extrasystolic dropped radial beats in which case the frustrated or abortive ventricular systole is audible either as to one or both its elements. Such silent intermissions *if unaffected by deep respiration* indicate a blocked impulse.

Silent pauses.

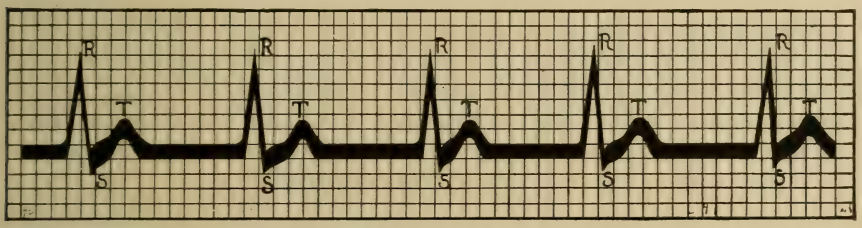


FIG. 286.—*Nodal rhythm.* Auricle and ventricle contract simultaneously. Auricular complex wholly lost. Note absence of "P," normal "R" and "T;" and absolute regularity equality of "R"s. Rate 48 per sec. When the ventricle is wholly dissociated from its auricle it adopts a rhythm of its own representing usually in rate about one-half the normal frequency. If impulses to contraction arising in the sinus are blocked at the junctional tissues ("His bundle") the auricles respond, but the ventricle follows its own steady slow (30–48) rate receiving its impulses to contraction from stimuli arising in the auriculo-ventricular bundle itself. Contrast this Electrocardiogram with the figure showing true heart block complete (Fig. 269). In this present instance both auricle and ventricle are contracting from the same junctional center in exact coincidence and the auricular complex is wholly submerged in the normal ventricular phases. The ordinary auriculo-ventricular heart block does not exist, but the sinus node, the normal "pacemaker" is inert. By the pulse it could not be distinguished from simple bradycardia or active auriculo-ventricular complete block (dissociation). (*After Thos. Lewis.*)

Lewis states that in a 2-1 heart block of *mitral stenosis*, in which ailment block of various degrees is common, one occasionally encounters two distinct murmurs of stenosis each with its thrill to each radial or carotid beat. This because of two distinct auricular contractions, each competent to force blood through the narrowed mitral orifice but only one capable of exciting a ventricular response. Such ventricular silences and dropped pulsations are well appreciated if palpation of the carotid and auscultation of the apex of the heart are practised simultaneously.

General Rule.—*Complete block is almost invariably present if the ventricular rate is under* 40 *and in such cases the rhythm is usually regular and peculiarly sedate though, as stated previously, faint echo-like ticks or muffled diastolic sounds over the auricles may indicate the blocked auricular contractions.*

Simultaneous Auricular and Ventricular Systoles.—It must happen at times in heart block with complete dissociation as in ventricular extrasystoles

that one auricular contraction accidentally "falls in" exactly with that of the ventricle.

In such cases, the first sound is usually loud and an extremely pronounced systolic jugular wave is present of the character described in connection with ventricular extrasystoles.

Alternating jugular pulse.

It is evident that this condition, if regularly repeated, as occasionally happens, would yield alternate large and small jugular waves as timed by the apex, one being ventricular-systolic made up of the accidentally systolic auricular contraction acting against auriculo-ventricular obstruction (from the premature valvular closure), reinforced by ventricular contraction itself; the other due to auricular contraction alone, the lagging, dissociated ventricle being at the time of the latter in its prolonged diastole.

Adams-Stokes Syndrome.—Any drop in the ventricular rate below 20 renders the patient unconscious precisely as in temporary complete cessation.

Important distinction.

Temporary attacks, strongly suggesting and often mistaken for the status epilepticus, occur in such cases when actual periods of asystole ensue, though convulsive movements are oftenest limited to twitching of the face and extremities and even severe attacks are unaccompanied by tongue-biting or the involuntary passage of urine. The author has observed but one proven case in which the latter event occurred.

Unmasking heart block.

Latent Heart Block.—It was believed formerly that a latent block might be unmasked by administration of digitalis to the point of toxic effect. In such cases atropin betters the condition to some extent or even removes it probably because the supposedly latent block is actually an active vagus effect produced by the toxic action of the digitalis itself.

The older belief in an actual persistent, pure, "vagus block" is nevertheless, practically abandoned.

Serious when persistent and severe.

Prognosis in Heart Block.—*The existence of any pure, primary and severe heart block without involvement of other structures is dubious, or at least very rare, and a genuine and decided block, if persistent, means a serious condition, a shortened after-lifetime and almost invariably indicates an extensive myocardial degeneration of which the changes in the bundle of His constitute but a part.*

Nevertheless, it is often extremely chronic and, of itself, and in the presence of an otherwise slightly affected myocardium, need not produce death for long periods.

May die in an attack.

Exitus usually occurs through the same mechanism as in other forms of cardiac disease or through an intercurrent ailment though it may occur during a severe attack of the Adams-Stokes type.

ROENTGEN DIAGNOSIS OF CARDIAC LESIONS

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While an elaborate discussion of technic does not properly lie within the province of a work of this character, a few points bearing thereon must be emphasized by way of preface to this chapter.

Technic of heart-study.

1. *To determine approximately the size of the heart one may resort to:* (*a*) **teleroentgenography;** (*b*) **teleroentgenoscopy;** (*c*) **orthodiagraphy.**

Procedures (*a*) and (*b*) require only that the tube be 2 meters distant from the plate or screen, the patient being closely approximated to the latter. Orthodiagraphy requires a special apparatus to the employment of which attaches considerable danger to the operator.

2. Simple roentgenography, with glass over the screen upon which may be traced various sections of the heart outline, offers much valuable information bearing upon the diagnosis and prognosis of heart lesions. One advantage of this method is that in the right oblique position the posterior mediastinum may be studied. This space may be found markedly encroached upon in pericardial effusion, hypertrophy of the right ventricle, dilatation of the left auricle, or aneurysm of the aorta. The usual measurements, however, are made in the postero-anterior position, the patient standing with his back toward the tube and chest against screen or plate.

Principle of Orthodiagraphy.—Since the X-rays take a divergent course from the focal point on the antikathode, any shadow resulting from their obstruction must of necessity be larger than the object causing the obstruction.

This distortion is greater in proportion to the distance of the object from the screen and the proximity of the object to the antikathode. Since the distance of the heart from the outer chest wall is a variable one, we would obtain variable measurements of hearts of a given size if we employed these divergent rays.

Modified Orthodiagraphy.—By means of a very small lead diaphragm, however, one may eliminate all but those rays which pass in a straight line from the antikathode to a given spot on the fluoroscopic screen.

If the tube is now carried around the heart so that this spot of light describes its borders, the latter may be traced accurately upon the glass covering the screen, which is disconnected from the tube box and held firmly against the chest wall. Tracings are also made of the lateral chest walls in order to determine the cardio-thoracic ratio, and of the diaphragm in expiration and inspiration.

The median line is drawn through the shadows of the dorsal spinous processes. The most distant outer margins of the cardiac silhouette are then indicated upon the orthodiagram and lines are drawn from these points to meet the median line. The sum of these two transverse lines represents the greater transverse diameter and they may be compared to ascertain the relative distance of the right and left cardiac borders.

This orthodiagraphic tracing may now be transferred to a transparent sheet of tracing paper laid over the glass.

Teleroentgenography.—Alban Kohler (Deutsch. med. Wochenschr., 1908), first described this method by which it is possible to avoid the more distorting effect of the divergent rays. He placed the roentgen tube at a

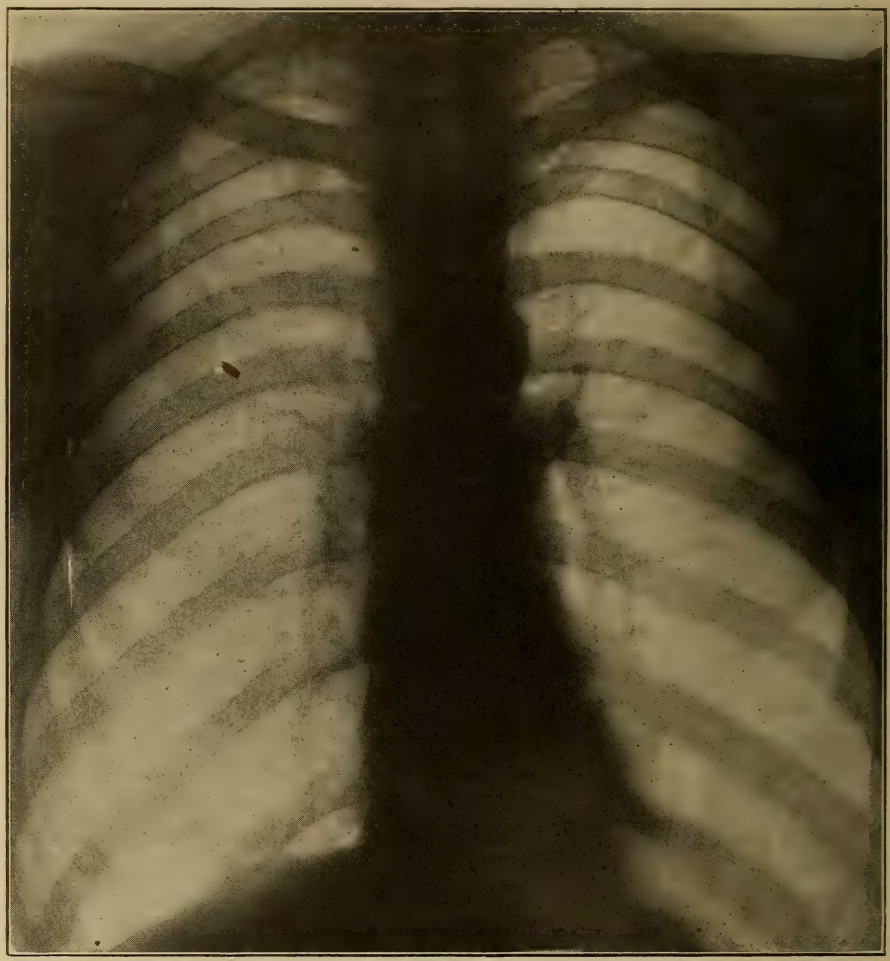

FIG. 287.—"Drop" heart. Extreme type. (*Author's series.*)

distance of 2 meters (about 6 feet 7 inches) from the patient's back; the projected shadow is then only a millimeter or two larger than the heart, and is recorded upon a photographic plate. This method is both safer and more convenient than the orthodiagraphic method and, probably, quite as accurate. Transverse and longitudinal measurements may be traced upon the teleroentgenogram exactly as on the orthodiagram.

Safer and better.

The "Drop" or Pendulum Heart.—The typical "drop" heart is long, narrow and on the screen appears to hang suspended from the great vessels above. Its right border, when visible beyond the vertebral shadow, is

formed by the right ventricle rather than the auricle as in the normal. These characteristics are the result of a low position of the diaphragm and a small atonic heart. Such hearts are extremely frequent in those of an asthenic

FIG. 288.—A modified form of "drop" heart. Mitral stenosis (Duroziez's type).

structural configuration but modified types of the small heart are also occasionally seen in individuals with broad chests.

As the author of this book has shown us, such hearts are readily dilatable and easily overstrained when the general nutrition of their possessors is impaired or an acute prostrating infection is present and even when greatly enlarged, from whatever cause, may most misleadingly present what appears to be a normal total transverse measurement.

Hence it is of great importance for the roentgenologist to be able to recognize the type even when hypertrophy or dilatation has increased the transverse cardiac measurements to normal or above normal. He may find some assistance in the following points of differentiation from the normal.

1. The contractions are sharp, the excursions appearing greater than normal as though one could actually see the heart rotate upon its long axis.

2. The right border is seen to contract synchronously with the left, indicating that the former is the right ventricle. This characteristic is observed, however, only in the typical "drop" heart.

3. The distance from the arch of the aorta to the sternoclavicular articulation is increased.

4. If one suspects dilated or hypertrophied drop heart, it is of some confirmatory value to examine the stomach when filled, thus perchance to demonstrate another sign of congenital asthenia—the ptosed stomach.

Heart profile.

THE NORMAL HEART.—The right and left borders of the heart appear sharply outlined in bold contrast to the air-filled lung on either side.

The lower border is concealed by the dense shadows of the diaphragm and abdominal organs, unless it is brought into relief by inflation of the stomach with gas.

The base is fused with the equally dense shadow of the large blood vessels. The latter should be considered a part of the cardiac silhouette, so closely are they associated both anatomically and pathologically.

Important divisions.

The Left Border.—*A careful study of the left border of the cardiac profile or silhouette makes it possible to separate it into three, sometimes four distinct sections. These sections are convexities separated by slight indentations in the silhouette.*

The uppermost one, marked and abrupt, represents the transverse and descending portions of the arch of the aorta. Below this, more perpendicular, and only slightly convex, is the section representing the pulmonary artery and

Left border notches.

the left auricle, frequently indistinguishable from each other in the normal heart. The third section, longer, more diagonal and tending toward the elliptical outline, is the left ventricle.

Right Border.—On the right side the shadows of the ascending aorta and the vena cava are usually overridden by that of the spinal column and sternum. Below, the border of the right auricle appears as a rather sharp convexity. The right ventricle is not determinable marginally, for it is the right auricle which forms the right border of the cardiac silhouette, save in the

Important exception.

case of "*drop*" *heart* in which the *right ventricle* forms it. Either the aorta or vena cava may be visible above without pathological significance; the former as a convex shadow, the latter as a straight perpendicular line. With this description of the normal heart outline one may proceed logically to a consideration of valvular disease.

Valvular Disease.—In our present state of knowledge it is more scientific perhaps to speak of a mitral, or aortic heart configuration than to attempt a

more accurate roentgen differentiation of various types. However, each lesion tends to modify the cardiac outline and the orthodiagraphic measurements in a somewhat characteristic manner.

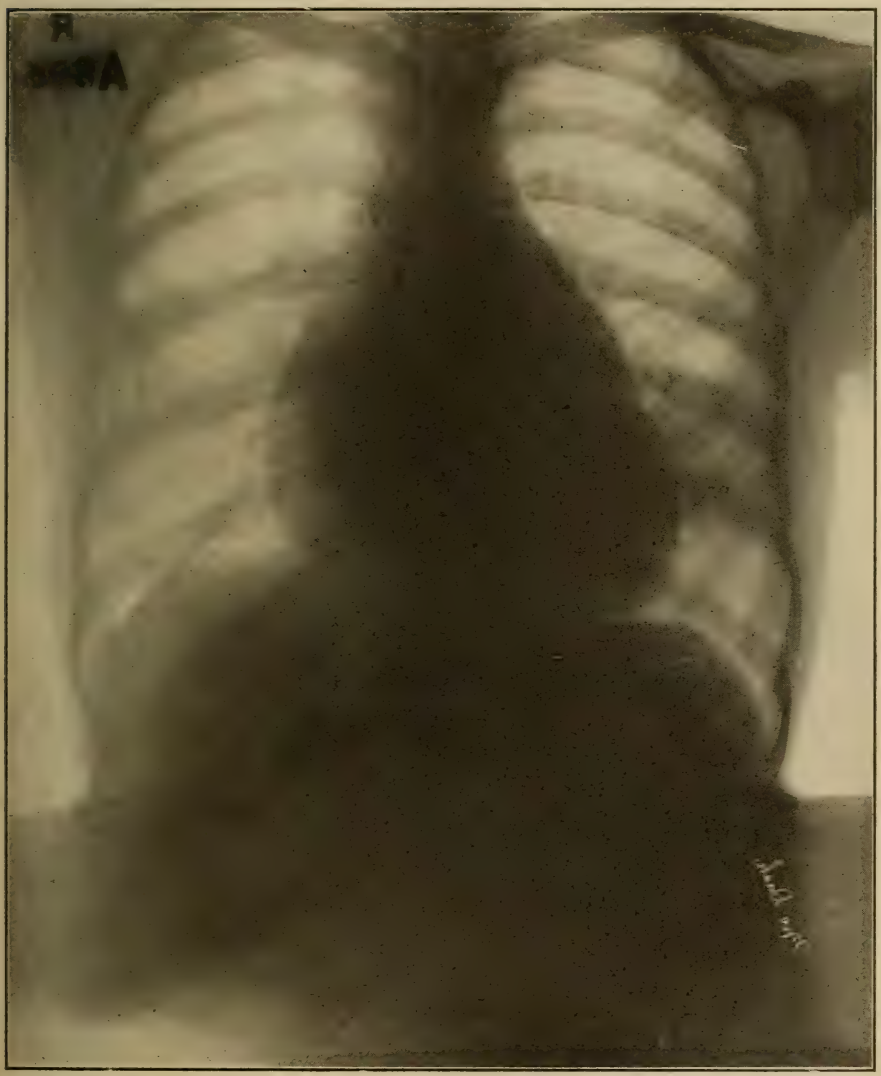

FIG. 289.—Advanced endocarditic mitral insufficiency. Note prominence of both convexities and elliptic contour of left ventricle. Lung shadow opposite left ventricle probably due to past embolic process. (*Dr. Frank S. Bissell.*)

Mitral Insufficiency.—If the mitral valve becomes freely incompetent a tendency to stasis and overload in the left auricle is at once evident. Both the pulmonary artery and the auricle thus become overdistended and a resultant alteration may be noted in the contour of the left cardiac border.

Change in left border.

The section intermediate between the aortic convexity above and that of the left ventricle below becomes rounded out so greatly as to equal or even to

exceed the former in prominence, the result being an obliteration of the normal concavity and the formation of a relatively or absolutely straight line from the aortic convexity above, obliquely downward to the apex. As the left ventricle increases in size through dilatation or hypertrophy its silhouette assumes a more rounded or elliptical contour and this, with the alterations noted above, gives the left heart border a semi-circular shape. If the right ventricle enlarges, it tends to force the already prominent right auricle into greater prominence, thus completing the circle. Here we have the typical mitral configuration. A triangle may be constructed around

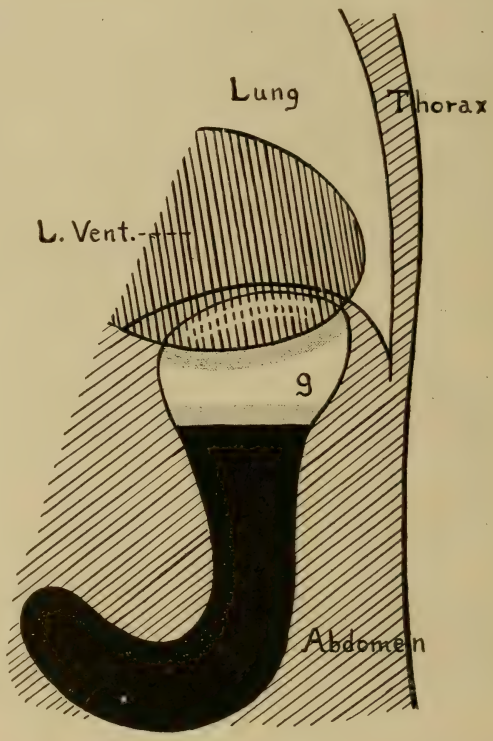

FIG. 290.—Fluoroscopically demonstrable pulsation of cardiac rhythm in aortic insufficiency with hypertrophy and dilatation of the left ventricle, transmitted to surface of gastric contents. (*After Holzknecht.*)

the circle by dropping lines from an interclavicular point (the apex of the triangle) along the right and left cardiac borders to the diaphragm to form the base.

The hypertrophied right ventricle may itself become visible as a perpendicular pulsating border immediately above the diaphragm and to the right of the sternum.

The orthodiagraphic measurements are modified by a marked increase in the total transverse diameter and less marked increase in the longitudinal diameter.

Mitral Stenosis.—In mitral stenosis, the enlargement of the left auricle is the most distinguishing feature, and this is best demonstrated in the left anterior oblique position, the patient being rotated to an angle of 50 degrees. The dilated left auricle is noted as a marked salient into the lower retro-cardial clear space, encroaching upon or merging into the shadow of the vertebral column. The right auricle may also be markedly enlarged while the left ventricle remains small.

Since pure mitral stenosis is extremely rare,* the outline usually encountered is that of the double lesion. The enlargement of the right ventricle and the auricles may be so predominant that the left ventricle is completely overshadowed, the entire silhouette being right ventricular and auricular. This is possible only in pure or stenotically dominant cases in which left ventricular hypertrophy is slight, absent or possibly replaced by atrophy. The left ventricle may be wholly forced away from the wall of the chest by its fellow or one may discern sometimes a division of the left ventricular silhouette into upper and lower curves, that above representing the border of the right ventricle, that below, the left. In both valvular stenosis and regurgitation, but especially in the former, or in the combined lesion, enlargement of the left auricle is inevitable and may assume enormous proportions. Any such increase may be rightly determined by oblique illumination which may reveal an enlargement equal to or much greater than the size of the fist and show clearly and definitely its presystolic pulsations, or its immobility if paralysis or fibrillation is present. The diastolic-presystolic pulsation in cases of very marked stenosis may assume the form of a laborious deliberate vermicular contraction representing the extra effort required to force the blood from the narrowed mitral orifice.

"Pure" stenosis rare.

Vermicular contraction.

In cases of extraordinary dilatation of the auricle, symptoms of pressure upon the left recurrent laryngeal nerve may be present, precisely as in certain cases of aortic aneurysms involving the first or second portions of the arch, or in certain massive pericardial effusions. In like manner decided pressure may be exerted upon the esophagus and the left primary bronchus. The former may be demonstrated readily by having the patient take and attempt to swallow large capsules of bismuth when these will be found to be checked or actually arrested for considerable periods at the point of esophageal obstruction. Curiously enough the patient is conscious of little trouble from

Pressure symptoms possible.

* According to the author's experience, cases of "*pure*" stenosis in the light afforded by modern technic are limited almost wholly to those classified under "Duroziez's disease," elsewhere described, and represent a "drop" heart in a congenitally asthenic, visceroptotic individual who has suffered from active tuberculosis in the past or shows, rather more clearly than the average, roentgenographic evidences of past infection. Several of the author's cases had carried for many years an active, but slow-burning and retarded, pulmonary tuberculosis. In mitral stenosis occurring in individuals of robust build, even when no murmur of regurgitation was demonstrable, the author cannot recall any typical of pure stenosis, and but few small hearts. On the contrary, some left ventricular enlargement has been manifest to some degree even in cases in which, doubtless, the regurgitant element was slight, and, in nearly all instances, a systolic *bruit* has become audible, ultimately, at some time or in some posture. (C. L. G.)

this phenomenon. If a bismuth suspension is taken swallow by swallow, transmitted rhythmic undulation of the esophageal content may be observed.

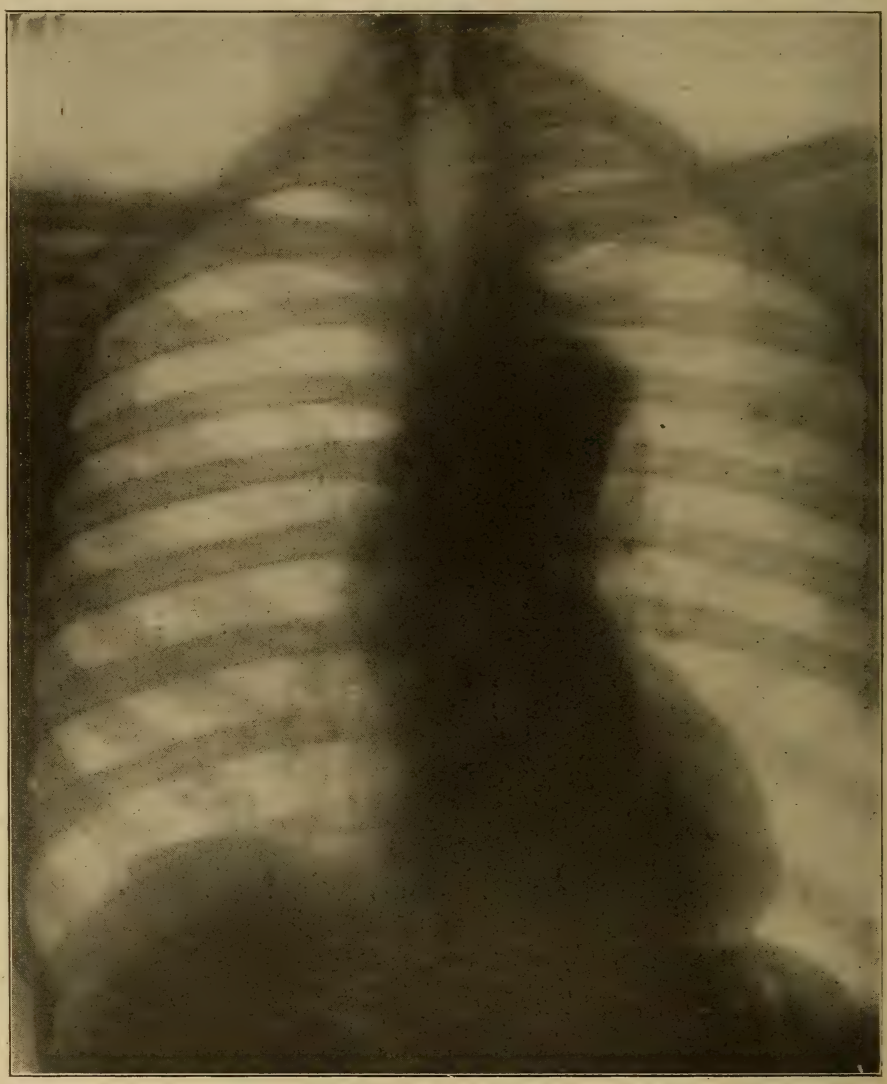

FIG. 291.—Aortic insufficiency with wide dilatation of descending aorta. (*Dr. Frank S. Bissell.*)

Aortic Insufficiency.—In this lesion, the predominant feature is left ventricular hypertrophy with little or no dilatation of the auricle. The orthodiagram therefore shows an elongated, elliptical or oval left ventricular border, a marked intermediate concavity, an increased longitudinal diameter and a relatively short horizontal diameter. The right heart border remains unchanged except when there is marked functional disturbance. The

right cavities may then be dilated. The radiological demonstration of a distended left auricle (previously described) is distinctive evidence of a concomitant mitral stenosis. In the case of such enormous hearts as result from the combined aortic and mitral lesions or from the aortic lesions alone (*cor-bovis*) the compression of the left lung and the resulting atelectasis may

A misleading shadow.

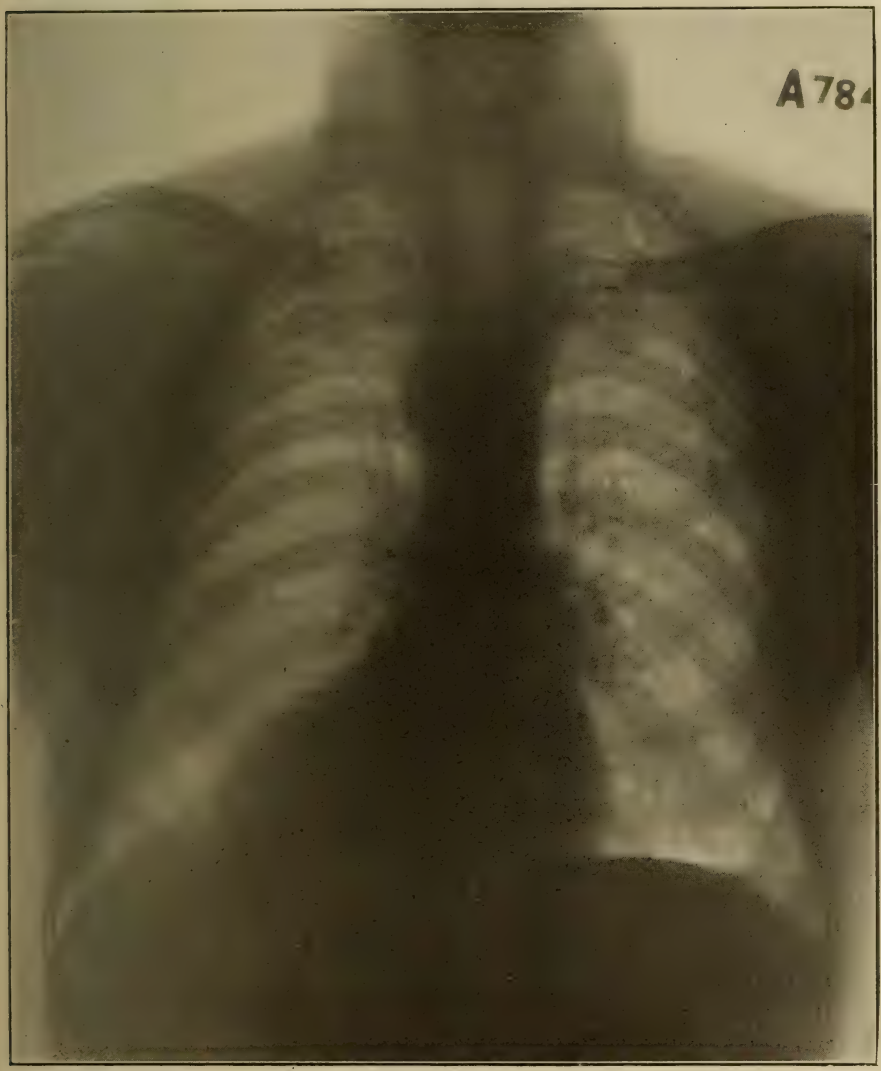

FIG. 292.—Aortic stenosis type of cardiac outline. Note contour of left ventricle "like an egg on its side." Dorso-ventral aspect. (*Dr. Frank S. Bissell.*)

cause so dense a shadow in the left phrenic-costal angle as actually to simulate a pleural exudate or make difficult the delimitation of the true left ventricular border unless the clarifying effect of deep forced inspiration is evoked. In such hearts the diaphragmatic movements are restricted and that struc-

Epigastric oppression.

ture itself may be pressed down 2, 3 or even 4 cm. on the left side. Cohn has reported rhythmic transmittent pulsation of the stomach contents in cases of this kind. Jürgensen believes that the instances of oppression

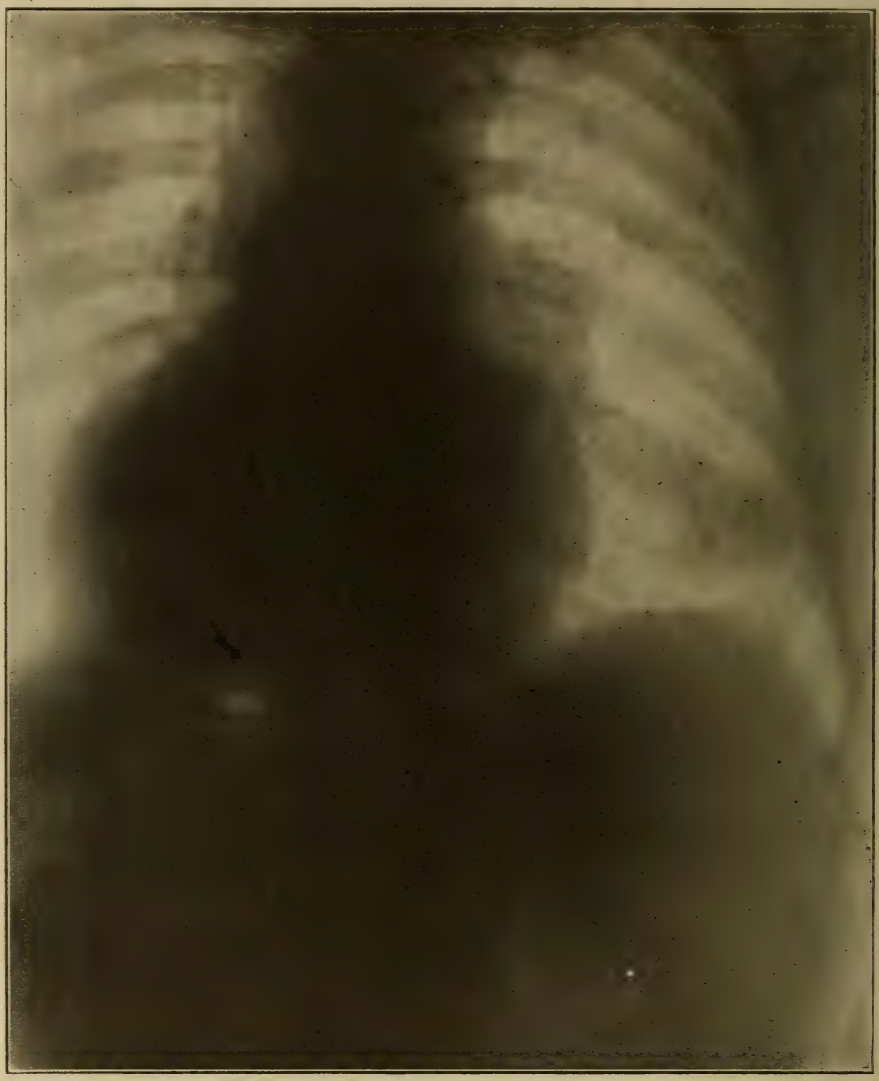

FIG. 293.—Pericarditis with effusion (typical). Note "stubby" neck above.

after meals experienced by many sufferers from heart diseases is due to left ventricular enlargement.*

* This doutless serves as one means of explanation for the extraordinary frequency of epigastric localization of the pain, distress or discomfort of cardiac origin in connection with heart lesions. In many instances, however, it seems to be due largely to *heightened irritability of the muscle* of an overtaxed or actually overstrained heart or one whose intrinsic

Arterial Changes in Aortic Insufficiency.—It is important from the standpoint of prognosis, to be able to differentiate an insufficiency of the endocarditic type from one of arterial origin. In the latter, the aortic shadow appears widened, either from its point of origin to the arch or throughout its visible extent. It usually pulsates more forcibly than normal and shows in the quality and extent of each excursion the water-hammer qualities so characteristically manifested in the peripheral arteries. In the endocarditic type (rheumatic type) changes are limited to the left ventricle, the aortic arch and right heart border remaining unmodified. The orthodiagram shows an increase in the long diameter and a relative decrease in the transverse diameter.

Aortic pulsation.

Combined Aortic and Mitral Regurgitation.—When the aortic and mitral valves both are diseased, the heart body appears more diagonally placed, the left border and the right alike are greatly extended and the apex yet more markedly displaced outward and downward, the exaggerated mid-section of the left silhouette border present in pure aortic lesions becoming filled out to approach or meet the widened areas above and below.

Aortic Stenosis.—Early in pure stenosis the changes in the cardiac outline are wholly absent or very slight and in the case of a "drop" heart especially or in any of small size, the dimensions of the organ *may be less than the normal maximum* even in the presence of an established lesion, for the ventricular changes are extremely slow and the hypertrophy induced is to a large degree, though not wholly, concentric. The radiographic picture of aortic stenosis closely resembles that of insufficiency, the enlargement tending to convert the normal parabolic curve of the ventricular border into an ellipse and in contrast to mitral configuration, to intensify the delimiting notches of the mid-curve of the left border. The apex may appear more blunt and rounded than in aortic insufficiency. In aneurysm, especially of the ascending aorta, and in cases of arterial hypertension, as in other conditions which tend to overload the left ventricle, one may see sometimes the same elliptic type of heart.

Outline changes slowly.

Pericarditis Exudativa.—*A large exudate into the pericardial sac forms a very striking and characteristic roentgen picture. The transverse measurements of the cardiac shadow may be extreme, reaching to the left almost to the lateral thoracic wall, and to the right for 8 or even 9 cm. beyond the sternum into the lung field. Its shape is distinctly that of a squat oval with left lateral predominance surmounted by the short plug-like shadow of the distended sheaths of the greater vessels above, and suggests a similarity to a poorly moulded decanter with a stubby broken neck.*

A squat oval.

A point of differential diagnosis between this configuration and that of chronic general cardiac insufficiency with wide dilatation is that the latter inclines to the triangular in outline. In such cases a slight, low-lying, *small* pericardial accumulation may intensify the triangular configuration and in both the pulsatory excursion of the borders is greatly diminished.

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blood supply is irregular, unresponsive or inadequate. Jürgensen also made the interesting observation that in pressure cases arterial tension was markedly increased unless the pressure of the gastric gas content was relieved. (C. L. G.)

A reference to the "*rationale*" of exudative pericarditis, fully set forth under its proper heading, will make evident the difficulty or impossibility of roentgenographic detection in the case of many small pericardial exudates.

All degrees of variation of the typical picture here depicted may be encountered.

FIG. 294.—Large aneurysm of the ascending aorta. (*Dr. Frank S. Bissell.*)

Sagittal illumination.

The Normal Aorta.—In the frontal (sagittal) position examination of the left side shows a shadow of semi circular contour above, which represents the projection of the upper descending portion of the aortic arch. Measurements are made to determine the total width of the arch at this point, and the distance which separates its point of origin from the sterno-clavicular

articulation. In normal adults of middle age this distance is on the average two or three centimeters. Its length diminishes in the old and tends to shortness in patients with a short thorax. However the upper margin of the aortic semicircle never overlaps the shadow of the left clavicle except in the presence of aneurysm of the arch. The above observations are made in the erect position and during shallow respiration.

Aorta largely hidden.

FIG. 295.—Aneurysm of aortic arch. Enlarged mediastinal glands. Fibrosis pulmonium. (*Dr. Frank S. Bissell.*)

The right anterior oblique position of 45 degrees offers the best view of the ascending aorta, extending with parallel borders toward the clavicle. At an oblique angle of 60 degrees the descending aorta appears as a convex projection into the retro-cardiac space.

Oblique illumination.

Source of error

In diffuse dilatation of the aortic arch a gently curving, slightly flattened convex shadow appears, showing expansile pulsation. To the left the normal degree of projection may be doubled or so enlarged that its outer border intersects that of the heart at the upper part of the left ventricular silhouette or even beyond that point. Such an aortic shadow right and left may strikingly suggest a large aneurysm and frequently is so interpreted.

FIG. 296.—Large aneurysm of the transverse and descending aorta. (*Dr. Frank S. Bissell.*)

'Middle lung field" or "retrocardial field."

In such cases the retrocardial light-field is narrowed but not obliterated. In the form of aneurysm which it simulates the illuminated strip is darkened above by the circumscribed projection which represents the aortic aneurysmal sac.

Expansile pulsation is usually present in aneurysm, but if the sac walls are excessively thick and rigid or the dilated portion itself is packed with clot, such pulsation may be absent or slight in that part accessible to view.

But one form of malignant new growth shows anything even approaching true aneurysmal *expansile* pulsation and that is an extremely vascular type of sarcoma rarely encountered.

Aneurysm.—Thoracic aneurysm occurs with decreasing frequency in the aortic arch, the ascending aorta and the descending aorta.

Often unsuspected.

It may attain large proportions before producing characteristic symptoms or physical signs, and may be and often is wholly unsuspected and accidentally or incidentally discovered in the course of routine screen examination.

Suggestive symptoms.

A special search for aneurysm should be made in all cases of middle or advanced age in which complaint is made of pain in the back, pains radiating into the arms and, especially, when the aortic tone is not perfectly normal.

Typical appearance.

Aneurysm of the Ascending Aorta.—*The typical appearance of aneurysm of the ascending aorta is that of a sharply rounded prominence of the aortic shadow projecting into the right lung field.**

Turning the patient into the first diagonal position with the screen to the right in front and the rays directed between the left scapula and spine, one may observe the same prominence projecting into the posterior mediastinum. If the aneurysm is of a moderate size and the ascending portion alone is affected, there are usually few pressure symptoms. When, however, the arch is also involved, the left bronchus and the esophagus may show early the effect of pressure. The arch appears widened in all directions; the course of a bismuth capsule showing the gullet to be displaced backward and to the right.

Dysphagia.

The upper margin of the semicircle formed by the aorta approaches the clavicle until, as stated above, there is an overlapping of the two shadows. There is also an increase in the length of a line drawn from the sterno-clavicular articulation to the junction of the aortic semicircle with the heart shadow.

Dysphagia.

Aneurysm of the Descending Aorta.—By reason of proximity of the esophagus to the descending aorta, displacement of this organ (with subjective difficulty in swallowing), is one of the early signs of aneurysm of this type. The shadow of the widened aorta projects into the left lung field, well beyond the normal heart shadow.

The differential diagnosis between aneurysm and mediastinal tumor may be difficult. A distinctly delimited and defined tumor showing expansile pulsation favors the former, whereas the irregular, less definitely outlined mass speaks for the latter, but the character of the pulsation is the safest guide, though sometimes fallible.† Transmitted pulsation is, of course, relatively common in new growths occupying this region.

* In three cases personally observed a massive tumor representing an aneurysm of the ascending portion of the arch extended downward and to the right, the percussion dulness in two of the cases reaching that of the liver. (C. L. G.)

† By an extraordinary coincidence, observed some years ago, a malignant growth and a saccular aneurysm co-existed and permitted a tentative diagnosis of the combined lesion based upon the (then very imperfect) roentgenographic procedure. (C. L. G.)

"Fatty" Heart.—Fatty overgrowth is characterized roentgenologically by the appearance, under the use of a soft tube, of a lesser gray shadow filling the cardiophrenic notch at the apex and tailing off from the pericardium overlying the left inferior border and apex to the upper surface of the diaphragm It is roughly triangular in most instances.

The "bob-tail" heart.

A similar gray shadow may occur at the corresponding angle on the right side.

The Heart Silhouette in Nephritis.—The cardiac hypertrophy which occurs as a result of increased blood pressure, arteriosclerotic kidney, etc., *offers no characteristic roentgen sign.* There may be uniform increase in all the dimensions of the heart, and both pulmonary and aortic shadows may increase in width. With the development of stasis in the pulmonary circulation the lung field becomes clouded in appearance.

The illustration of "general dilatation and insufficiency" here shown depicts a common late or terminal type.

THE DISEASES OF THE HEART AND BLOOD VESSELS

The Importance of Prophylaxis and Early Diagnosis

Great Advance in Knowledge.—With relation to the causes and prevention of cardiovascular diseases much of the recent research work is of great value.

Syphilis.—*Lesions of the aorta, aortic valves and myocardium are found to be far more commonly associated with syphilitic infection than we have formerly believed, this fact accounting, no doubt, in large measure, for the relative rarity of aortic valvular lesions in childhood and for the effectiveness of the iodides and of fractional doses of salvarsan in many adult cardiopaths.*

Extremely common.

It is evident also that in the heart, the *luetic* process, as related to the stage of the disease, is one of relatively early inception, and slow but progressive development. (See Cardiovascular Syphilis.)

Begins in early stages.

Denial of Syphilitic Infection.—In direct opposition to the views of some syphilologists recent reports on cardiac syphilis based upon the specific diagnostic tests and the result of autopsy, emphasize the extraordinary frequency with which luetic infection is denied even in public services.

Denial of infection the rule.

These facts emphasize the diagnostic and protective value of positive Wassermann and luetin tests in obscure cases, the importance of prompt antisyphilitic treatment in proven cases of infection and the propriety of test treatment in resistant aortic and myocardial cases.

Test treatment.

Space does not permit a general consideration of the entire etiology of arteriosclerosis or of myocardial inflammation and degeneration in general, but a word may be said in relation to acute rheumatism and certain other infections producing endocarditis and myocarditis.

* Dr. Thos. B. Hartzell, Dr. Henrici, and Dr. Gray working in the medical clinic of the University of Minnesota, have demonstrated the extraordinary frequency of occurrence of the *streptococcus viridans* in pure culture in the peridental abscesses so commonly associated with chronic joint lesions.

Rheumatism.—*The onset and course of acute rheumatism have always suggested an infection of a modified septic type*, and the relatively recent work of Poynton and Payne, Beattie, Beatson, Longcope, E. C. Rosenow and many other observers has shown that a mildly virulent microörganism assuming any of three forms (a diplococcus, micrococcus or streptococcus) in its different strains, may be quite constantly recovered *from the subsynovial areolar tissue of the inflamed joints of the rheumatic patients* (*Poynton*).

Modified sepsis.

Recovery of organisms.

Grown in pure culture and passed through a series of susceptible young animals these organisms are said to produce consistently an acute arthritis. Being extremely susceptible to phagocytosis, they are quickly destroyed, are recovered somewhat rarely from the blood, in which they do not multiply, and with difficulty from the joint exudate (*Poynton*).

Foci of Infection.—The intimate relationship between acute and chronic non-suppurative arthritis, and chronic streptococcus infections of the tonsil, and, frequently, of the accessory nasal sinuses, jaw or prostate, seems to be definitely established not alone by the extraordinary frequency with which pure or nearly pure cultures of various strains of the pathogenic streptococci occur in the depths of the tonsils of rheumatic patients or in dental abscesses, but also by the astonishing immunity to or relief from the disease following the complete painstaking removal of all tonsillar tissue or the radical cure of other foci of infection.

Portals of infection.

Streptococcus strains.

Rosenow's work, in clinical collaboration with Frank Billings, later indicates that the organisms above described, together with the *streptococcus viridans*, are strains of the hemolytic streptococcus, modified by the variations of oxygen pressure attending their growth, and that the different varieties show more or less well-defined selective affinities for the endocardium, synovial membrane, myocardium and skeletal muscles respectively.

Radical Treatment of the Chief Etiologic Factor.—Guerich advocates the removal of suspected tonsils at any stage of active rheumatism,* and though we may consider this a too radical rule of action, we must admit that, *if diseased, or if past attacks of tonsillitis have occurred, their complete extirpation is indicated in all persons able to bear the operation who show a rheumatic tendency or who have passed through an attack of the disease.*

Radical suggestion.

Justifiable procedure.

Indeed, bearing in mind the early or primary involvement of these structures in diphtheria, influenza, scarlatina and other acute infections in which endocarditis and myocarditis frequently occur together, with their etiologic potency in relation to chronic arthritis, anemia, and probably in many other chronic ailments, it would seem that with respect to the urgency of surgical procedure we are justified in placing tonsils in the same category and *at the head of the list* which comprises adenoids, chronically infected accessory sinuses, chronic appendicitis, cholecystitis, prostatitis and the peridental infections.

Relation to other infections.

Tonsils head the list.

The author finds that many cases carrying curiously unstable intermittently toxic hearts, quite apart from actual valvular disease, and often with no history

* In the relapsing form of rheumatism this procedure may be quite justifiable and initiate permanent recovery after one or two days of post-operative exacerbation.

Chronic intermittent myocardial toxemia.

of rheumatism or any related acute infection, recover myocardial tonus only after such foci are discovered and removed.

May appear normal.

Diseased tonsils, especially, are natural incubators, transmutators and commissaries for invading organisms, and may appear misleadingly normal on inspection even while breeding successive generations of pathogenic germs or containing actual pus in considerable quantity.

Partial removal is a poor and usually a wholly ineffective substitute for complete enucleation.

Again and again the Author has seen acute rheumatism develop after a tonsillitis affecting the tonsillar remnants persisting after a partial extirpation.

Misleading Delayed Onset.—In seeking to establish the etiologic factors in a given case of acute rheumatism or endocarditis, one may readily overlook acute tonsillar inflammation.

An acute tonsillitis though rarely absent in a carefully taken history, seldom accompanies the actual onset of arthritis, but more frequently precedes it by a period varying from a few days to three weeks, and may be very mild.

Prostrating infections.

Physical Debility and Cardiac Weakness.—Yet another point of practical value in relation to etiology, diagnosis and therapy is to be found in the consideration of acute rheumatism, diphtheria, scarlatina, influenza and indeed any acute infection *associated with marked physical prostration.*

To the author it seems probable that in such cases the excessive, genuine weakness so often encountered is ordinarily not one primarily or chiefly resident in or directly affecting the skeletal musculature, though this and the nervous system must be important factors, but, *in a large degree, represents the relative inadequacy of a more or less poisoned myocardium.*

Myocardial toxemia.

In the fatal cases of acute infections the cardiac muscle invariably shows at autopsy, greater or lesser local effects of circulating toxins even though a grossly demonstrable endocarditis or actual myocarditis be absent. The assumption that in all acute *prostrating* infections the myocardium suffers at least temporary damage is justified apparently by our present better knowledge of the symptomatology of minor cardiac insufficiencies and, of late, is generally accepted. The vasomotor control is also greatly disturbed.

Heart Muscle vs. Skeletal Muscle.—The heart's intrinsic circulatory capacity is ten times that of skeletal muscles. It is an extraordinarily delicate and highly specialized tissue, and must maintain constant activity under conditions of direct and disproportionate exposure of its naked fibers to the toxins of disease.*

A striking contrast.

The less delicate and highly organized skeletal muscles, on the other hand, are at rest during an illness, receive relatively a far less abundant supply of toxic blood and, in most instances of brief prostrating infections, fail to show under dynamometric tests any such degree of weakness as accords with the subjective and objective manifestations of exhaustion which may be present.

Instrumental methods, moreover, have shown that auricular fibrillation, the

* Heart-muscle fibres are unsheathed.

lesser degree of heart block and extrasystolic irregularities occur with suggestive frequency in various acute infections followed by recovery.

Toxic arrhythmias.

Percussion Outlines.—In many of these cases, during such periods of weakness, carefully conducted percussion by modern methods, especially after slight exertion, reveals an abnormally increased cardiac outline, most frequent and most marked in such persons as structurally fall under the head of "chronic congenital asthenia," but by no means limited to that group.

Minor dilatations common.

Abnormal Heart Sounds and Bruits.—*Furthermore, one will often find, under like conditions, and even in the absence of profound anemia or actual endocarditis, not only transitory or inconstant murmurs, but also a weakness, undue sharpness, muffling, or abnormal accentuation of the cardiac tones, indicative of distinct though usually transient myocardial damage.*

Significant signs.

Rest after Acute Prostrating Infections.—Many a case of persisting cardiac weakness and ultimate chronic degeneration of the myocardium dates its inception from the onset of such an attack, unrecognized and unsuspected, and its persistence to a premature resumption of customary activities.

A neglected precaution.

Prolonged cardiac rest, rational heart stimulation, and a gradual and guarded resumption of normal activity, after diphtheria, acute rheumatism, scarlet fever, typhoid, severe influenza, and in fact, all acute infections attended by profound physical prostration, is a matter of vital prophylactic importance too little regarded at the present time.

The almost universal failure to examine such cases thoroughly and critically after the resumption of full activity, results in the loss of many golden opportunities to detect chronic cardiac and cardiorenal lesions in their early stages.

The Viewpoint.—Obviously, a mere detailed study of individual lesions is by no means sufficient to prepare one to meet and deal with a branch of medicine characterized alike by the diversity and complexity of its problems and the interdependence and logical sequence of its *morbid* phenomena. Therefore, we must understand the genesis of such lesions and acquire a thorough knowledge of the symptomatic expressions of cardiovascular inadequacy, more or less common to every organic lesion.

THE VITAL POINT.—*Heart-muscle is the paramount clinical factor in cardiovascular problems even though primary, associated, or consecutive valvular lesions, arteriosclerosis or pericardial inflammation are the most obvious and obtrusive elements in the given case.*

Always the myocardium.

Diagnostic Limitations.—*An impeccable antemortem differentiation of the many forms of pathologic change which may enter into myocardial degeneration and resulting cardiac insufficiency is an absolute impossibility.*

We cannot clinically differentiate the clinically indivisible, but we can and should recognize the resulting cardiovascular insufficiency.

Nor is it vitally necessary that we should finically differentiate the myocardial degenerative processes, if, relatively early, we are able to detect their presence and to retard their damaging effects upon the circulation and the body tissues which this nourishes and purifies.

That which, even upon the autopsy table, leaves much for the *microscope* to determine does not lend itself to hair-splitting clinical diagnosis, and

though one may definitely place an arrhythmia or a specific valvular lesion, the exact *anatomic* alterations of the *heart muscle* or of its coronary arteries cannot be determined *antemortem*.

The Problem is Always Myocardial.—One properly may reiterate the fact that valvular lesions and the arrhythmias alone offer an opportunity for specific diagnosis and indeed their separate recognition is of great value and may strongly influence our management of the case. At bottom, nevertheless, practically all problems are those of heart muscle and even our pathologists give few clean-cut differentiations of that *composite of myocardial degenerations, invariably encountered post-mortem* in the *diseased* heart. *The question ever is that of predominance* of one of the many pathologic processes present.

No clean-cut divisions.

Many Possibilities.—A heart muscle may be congenitally unstable in strength and tonus, as in congenital asthenia; weakened by severe infections, by cryptogenetic sepsis, or by the damage wrought by the physical handicaps and associated myocardial damage, inseparable from valvular defects. To either condition, fatty overgrowth, fatty degeneration, cardiosclerosis, acute or chronic toxemias, actual bacillary invasion, coronary sclerosis, fresh endocarditis, or a pericarditis, may be superadded.

Phasic Changes in Co-existent Lesions.—Several important lesions, valvular, vascular, or myocardial, may co-exist in the same heart, and, with the passage of time, the primary dominance of one may be submerged in that of another, and, ultimately, the combined, cumulative effect of all may be exerted.

Shifts and changes.

It is with relation to the myocardium especially, quite apart from manifest dilatation, that *subjective manifestations* are more important than objective symptoms as aids to early diagnosis and, as in the case of a positive Wassermann or the recovery of the streptococcus viridans or streptococcus rheumaticus from a blood culture, may outweigh in therapeutic importance all the murmurs and thrills in the category.

Subjective vs. objective symptoms.

The basic symptoms are ever those of **cardiovascular weakness, and impaired "reserve"** *manifested in all varieties by much the same general symptoms and permitting no finely spun conclusions of a clinical sort as to the dominance of this or that form of degenerative process.*

"Chronic Myocarditis."—*Myocardial degeneration* or even *"myocardial insufficiency"* is a far better clinical term than the much employed "chronic myocarditis" until we shall have been told what actual chronic myocarditis really means *in clinical terms*.

Loose use of term.

The author prefers to limit the latter term to cover, at most, the earlier periods in cases of chronic insufficiency distinctly following the acute infections, in which *severe* acute myocardial processes, recognizable or not, have left residual nutritive deficiencies and degenerative processes which latter result in a *dominance* of fibrosis, fatty degeneration, brown atrophy or what not.

Suggested limitation.

We can no more than assume as **probable** *the presence of a true chronic myocardial inflammation, from any antecedent history of a recent acute infection during which clinical signs and symptoms of myocardial insufficiency, still in part or whole existent, were clearly in evidence.*

A probability not a certainty.

Recognition of Insufficiency Imperative.—*Inasmuch as cardiac insufficiency arising from any one of its many causes is so often exactly like that of the others, we must strive to recognize "insufficiency" itself, and as such. Upon that foundation, we may build as stable a diagnostic structure as the ascertainable, additional data permit and by this determine our therapeutic initiative.*

The primary necessity.

Cardiovascular Reserve.—A knowledge of these signs and symptoms peculiar to individual valvular lesions and those of gross myocardial insufficiency together with the technical training necessary to their recognition are alike indispensable, but the therapeutically productive, life-extending field is that of conservation of cardiovascular reserve, and, as previously emphasized, the chief duty of the physician lies in the careful study of the signs and symptoms of the *earlier and lesser compensatory defects.*

Early recognition imperative.

The Lesser Symptom-producing Insufficiencies.—These lesser cardiac insufficiencies and minor dilatations merit a far greater amount of study and therapeutic attention than they have hitherto received, for modern experience and investigation shows that damaging, acute and chronic, persistent, or temporary, but recurrent, toxemic overstrains with or without myocardial degeneration or underlying valvular disease, are relatively common sources of many misinterpreted subjective or even objective symptoms.*

Unrecognized inadequacies.

CARDIOVASCULAR "SUFFICIENCY" AND "INSUFFICIENCY".—What Constitutes Compensation.—Although the so-called "compensatory" cardiac changes associated with and necessitated by actual diseases of the heart are usually adaptive rather than truly *compensatory*, they are nevertheless wonderful in their efficiency and may remain effective as to the individual for decades in the case of certain lesions. Yet nothing can compensate more than partially or temporarily for the loss of a normal cardiovascular mechanism, *and in certain forms of cardiac disease and in certain individuals compensatory changes are extremely imperfect.*

Adaptation to conditions.

Even now we overestimate greatly the efficacy of those remarkable but imperfect retarding processes which we term "compensation." Those of us who were privileged to serve in the Great War saw compensatory changes in the juvenile heart at their best, and it was difficult to realize the fact that if these young cardiopathic cases were *as a body* to be taken over by an insurance company the loss would run anywhere from 250 to 500 per cent. above that of an equal number of normal men.

Nine hundred and ninety-nine out of one thousand confirmed cardiopaths today owe the major part of their protection from gross decompensation to symptoms, recognized or not by them, which automatically enforce a slowing down of their activities, and most cardiopaths, with boaderline reserve, are constantly forcing their diseased hearts.

Obligatory Dilatation.—In valvular lesions dilatation of some degree is

* Horning's report upon a radiographic series of 1100 apparently *normal* hearts among which he found a considerable number of acute transient heart strains has served to show that the condition is more frequent than is generally supposed.

Had his observations been directed especially to the "drop" heart and the remediable minor dilatations of the diseased heart his figures would have been yet more illuminating.

primary and, ultimately, a certain increase in the cubic capacity of the heart chambers *must* be permanent. Secondary hypertrophy, responsive to the need of increased energy, limits dilatation and maintains reserve. Never strictly concentric, it is often a source of *vascular* degeneration from the associated increase of initial pressure involved. Almost invariably, it is soon in gradual process of being insidiously undermined by progressive myocardial degeneration and an impaired coronary blood supply. *The existence of aclinically and pathologically recognizable* **pure** *work-hypertrophy has never been demonstrated.*

Theory vs. Experience.

Theoretically, hypertrophy may overcome completely any primary dilatation or be gradually induced *without it* as in the case of gradually developed arterial hypertension or aortic stenosis, and adequately maintain the circulation for years by simple increase of ventricular force.

Few, if any, cases justifying this assumption appear upon the autopsy table whatever the cause of death, nor can one understand the *continued and stable* effectiveness of such force increase, *associated with normal rhythm*, whether in the presence of free mitral or aortic regurgitation, aortic stenosis, or chronic hypertension, unless there is combined with hypertrophy an increase in the cubic content of the ventricular cavity. A certain amount of fixed dilatation is a necessary *ultimate* accompaniment of all such conditions, and with that the physician need not and cannot interfere. **It is when tonicity and contractility begin to fail that action is demanded.**

Cardinal point.

Morbid Dilatation.—Hypertrophy by limiting *dilatation* prevents over long periods that "*overdistension*" which represents the dominance of acute or chronic overstrain and excessive stimulus to contraction due to residual blood overload, venous stasis or both conditions in the presence of a diminished myocardial tonus. Such morbid dilatation produces symptoms, subjective and objective, in varying degree; narrows cardiac reserve; accelerrates existing degenerative processes; and may properly be called *morbid dilatation*.

An Apparent Fallacy.—One of the apparent fallacies of cardiac pathology is the theoretic assumption that inasmuch as the heart possesses the power to exert a thirteen-fold increase of strength to cover the field of response represented by minimal and maximal demands, absolute rest on the one hand and severe physical strain on the other, it is quite capable of tiding over unaided *and without additional damage*, the period between the onset of a *valvular* lesion and the occurrence of established and adequate hypertrophy, or even the increased dilatation which may result from infection, shock, or physical overstrain in established lesions.

The *normal* heart of the individual who is reasonably "fit" may, and almost always does recover from temporary overstrain and dilatation provided that relative rest is secured. Otherwise, many, rather than a small minority, of college athletes would carry damaged or even crippled hearts."*

* The heart of the perfectly trained robustly built athlete will actually diminish in size with quickened rhythm under strenuous exercise even if sustained during considerable periods, but the author has seen many *congenitally asthenic* or badly trained young athletes retired with badly dilated and extremely irritable hearts. Permanent damage is not uncommon.

It cannot be true of the heart which had weakened and dilated under an acute toxemia of the sort associated with acute endocarditis or myocarditis or one which is more or less continuously subjected to chronic toxemia or sepsis and recurrent or persistent overstrain.

Fortunately, however, from its large primary reserve fund enough usually is left available to keep even these hearts safe *from actual disaster to their*

FIG. 297.—Normal heart, taken in inspiratory phase. Transverse measurement 13 cm.

possessors until the necessary relief occurs, and some further self-protection is afforded the patient *by the subjective sense of weakness, or an actual physical disability, which limits his activity during this period, but which he, unwatched and uninstructed, all too frequently disregards.*

The automatic brake.

That no damage is done in such cases is hardly conceivable nor is it in accord either with clinical experience or experimental work.

Exaggerated Estimate of the Normal Dimensions of the Heart.—No radiographic examples of a *normal* heart of excessive size (exceeding 13.5 cm. in total transverse diameter) have come under the author's observation. (See Cohn's Table under "Heart."p. 429.)

Big normal hearts rare or non-existent.

FIG. 298.—Beautiful example of the "hanging" ("drop") heart. The same quality is more or less well marked in all, if viewed fluoroscopically during full inspiration. Total transverse, 9.5 cm.

Damaging error.

Many abnormally dilated hearts are accepted as normal in size because of a faulty conception of what constitutes a normal outline, together with a disregard of the law of proportionate weights and of individual peculiarities of body structure and conformation.

A common type.

THE "DROP" HEART OF THE CONGENITALLY ASTHENIC INDIVIDUAL.—During the past ten years the author has been deeply interested

in the study of a host of cardiopaths showing predominant subjective manifestations, together with clearly defined evidences of impairment of tonicity with or without either persistent cardiac dilatation or extreme dilatability, *though the heart diameter, radiographically, and by percussion, falls within the maximum "normal."*

The underlying condition corresponds apparently to what Mackenzie called the "X disease." His description is precisely that of a common type of

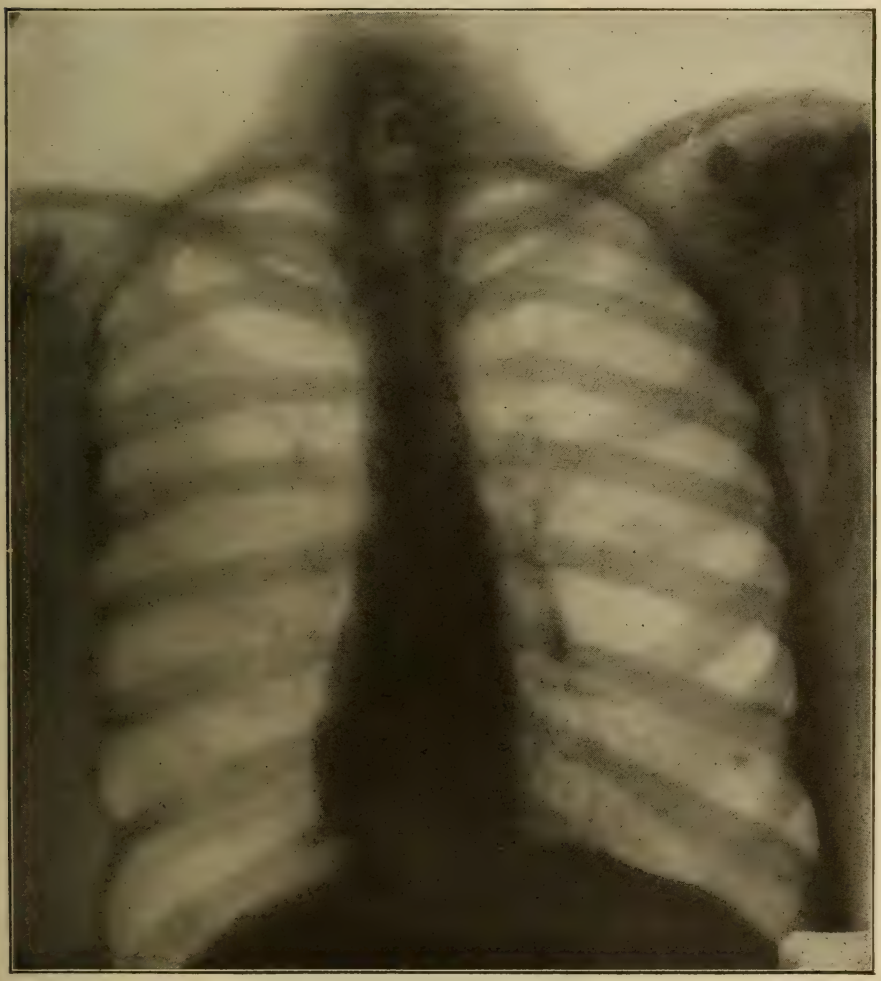


FIG. 299.—An extreme instance of the "drop" heart (dorso-ventral aspect). Dimensions: Mr., 2 cm.; Ml., 5.5 cm.; total transverse, 7.5 cm. (Same heart shown in Fig. 158.)

so-called "passive neurasthenia," and the physical and mental characteristics of these patients, as described by him, would seem to justify one in replacing his "sign of the unknown quantity" by the term "*chronic congenital asthenia.*"

What it represents.

This, the constitutional defect of structure and function of which Berthold Stiller, of Budapest, has drawn so clear and vivid a picture as to secure

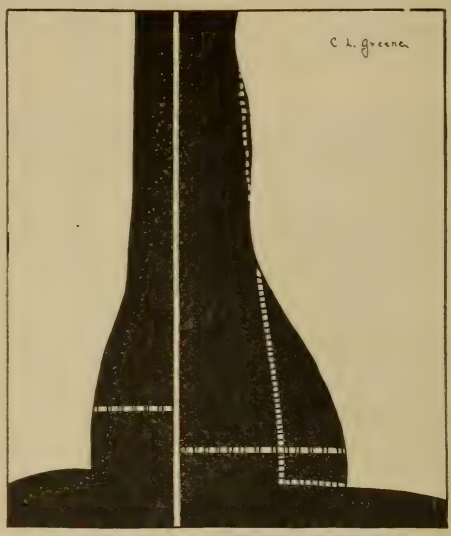


FIG. 300.—Dilated "drop" heart. (Outlines drawn from original roentgenograms (by E. Brill) and superimposed.) Dotted vertical white lines indicate outline after treatment. Case of premature resumption of activity after a very severe attack of lobar pneumonia. Note that in the case of this ambulant patient carrying a decidedly dilated heart the total transverse measurement is but 12 cm. Original measurements: Mr., 4.0; Ml., 8.0; total, 12 cm. Final: Mr. 4.0; Ml., 5.3; total, 9.3 cm.

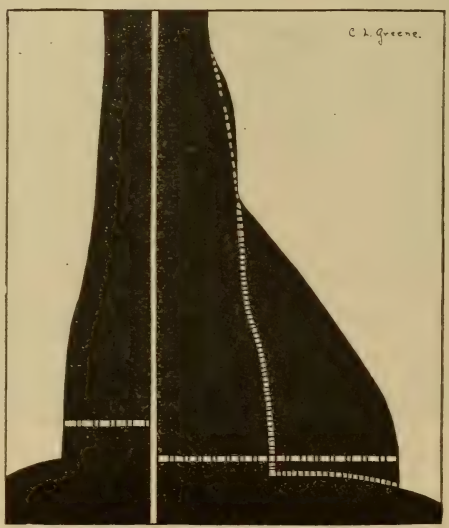


FIG. 301.—Dilated "drop" heart. (Original outline determined by orthopercussion; final profile, by roentgenogram.) Acute toxic dilatation associated with premature resumption of activity, following a severe attack of articular rheumatism. Syncopal attacks. Dotted vertical white line represents border after return to normal "drop." Original measurements: Mr., 4.3; Ml., 11.5; total, 15.8 cm. Final: Mr., 4; Ml., 6.5; total, 10.3 cm.

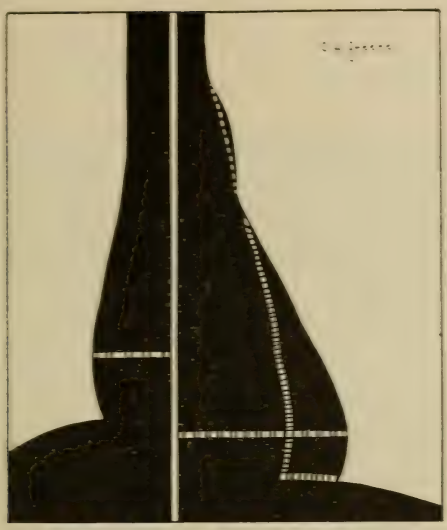


FIG. 302.—Dilated "drop" heart. (Outlines drawn from original roentgenograms by E. Brill.) Note small transverse area (12.2 cm.) of the heart even when dilated. Vertical white dotted line shows left border after treatment. Cause of dilatation unknown in this case. Right border of "drop" heart is ventricular not auricular as in normal heart. No objective symptoms of incompensation. Exertion dyspnea. Original measurements: Mr., 4.0; Ml., 8.2; total, 12.2 cm. Final: Mr., 4; Ml., 5.7; total, 9.7 cm.

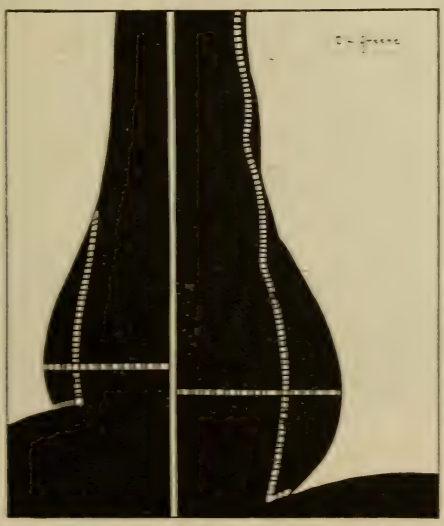


FIG. 303.—Dilated "drop" heart. (Drawn from original roentgenograms by E. Brill.) Perhaps the commoner acute type, showing decided involvement of the right ventricle. Case of influenza with marked prostration. Premature resumption of activity. Vertical dotted white lines show right and left borders after subsidence of dilatation. No objective signs save a soft apical systolic *bruit* and *exertion* dyspnea. Complained of persistent sense of fatigue drowsiness during the day. Insomnia at night. Original measurements: Mr., 6.3; Ml., 8.0; total, 14.3 cm. Final: Mr., 4.81; Ml., 5.8; total, 10.6 cm.

its wide recognition by his European colleagues, find sits expression in a protean and kaleidoscopic symptomatology which satisfies a large portion of the terminology of several hitherto obscure syndromes, of which it constitutes apparently the basic factor.

Clarifies obscure complexes.

FIG. 304.—Recession of borders of a dilated "drop" heart. Figures 304 and 305 show the remarkable retraction attending the treatment of a dilated "drop" heart. The case was one of influenzal bronchopneumonia of long duration. Patient was ambulant when first seen by the author on February 17. Heart sounds short and sharp. Systolic bruits at apex and in pulmonary area. Apex beat diffuse. Systolic retractions over right ventricle. Pulse extremely labile but regular. Radiography showed a heart 16 cm. in total transverse diameter. On March 20 a second radiograph showed a reduction to 13 cm. and a third taken on June 2, a further reduction to 9.5 cm. (Fig. 305).

The "hanging" heart, "cor mobile."

The Heart of Congenital Asthenia.—*If dilatation and hypertrophy are absent, these patients show usually a misleadingly narrow, attenuated, extremely mobile, more or less low-lying, "hanging," heart (tropenform Herz), often asso-*

ciated with a low diaphragm, arterial hypoplasia and marked vasomotor and general circulatory instability and lability.

During periods of impaired nutrition or toxemia the heart muscle is *singularly deficient in sustained tonus* or in any tendency to undergo decided

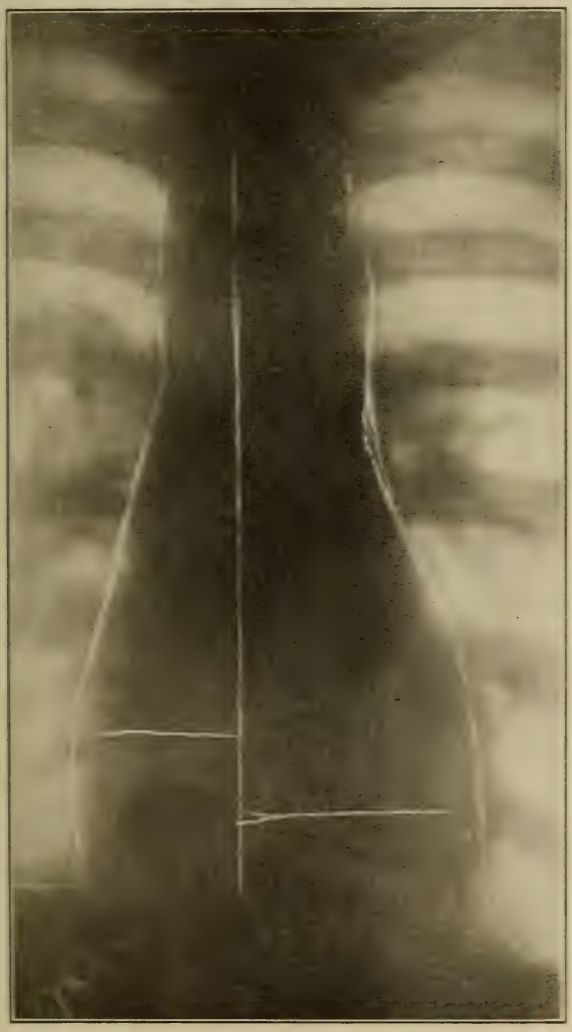


FIG. 305.—Resumption of an approximately normal outline on June 2d and full revelation of the "drop" heart. The peculiar initial outline of this universally dilated, toxic, congenitally weak heart offers an interesting contrast in its upper portion to the square outline of massive pericardial effusion.

hypertrophy even when the seat of recurrent or more or less persistent minor overstrains, but the readiness with which minor insufficiency or even dilatation occurs, under physical or emotional overstrain and, especially in acute infection, with or without associated murmurs of a peculiarly transitory

Dilatability

and evanescent character, is a striking clinical phenomenon which hitherto seems to have escaped specific recognition.*

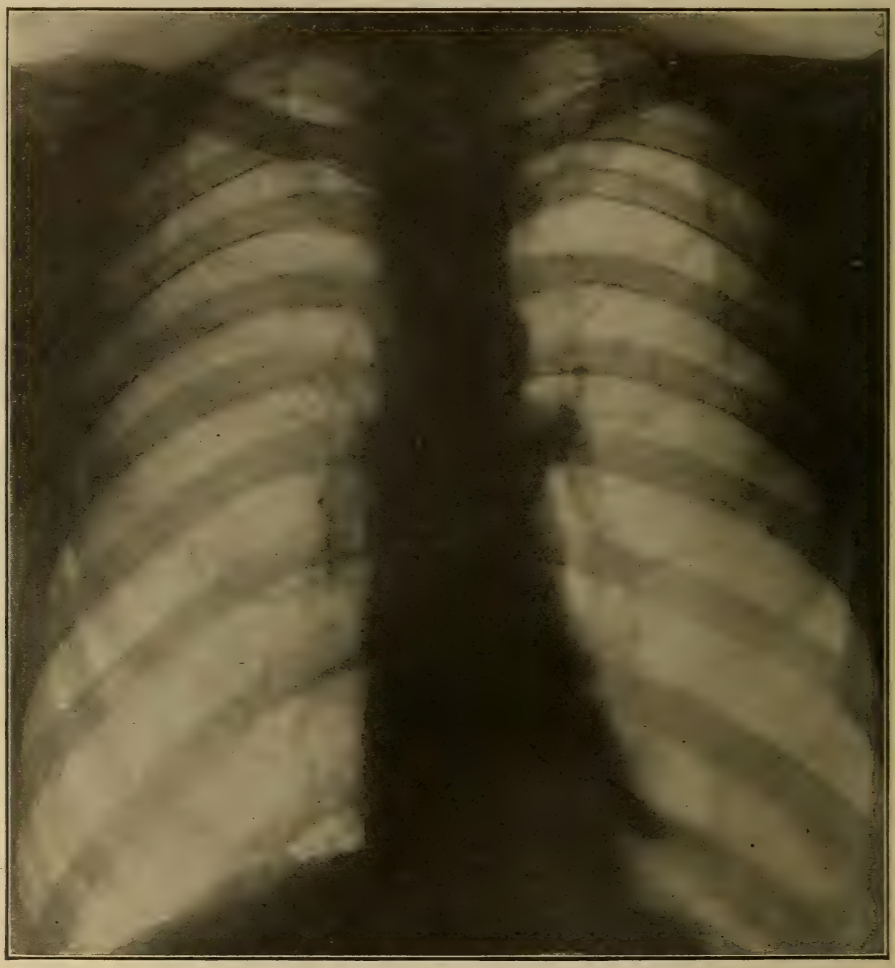


FIG. 306.—"Drop" heart of a man six feet two inches in height and of the asthenic type. He attends to business daily, but has so marked an *exertion* dyspnea as to make the ascent of a half dozen steps a cause of breathlessness. Reading aloud is difficult. Dimensions: Mr., 6.8 cm.; Ml., 2.8 cm.; total transverse diameter, 9.6 cm. There is no evidence of any attempt on the part of nature to repair the congenital defect.

The signs of insufficiency are seldom strikingly objective even in decided dilatation and the change in the cardiac profile may be either very great, as in

* So far as the author is aware attention to the direct clinical importance of this form of heart in the relationships here discussed dates from his delivery of the "Oration in Medicine," read before the American Medical Association in 1912. There is a wealth of literature bearing upon the "drop" heart under its many variants of title. But the significance of its obviously defective musculature, misleadingly common clinical occurrence, tendency to obscure its own dilatations even when decided, and direct and constant relationship to visceroptosis seem to have been disregarded. See "Prognosis in Chronic Heart Disease as Adversely Affected by Certain Medical Traditions." Jour. A.M.A. vol. 59, p. 685–690, 1912.

the case of severe acute dilatation in a prostrating acute infection or so slight as to make its recognition a matter of difficulty. In the latter cases, the quick response to rest and digitalis is illuminating.

Effect of Environment and of Sex.—The series of illustrations offered show clearly the variations in form that these hearts may assume and suggest strongly *the basic unity* of the so-called "*small heart*" and the "drop" heart.

FIG. 307.—Stomach of patient whose heart is shown in Fig. 302.

They also serve to explain the puzzling orthodiagraphic contours which have been presented, for example, by Adler and Krehbiel.*

A dilated or a dilated and hypertrophied "drop" heart may produce such outlines; a fundamentally normal heart, never.

The so-called "*small-heart*" will be found almost invariably in suggestive association with the visceroptosis of congenitally asthenic individuals and in most instances represents a dilated or hypertrophied "drop" heart.

* Archiv. Int. Med., Chicago, vol. ix, p. 346–361, 1912.

Modified forms.

The "drop" heart is *extremely common* in the female; *common* in the male, and, probably, the effect of environment and mode of life in childhood has a great effect in determining its strict adherence or relative nonadherence to the type in development.

In many if not most instances these individuals attain a degree of myocardial adequacy which enables them to lead an active life.

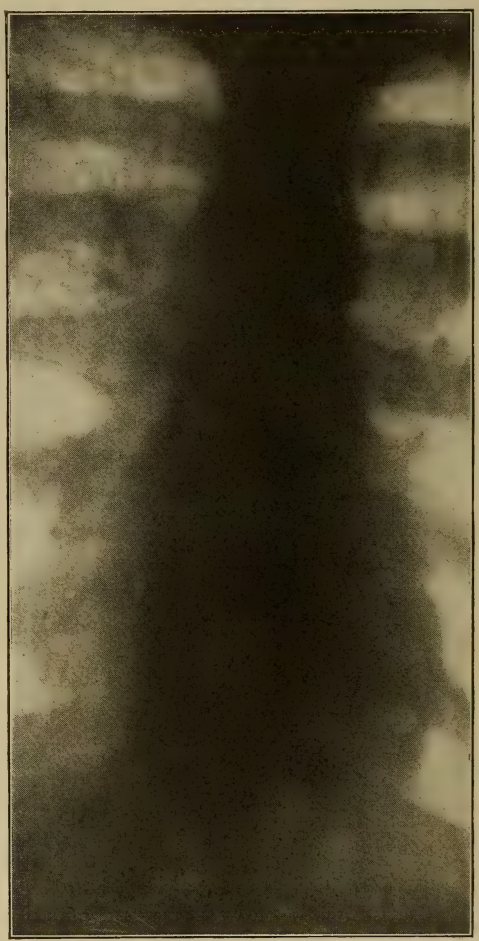


FIG. 308.—Modified "drop" heart. Case showing distinct impairment of cardiac reserve, although heart measures but 9 cm. in total transverse diameter: Mr., 3 cm.; Ml., 6 cm. A soft localized systolic murmur was present at the apex. Right ventricle is enlarged, left also probably.

To what extent the almost universally present roentgenographic signs of obsolete, latent or, more rarely, active tuberculous infection, affects the development of this type of heart is a question of interest. Such infections almost wholly, no doubt, date from childhood; for the "asthenic" tissues afford a favorable locus for the tubercle bacillus.

Misleading Bruits.—Murmurs when present over these hearts are systolic and may be maximal in the mitral, tricuspid or pulmonic auscultation areas.

The last are usually ascribed to anemia alone, but in such cases they may and often do occur in its absence or tend to persist or recur after any existing initial anemia is relieved. (See "Anemic" and "Accidental Murmurs.")

Therapeutic Test.—*In such cases the administration of full test doses of digitalis with, or oftentimes without, enforced rest, usually causes distinct amelioration or disappearance of some or even all subjective symptoms and, not*

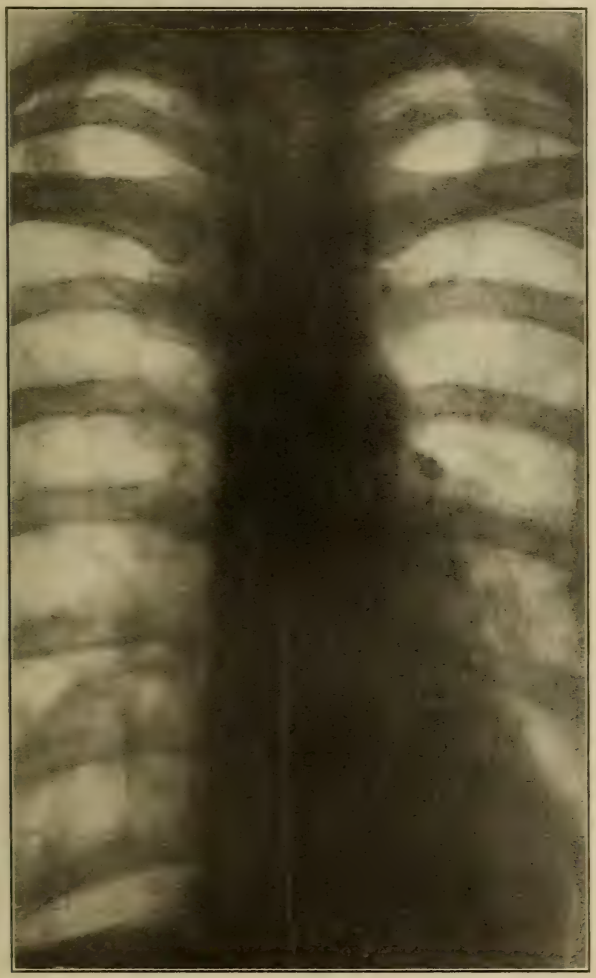


FIG. 309.—"Drop" heart. It will be noted that the right border is *hidden by the sternum*. Ml. in this case is 5 cm.

infrequently, a more or less decided but demonstrable shrinkage in the cardiac area of what may have seemed originally to be a heart of normal dimensions, but which proves to have been a dilated "drop."

The cardinal point.

As previously stated, a "normal" transverse diameter, as usually defined, represents in the heart of the congenital asthenic an enlargement, or, in

other words, the heart that is normal for the congenital asthenic is abnormally narrow for the individual of good physique.

Deceptive measurements.

The undilated and non-hypertrophied asthenic heart may measure no more than 7.5 cm. in total transverse diameter in thin subjects and 9.5 to 10.5 cm. constitute the usual normal.

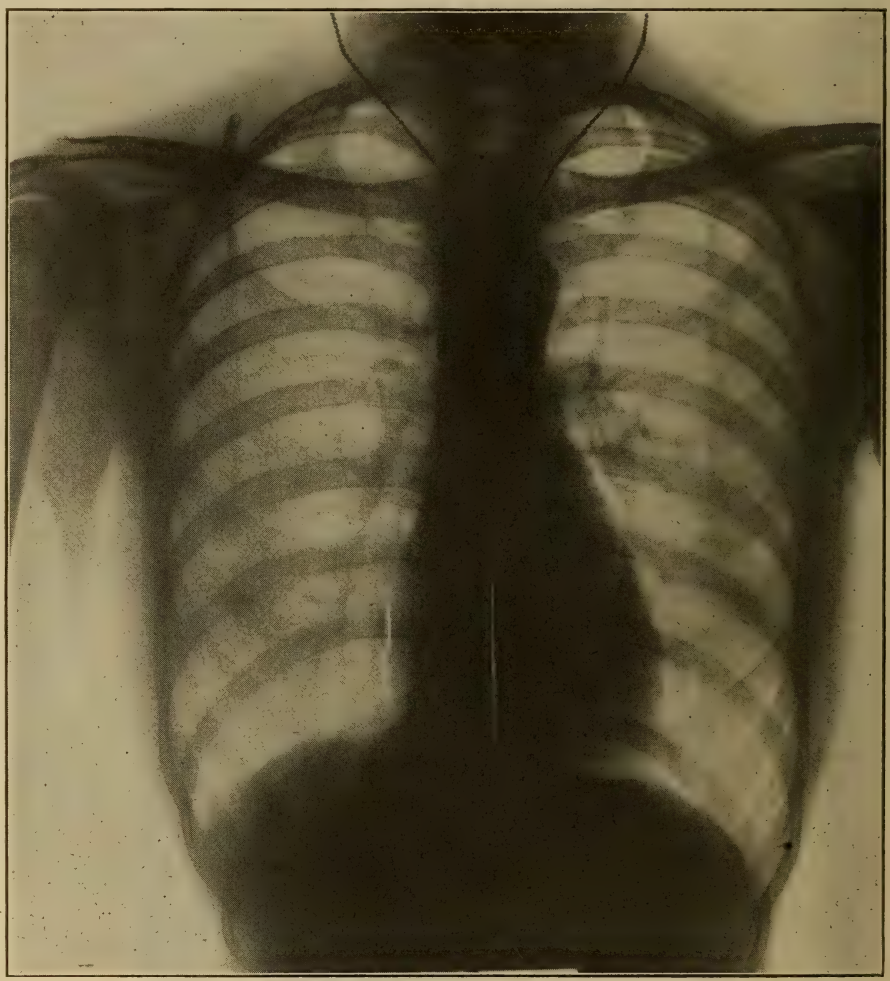


FIG. 310.—A modified "drop" heart associated with a soft mitral murmur and distinct signs of minor incompensation. A common form. Heart dimensions: Ml., 6 cm.; Mr., 4 cm.; total transverse, 10 cm.

Occurrence of Symptoms.—Possessors of "drop" hearts even of the narrowest type need yield no subjective symptoms or physical signs abnormal for the (abnormal) individual unless an acute infection, emotional shock, or actual and unusual physical overexertion initiates it. Furthermore, their resistance to such influences seems to depend to an amazing degree upon the state of nutrition of the individual, all well-marked and physically unregenerate asthenics being nutritional barometers.

Such hearts often adequate.

In addition to subjective symptoms due to cardiac inadequacy these individuals, as patients, show a wealth of obscurant subjective symptoms resulting from their general structural deficiencies, and their chronic tendency to subnutrition and psychasthenia. A congenital muscular weakness and relaxation is present, which, as manifested by uterine displacements, loose kidneys, gastroptosis, intestinal kinks and the like, have afforded a large, though usually a thankless field for surgery.

Unstable nutrition.

FIG. 311.—Stomach of same patient showing high grade gastroptosis. These cardiac and gastric types are apparently well nigh, if not wholly, inseparable.

Constancy of Concurrence of "Drop" Heart and Visceroptosis.—So constant is the concurrence of this *cardioptosis* with abdominal visceroptosis that, finding a decided "drop" heart, one may assume with certainty a demonstrable gastroptosis and enteroptosis, and *vice versa*. Within slightly varying limits the degree of ptosis in the one is reflected in that of the other.

Frequency of Occurrence.—One who is constantly using roentgenography as a part of the routine examination of patients, will find the "drop" heart in

Extremely common.

some of its various forms just as common as is the abdominal visceroptosis with which it has run parallel in the author's experience.

A bastard syndrome.

"Neurasthenia."—In this connection the author ventures the hope that the term "neurasthenia," which, though professedly a descriptive name for a supposedly concrete ailment, at present serves to obscure the true nature of so many of these cases, will either drop out of medical literature or be con-

FIG. 312.—Modified "drop" heart. Periods of decided incompensation marked by syncopal attacks, exertion dyspnea, and precordial and epigastric distress. Total transverse diameter, 9.5 cm.

fined to the extremely few cases left without a more definite assignment under the application of modern diagnostic methods. (See "Neurasthenia.")

Psychasthenia.*—Recurrent or long-persisting subjective mental fatigue and excessive psychic irritability are the logical symptomatic results of a basic condition in which a constant tendency toward poor circulation, chronic

* The term is used in its literal sense.

subnutrition, toxemia, anemia, and secretory anomalies of the digestive tract and ductless glands, play the important part, and an hereditary structural and constitutional disposition to psychic instability furnishes a fertile soil for the ready production of diverse and often *bizarre* symptoms.

The Rest Cure.—Fortunately the "*rest cure*," so widely and effectively employed under the diagnosis of "neurasthenia," exactly suits the needs of

FIG. 313.—Same case showing associated marked gastroptosis. Usual accompaniment of "drop" heart.

the asthenic cardiopath from the standpoint of both nutrition and cardiac muscle tone, as indeed it fits the therapeutic needs of a vast number of other chronic diseases.

SOLDIER'S HEART.—For the past ten years the author has been trying to emphasize the great clinical importance of the structural peculiarities of these people with relation to the *heart itself*. In 1915, in making a revision

A fulfilled prediction.

of his "Medical Diagnosis" he predicted that a vast number of such cases would arise in army service and prove an embarrassment to the carrying on of military operations and also outlined the clinical picture which such individuals would present.*

"Civil War" Cases.—These congenitally underpowered, functionally unstable persons are born with defective or potentially inadequate (*not diseased*) myocardia and furnish the most typical examples in army service of that condition described originally by the elder DaCosta during our Civil War as the "irritable heart of the soldier," and better and much more aptly by Dr. Hartshorn, also of Philadelphia, as "*heart exhaustion.*"

"Heart exhaustion."

Basic Factors.—During the war just past great numbers of cases of profound myocardial exhaustion were denominated "*soldier's heart,*" "*neurocirculatory asthenia,*" "*neurocirculatory myasthenia,*" or "*effort syndrome,*" but fundamentally the larger and more interesting group was composed of victims of universal congenital asthenia wholly free from any actual disease of the heart yet showing abundant evidence of myocardial exhaustion. The condition of the heart in these soldiers differed in no essential feature from that encountered by the author in civil practice during the past decade, save that the degree of decompensation present was much greater on the average.

Heart disease may be lacking.

The Commoner Symptoms.—The cardiovascular symptoms present in these "soldier's hearts" are simply those of myocardial exhaustion or impairment of various degrees accompanied no doubt in many instances by those of an acute dilatation which in all probability was recognized as such only in the rarest instances because of the initial narrow diameters of the "drop" heart and its modification. These may be summarized as follows:

Myocardial exhaustion.

(*a*) *Persisting sense of fatigue* or an unusual fatigability.

* "The elements of shock, emotional crises and physical exertion which enter so greatly into modern warfare must produce acute heart strain in vast numbers of young soldiers of the slender congenitally asthenic type. One may venture to predict that the condition will prove to be extremely common in the European war and that it will fall upon this type of individual in a large proportion of the cases. This being the case the clinical picture presented will be one of profound physical exhaustion, a high pulse rate, without any marked constancy of arrhythmias save of the juvenile type, except there be a history of antecedent "trench fever," "rheumatism," "tonsillitis" or antecedent organic heart disease.

Arrhythmias should occur chiefly in the older groups in whom antecedent degenerative myocardial processes might be anticipated.

In young asthenics high grades of functional nervous disturbances might be expected.

The heart outline in the older or previously tainted individuals might be abnormally large.

In members of the asthenic group it should seldom exceed greatly the normal maximum diameter (13 cm.), by reason of the fundamentally small size of the "drop" hearts which constitute their normal.

Both subjective and exertion dyspnea should be present initially and vertigo, faintness or actual syncopal seizures would be noted at the onset of the attack in many instances.

The symptoms of more or less profound psychasthenia might be assumed as inevitable in a large number of cases."

(*b*) *Dyspnea on exertion* or, in the severer cases, more or less persistent even when at rest.

(*c*) *Vertigo* in the more extreme cases.

(*d*) *Discomfort* and, in the more acute cases, *actual precardial pain.* This sensation varied from a mere sense of substernal fulness, crowding or pressure, dull aching, to actual angina of the severest type.

(*e*) *Syncope.* Syncopal attacks seem to have occurred with far greater frequency than would be the case in civil practice, no doubt because of the more complete and acute exhaustion sustained by the myocardium under service conditions.

(*f*) *Tachycardia.* As might be expected, tachycardia was a symptom very generally encountered and this sometimes persisted over long periods or even after treatment had been abandoned. On the other hand, persistent arrhythmias seem to have been relatively infrequent.

Heart murmurs, if present, were slight, often transient, systolic, and of the type usually termed functional or accidental.

Failures in Primary Selection.—In 1917, when acting on a Cardiovascular Board, the author found and reported that we were passing large numbers of these congenital asthenics into all branches of army service who could not prove otherwise than a burden to any command, but practically no recognition had been given to this clinical group and consequently there were no rules of selection in effect adequate to protect the army from their undesirable presence.

Non-recognition.

As is now known, to an astonishing degree they proved an element of embarrassment in the conduct of military operations, many breaking at the outset and during training, others going on and yielding only under the stress of actual front-line service.

Its result.

Vital Points.—Surprisingly, one finds even in the recent literature little understanding of the fundamental *structural and functional inferiority* which pertains to this most interesting body of men and women.

The author would especially lay stress upon the fact that, although in them as in other individuals, *any and all kinds and degrees of organic myocardial or valvular defects* may occur, yet the *interesting and outstanding feature* is the relative ease with which varying degrees of myocardial insufficiency, from slight to grave, may be produced in the *absence of actual disease of the heart muscle*, its valves, or its blood vessels, by causes insufficient to affect adversely the hearts of sthenic individuals.

Adaptation and Self-protection.—Furthermore, a large proportion of these congenital asthenics, though born under a handicap, achieve through favorable environment an amount of myocardial reserve and a degree of functional sufficiency which does not permit us to distinguish them from the more favored individuals of our general population except by the detection and assessment of certain stigmata to be referred to later.

In *civil life* the congenitally asthenic individual in many instances consciously or unconsciously acquires the habitude of self-limitation of effort and self-protection from adverse influences which threaten his

Self-protection.

inadequate myocardial reserve. *In the Army such self-protection was impossible.*

Period of Breakdown.—The cases which figure so largely in the medical reports of all armies engaged in the "Great War" fall naturally into two groups.

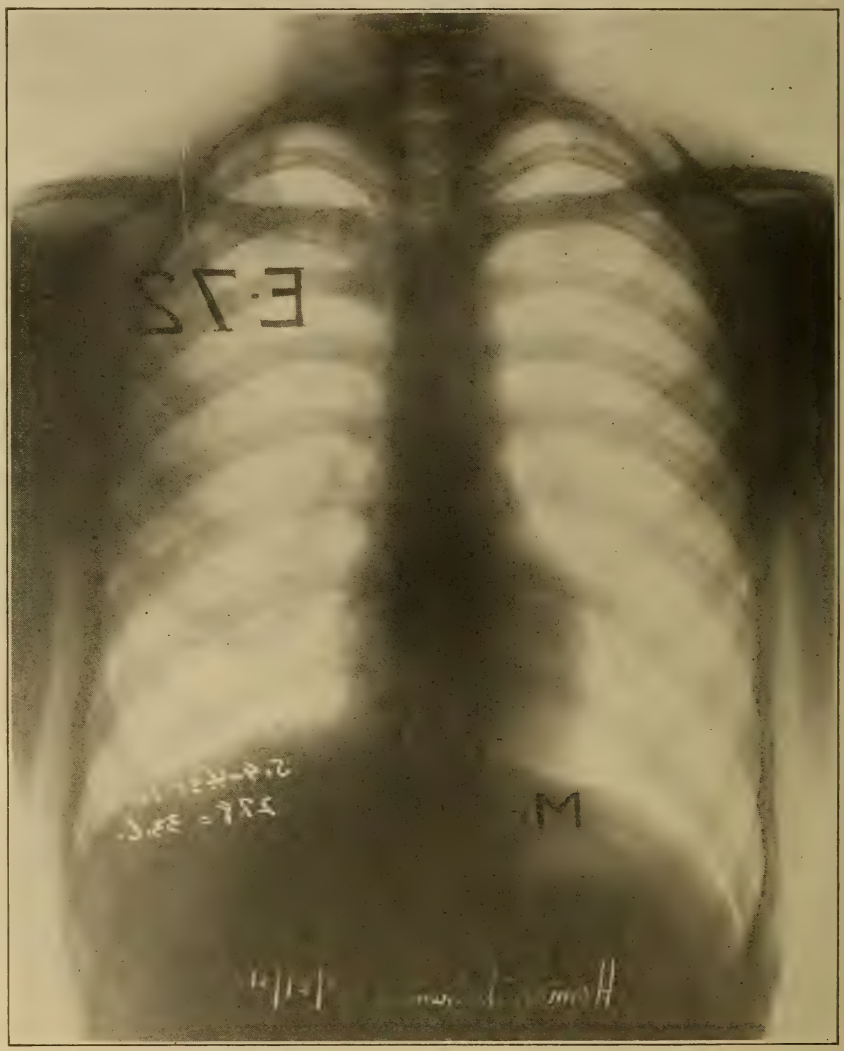


FIG. 314.—(E 72). So-called "soldier's heart" in civilian patient. A typical example of what is so frequently described as the "small" heart. Is actually an enlarged drop heart. The electrocardiogram shows marked right ventricular preponderance.

1. The particularly unstable group which broke down at the outset.
2. Those who "carried on" until their greater yet fundamentally lessened reserve yielded to the tremendous strains imposed upon them in actual service or in combat warfare.

It may be added that this same group of individuals furnished a large proportion of the persisting psychoneuroses which were returned to our country and to others for treatment.

Leading Characteristics.—The chief characteristics of this group, aside from the presence of the "drop heart" or its modifications (usually described as the "small" heart), are:

FIG. 315.—"Drop" heart in patient suffering at intervals from definite but not extreme decompensation. All through life there has been conscious self protection from overexertion. No murmurs were present even during periods of decompensation. Electrocardiogram shows right heart preponderance.

(*a*) A slender build, in general representing what used to be called the "pre-phthisical habitus," with slender bones, slender and oftentimes poorly-

developed muscles, an especial flabbiness being present in many cases in the abdominal musculature.

(*b*) Most of them are possessed of a long and narrow or flattened thorax with a sharp or relatively sharp intercostal angle.

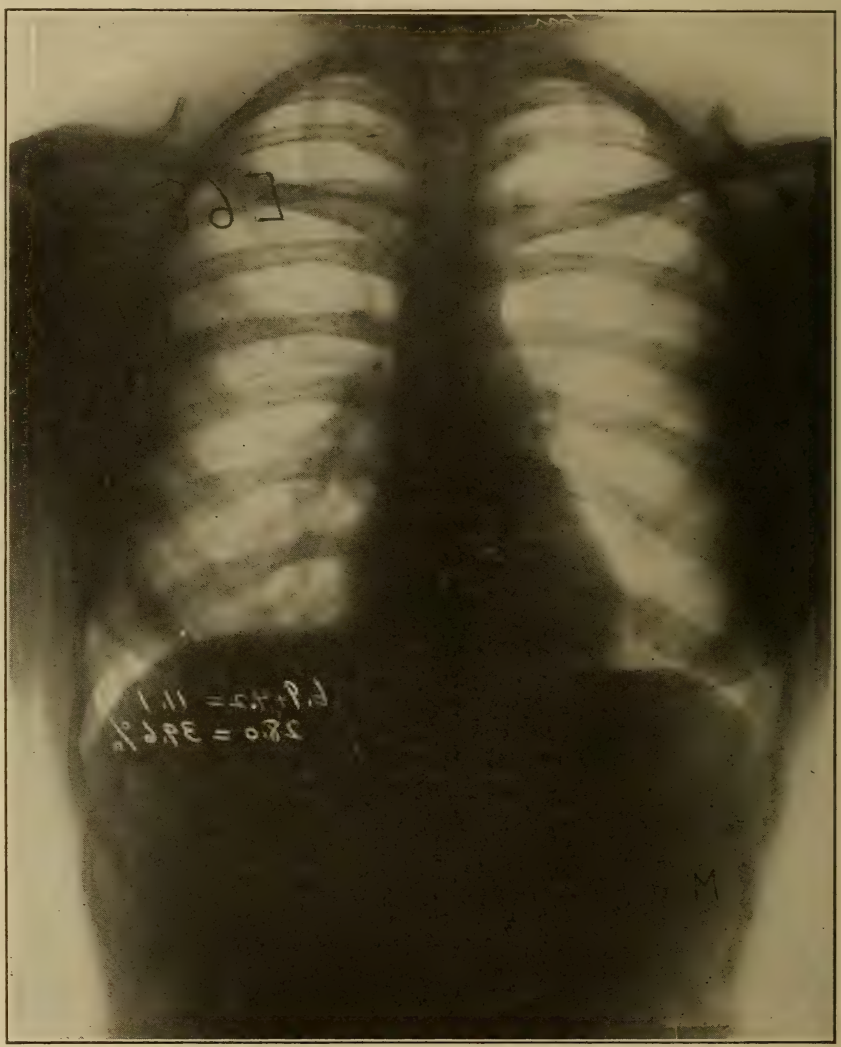


FIG. 316.—Enlarged and insufficient "drop" heart. Total transverse diameter 11.1 cm. The type of heart frequently miscalled the "small" heart. Skeletal musculature in this young woman unusually developed. Abdominal muscles relatively flabby. Marked gastroptosis present with atony. Dyspnea on exertion pronounced; fatigability decided. Patient has been very strong with respect to individual and brief physical effort but has lacked endurance all her life. Such hearts at present are almost invariably passed as normal by the physician or life insurance examiner.

This does not mean that such individuals are necessarily meagre, emaciated, or underweight. Indeed many are "heavyweights" and some carry an amount of adipose tissue which tends to obscure their structural peculiarities.

(*c*) All are *predisposed* to the curious instability of function which is universal, and to another peculiarity of the utmost importance in connection with war, namely, *a remarkable dependence upon nutritional reserve* and a peculiar instability of this reserve. Furthermore, *they are especially vulner-*

Two vital factors in causation.

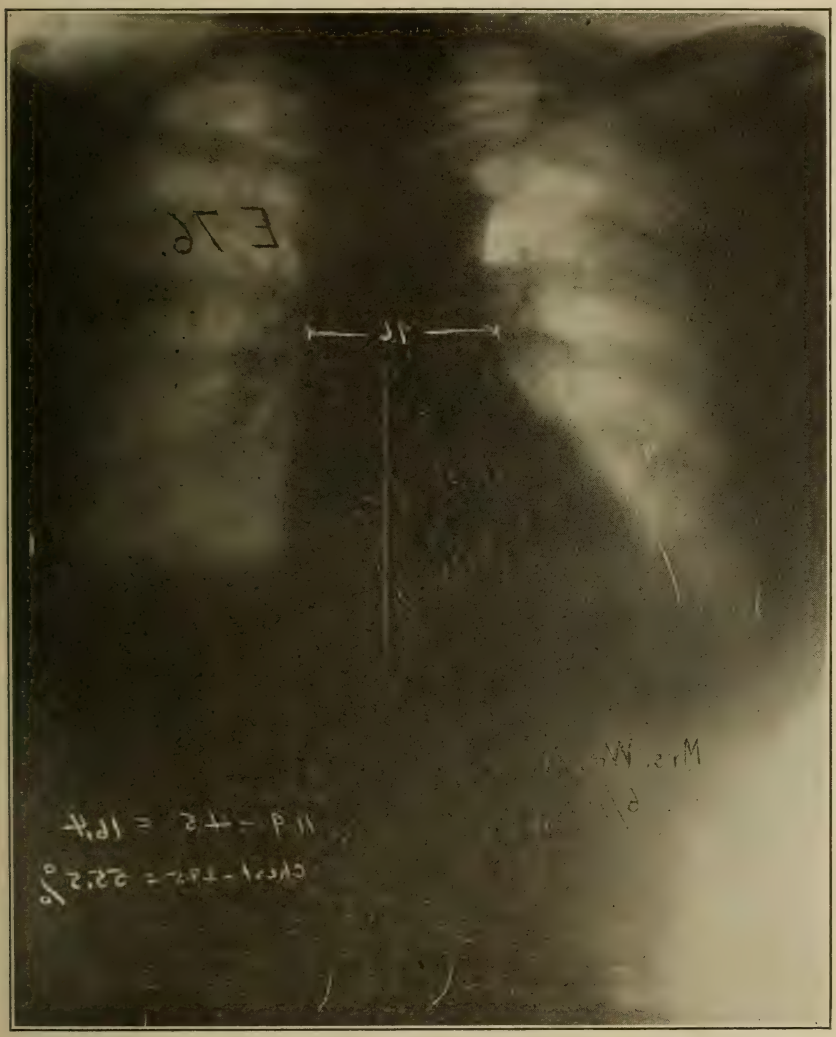


FIG. 317.—Markedly dilated "drop" heart the seat of myocardial degeneration. Symptoms of decompensation marked during preceding six years. No murmurs present. Gastric disturbance a prominent feature. Dyspnea on exertion, constant fatigue, slight edema of lower extremities, marked tenderness over left heart border. Gastroptosis decided. Patient a woman, weighs 200 pounds. Under treatment the diameter of this heart (16.4 cm.) was reduced to 13.8 cm. as shown in Fig. 318. It will be noted that the original "drop" heart contour has been measurably resumed although both right and left ventricles are hypertrophied. Such silent degenerated "drop" heart are especially deceptive and misleading.

able with respect to infections, and well-defined cases convalesce slowly and oftentimes imperfectly from such infections.

(*d*) The great majority of them go on without any symptoms so long as they are well-fed, well-nourished, and well-environed. Reverse this and they become subjectively, or, less often, frankly ill.

(*e*) More vital, from a diagnostic standpoint, than these traits just named is the presence of universal visceroptosis in varying degrees.

FIG. 318.—Same heart as shown in Fig. 317 showing shrinkage of outline under treatment and the approach to the original "drop" heart outline, which has been obscured by the dilatation existing over a long period of time.

Visceroptosis.

Many years ago the author pointed out that, with rare exceptions, in fluoroscopic examinations one might make an immediate diagnosis of "drop-heart" in examining the abdomen and finding a gastroptosis or decidedly movable kidney. So on the other hand, finding the heart of congenital asthenia, one could accurately affirm the presence of a ptotic stomach.

One must not understand that all cases of so-called "effort syndrome," or "soldier's heart" are congenital asthenics. The symptoms characteristic of this syndrome are merely those of profound myocardial exhaustion, extreme narrowing of reserve, and may be produced most readily in these individuals congenitally unfit or potentially so.

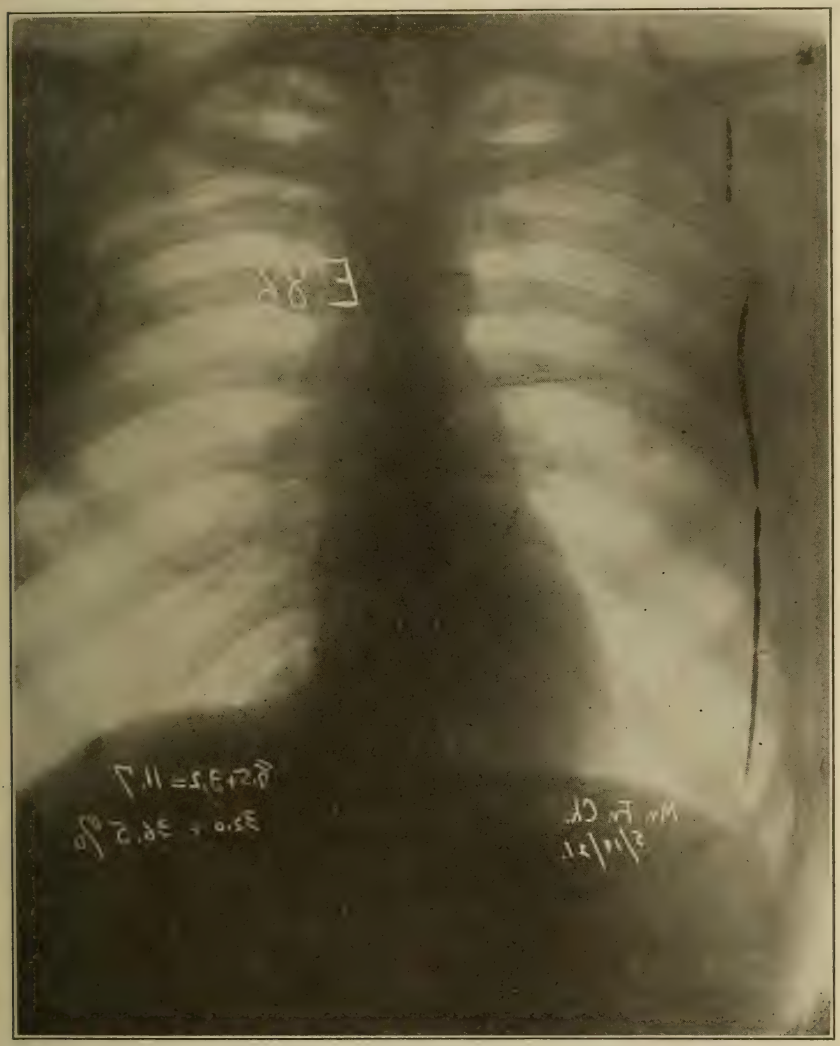


FIG. 319.—(E 86). "Soldier's heart" in civilian male. Type of individual who in the Army broke down under the severer stress and strain of actual campaigning. This man had lead an active out-of-door life as a ranchman but following severe overstrain complained of syncopal attacks, precordial pain on or following exertion, marked fatigability, recurrent edema of ankles and feet, and intervals of arrhythmia apparently due to recurrent fibrillation.

Note that total transverse diameter of heart at this time was but 11.7 cm. The gastroptosis present in this case is shown in Fig. 320. (See also Fig. 321.)

On the other hand any and every cause operating upon any heart and adequate to produce a marked diminution of tonus may produce exactly the

same symptoms, for *these are identical with those shown by individuals carrying normal hearts but subjected to intense exhausting myocardial overstrain.*

Indeed, the recognition of the "soldier's heart" rested largely upon the ease with which these symptoms were produced under physical effort, mental strain, or both combined in individuals lacking the signs of frank organic heart disease.

FIG. 320.—Gastroptosis associated with insufficient drop heart shown in Fig. 319.

Obligatory Exposure to Adverse Influences.—The adverse influences operating upon the victim of universal congenital asthenia who had been inducted into army service varied greatly with the type of service demanded of him but embraced on the whole every factor calculated to break down

the reserve of a considerable proportion of the young adults carrying this constitutional defect.

As has been stated, the element of self-protection often unconsciously plays a large part in the ability of large numbers of the congenital asthenics to endure such stress and strain of life as falls upon them, but when brought

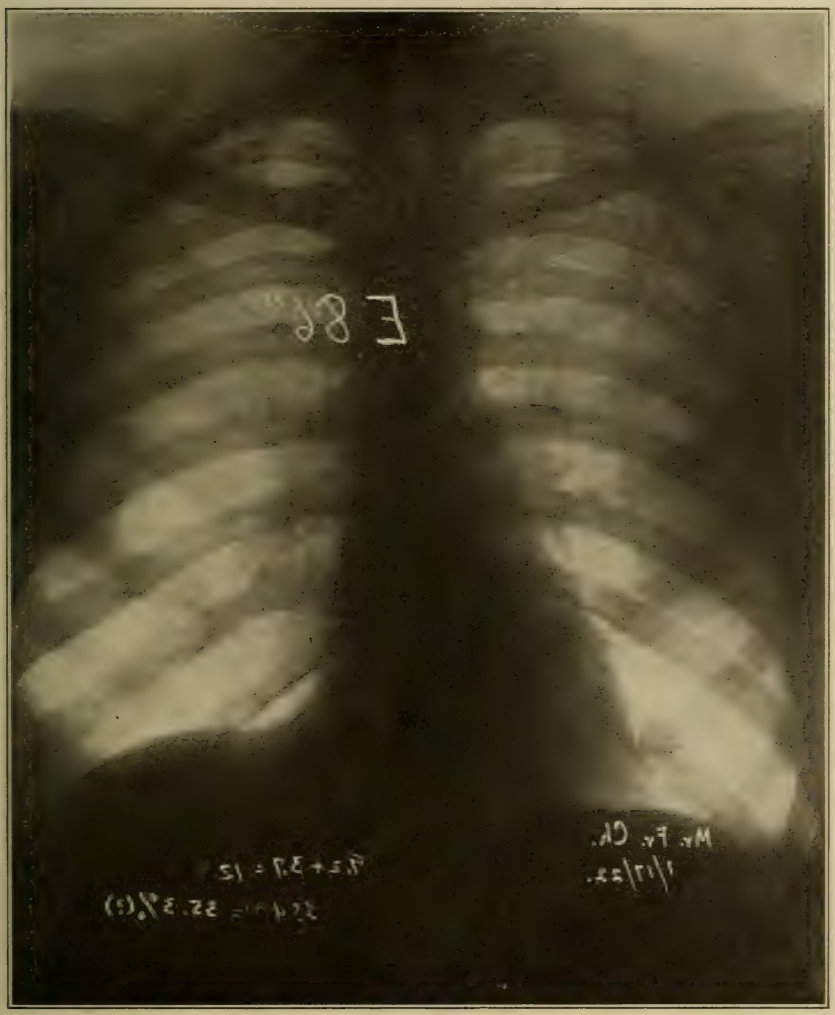


FIG. 321.—Same heart as shown in Fig. 319 showing increase of diameter to 12.7 cm. following the undertaking of heavy work against physician's orders. This enlargement was associated with a renewal of the old symptoms which had disappeared under treatment.

Note that even when thus dilated the heart falls well within the so-called "normal" transverse cardiac diameter.

into army service these safeguards, consciously or unconsciously established by them, were of necessity abolished. As a result large numbers could not withstand the "hikes" and drills even of the opening days or weeks of their period of military training. Others with greater reserve weathered the

training period only to become unfitted for service under conditions of actual campaigning across the water or the yet more strenuous demands, psychic and physical alike, of actual fighting at the front.

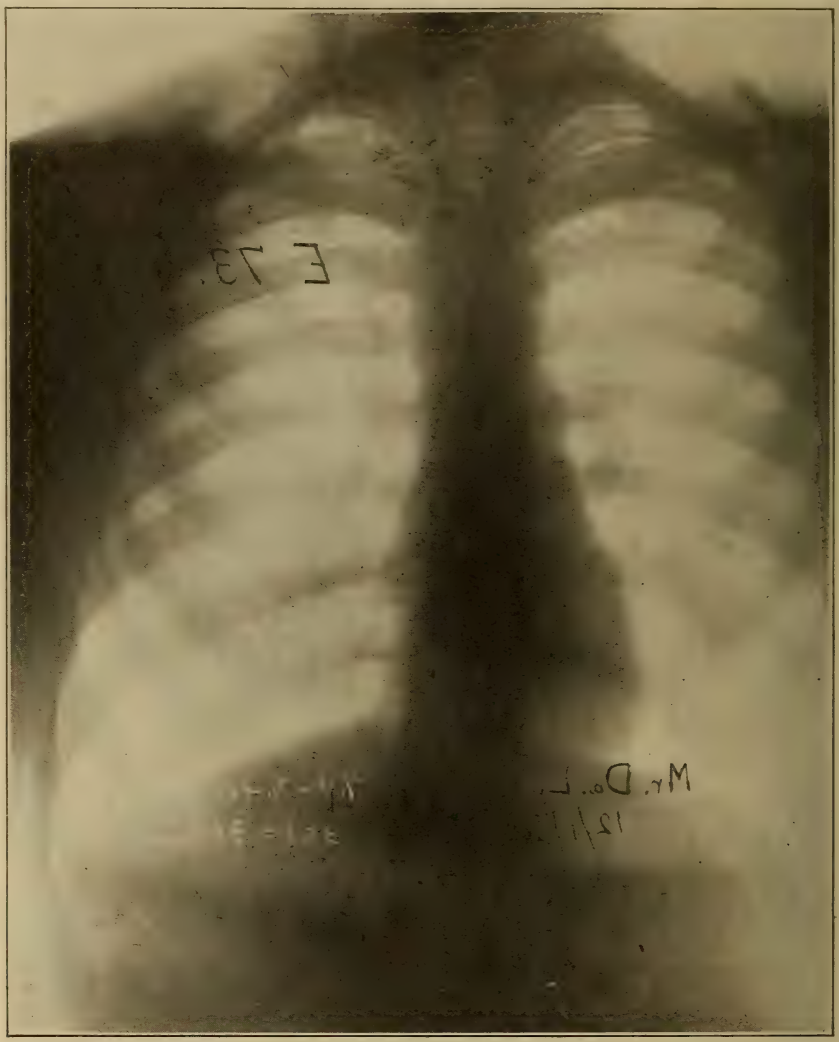


FIG. 322.—"Soldier's heart" in civilian. Another example of the "drop" heart which played so important a part in the cardiac disabilities of our troops in Army service. This individual was able to do heavy farm work up to the age of 18. Collapsed while working in hayfield during very hot weather. At the time this exposure was made, showing a heart only 11.7 cm. in total transverse diameter, decompensation symptoms were pronounced, there being marked dyspnea on exertion, subjective persisting dyspnea and precordial discomfort. Patient's recovery was reasonably prompt under treatment and all active symptoms disappeared. Nevertheless his reserve is permanently impaired and heavy work cannot now be undertaken.

Myocardial Toxemia.—Aside from this, it should be remembered that a very large proportion of these individuals carried concealed septic foci or larval tuberculous infection such as might be, and often were, relighted under conditions of army service.

Furthermore, many were subjected to primary acute infections such as mumps, measles, pneumonia, and influenza and were not given that prolonged period of convalescence necessary to individuals of this type. Infections.

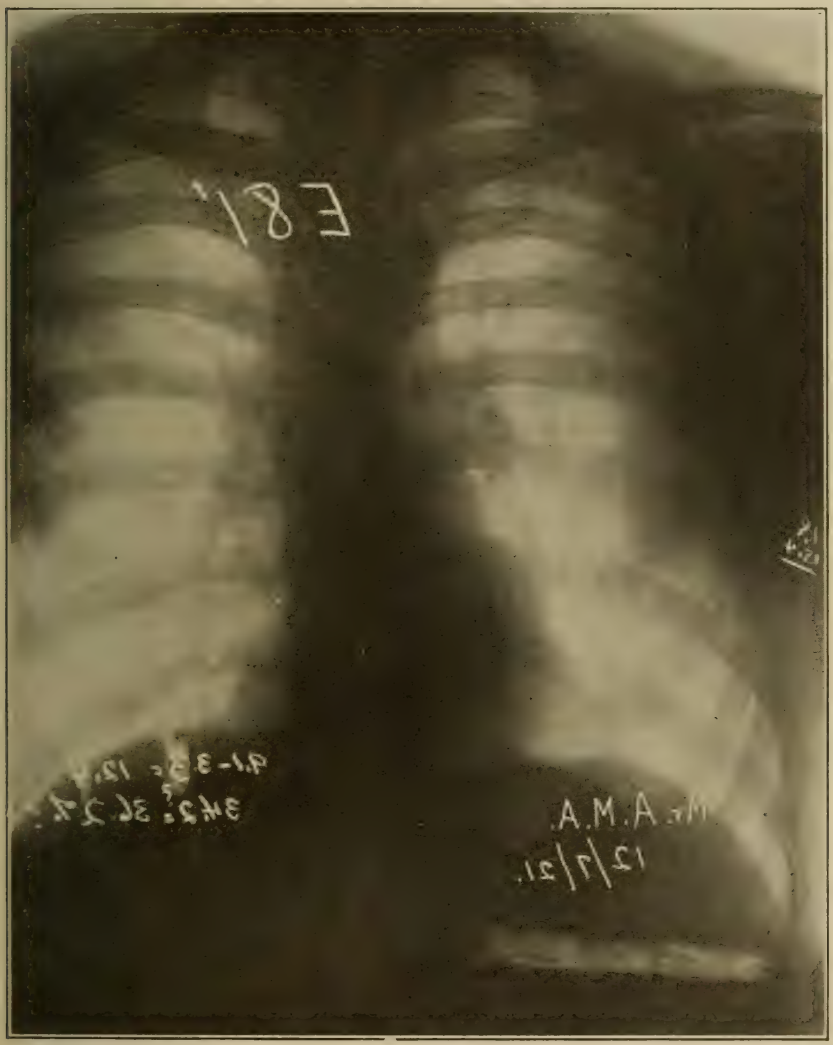


FIG. 323.—"Soldier's heart" in civilian. A type frequently encountered in Army service. Reserve capacity good but failing under great physical and mental stress, or both of these elements combined. This individual was seen first in an attack of acute dilatation associated with fibrillation; both of which conditions endured only sixteen hours as nearly as could be determined at the time. Young active man. The outline is that of a "drop" heart. There still exists (after treatment covering about three weeks) manifest enlargement of the left ventricle which probably is permanent and represents hypertrophy. This finding is confirmed by the electrocardiogram.

Condition Common to Civilian and Soldier.—The author would repeat that "soldier's heart," "effort syndrome," "neurocirculatory asthenia,"

"neurocirculatory myasthenia" not only are one and the same thing but represent conditions in no way peculiar to army service.

As stated previously, the cardiovascular symptoms presented by these cases differed in no way from thoseeencounyered every day in cival practice though in a larger proportion the evidence of exhausted myocardial reserve were more extreme and the difficulties attending its rehabilitation and restoration were correspondingly greater.

Non-recognition of dilatations.

Undoubtedly, owing to the adoption of faulty standards with relation to heart measurements, a great number of cases of actual dilatation were overlooked and, furthermore, the treatment which these men received in general was not such as would in the highest degree promote their permanent recovery.

One is not surprised, therefore, to find that a large percentage of such cases were discharged unbenefited, or but partially restored to health.

Unstable Reserve.—In the heart of the congenital asthenic there is a degree of instability with relation to reserve which makes the rebuilding of the myocardial strength a matter of the utmost difficulty. This is peculiarly true of such cases as arose in a multitude of instances during army service where, because of unavoidable and obligatory exposure to adverse influences, the exhaustion of the myocardium was unusually profound.

Fundamentals in Treatment.—In treating profoundly exhausted cases the utmost care should be observed throughout with respect to the resumption of physical activity. One must be satisfied with slow, tedious, incremental renewal of reserve power through primary rest followed by a most carefully regulated resumption of *walking* exercise. Under no circumstances should such individuals be "tested out" from time to time to a point of fatigue or the revival of any of those symptoms characterizing their condition.

According to the author's experience in civil practice, it is as easy to empty such hearts of newly acquired reserve in the earlier stages of their improvement as it is to pour water out of a glass, and the utmost care is necessary to *prevent* the patient from putting his newly acquired strength to tests long before he has reached a degree of restoration which makes this safe. He must not only be kept within the limits of his reserve during his slow progression but *always well within the safety zone.*

Another curious factor in relation to such cases with marked degrees of reserve impairment has been the deleterious effect of upper-arm exercises or marked body flexion, and in the author's private practice these are carefully avoided.

It is to be hoped that in the deplorable event of another war the rules and the means of selection may be such as will better protect the army and its various services from those members of the congenitally asthenic class who are likely to break down under the stress of war, and, on the other hand, secure to selected members of this group opportunity for patriotic service suited to their physical ability.

MINOR INSUFFICIENCIES.—*The symptoms of cardiac origin as manifest oftentimes in such cases as fall under the ailment just described, namely, those of chronic congenital asthenia with weakened hearts, throw much light on the*

FIG. 324.—Modified "drop" heart often encountered. Young man of nineteen Never robust, yet six feet tall and inclined to athletics, in which he is fairly proficient Inherits the powerful build of his mother's family and the "drop" heart of his small-boned father (radiographically demonstrated). Boys may attain a good heart despite congenital handicaps, but frequently pass through phases of partial inadequacy. Many break down on the athletic field, but only temporarily in most instances. A young man of this type with exactly the same inheritance and heart outline, stroked one of the greatest crews in this country for a year, only to break down in his second year with wide dilatation.

subjective manifestations of cardiac insufficiency of minor grades arising from more serious causes.

The peculiar clinical features of the ptotic heart and its chief relationships may be thus summarized.

The "drop" heart is merely one manifestation of what is usually a general visceroptosis, slight or decided, and may or may not constitute its dominant feature clinically and anatomically.

The author is convinced that the parallel between the "drop" heart and the "drop" stomach especially is a relatively exact one.

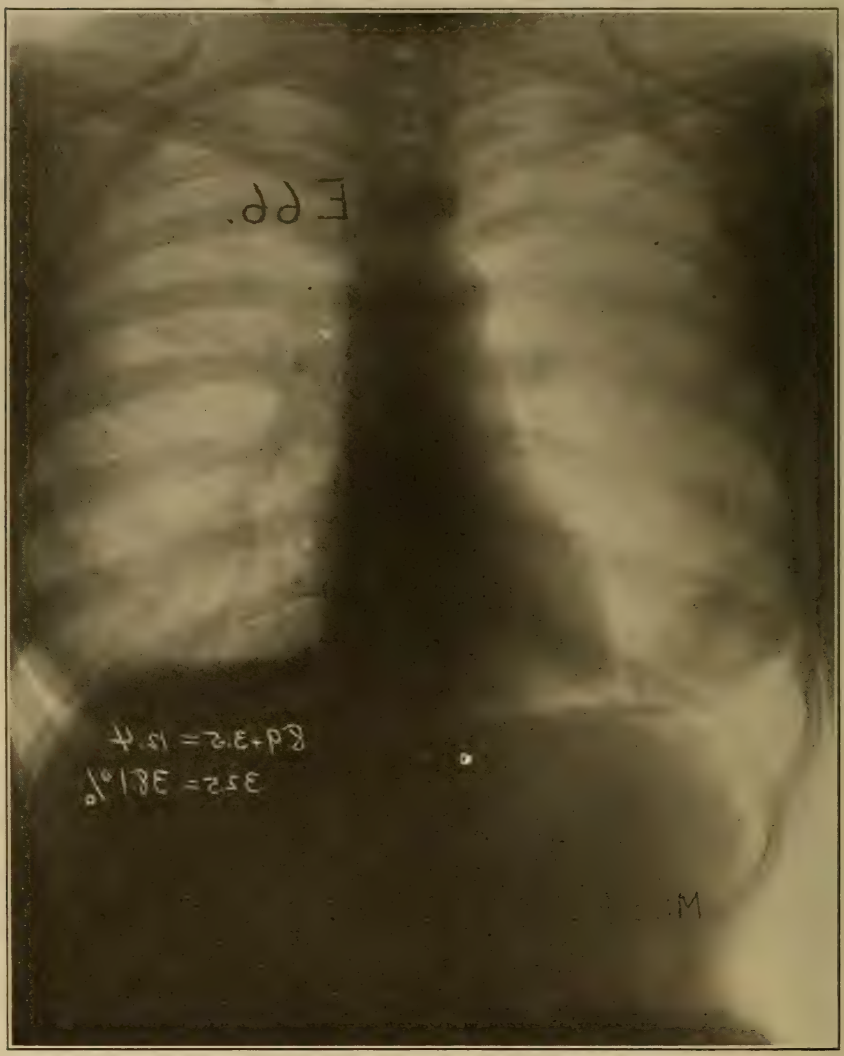


FIG. 325.—(E 66). Interesting example of enlarged "drop" heart in a female of Amazonian type, representing probably a case of crossed inheritance. General build and skeletal musculature was extraordinarily good. The symptoms were almost wholly gastric. There was slight edema of the lower extremities which had gone unnoticed by the patient. Slight exertion dyspnea also was present. Gastroptosis decided. (See Fig. 259 for comparison.)

Both are evidences of constitutional defect in great measure.

Both are extremely common but may show all gradations from the typical to that which closely approximates the normal. Both tend to show

symptoms only under conditions of impaired nutrition and in each these take the form of atony, functional instability and the production of troublesome subjective symptoms often of a most misleading character.

Both are peculiarly responsive to functional rest and other measures calculated to restore their labile nutritional balance and increase their tonus.

FIG. 326.—(E 66′). Outline of heart shown in Fig. 325 following treatment. Total transverse diameter has been reduced 1 cm. All symptoms have disappeared and the heart is shown to be of the modified "drop" type with enlargement of both right and left ventricles. This is the type of heart so often described misleadingly as the "small" heart.

Neither, of itself, is a fatal or even serious ailment and under favorable environment both conditions may be carried throughout a lifetime replete with good health and characterized by what is essentially normal activity, both mental and physical.

On the other hand, both may make their possessor trouble from childhood to the day of his or her death from some other cause.

The heart by reason of the peculiarly insistent and persistent demand upon its musculature and its abnormal sensitivity to acute and chronic

FIG. 327.—Modified insufficient "drop" heart in a heavy woman carrying beneath her fat the basic asthenic stigmata. Transverse measurement 8.5 cm.

toxemias is especially liable, remittently or persistently, to show a diminution of reserve and consequent limitation of the field of symptomless forced response.

In the heart both periods of impaired tonicity and even of actual minor

but pathologic dilatation are extremely common. See Figs. 329 and 330 and, especially, Figs. 356 and 357.

Finally, that these periods have, in the past, almost wholly been overlooked and disregarded.

Fundamental Assumption.—It is obvious that one would not await the onset of pulmonary or renal congestion, hepatic engorgement, marked dyspnea, ascending edema and flagrant and obtrusive dilatation, before feeling justified in active therapeutic interference in heart disease if he possessed the means of recognizing with some degree of certainty the minor grades of actual insufficiency. Avoidable delay.

These major symptoms represent the expression of serious or extreme cardiac

FIG. 328.—Heart of a fat male asthenic with poor resisting power and endurance. "Hanging-drop" heart typical save that it gives evidence by its contour of a musculature far above the normal for that type. Total transverse measurement only 9.2 cm.

weakness, a dangerously narrowed field of myocardial response, and, fortunately for the patient, often serve him both as danger signals and emergency brakes, but they should not constitute the sole criteria of therapeutic initiative.

Unavoidable Effects of Cardiovascular Lesions.—However chronic and slowly progressive the condition, crippling valvular lesions, progressive myocardial degenerations, sclerosis of the coronary arteries or of the entire vascular system, high arterial tension and the like, once established, after a variable but usually greatly prolonged period of adequate balance inevitably embarrass and hamper the intricate, exquisitely responsive and delicately balanced mechanism of the circulation and tend constantly or intermittently to overwork the heart and blood vessels, *narrow the field of previously effortless response* and reduce the legitimate life expectancy of the patient.

COMPENSATION AND DECOMPENSATION.—Sequence of Events in Decompensation.—This varies somewhat with the nature and site of the

Over-distention

primary or dominant lesion. If one assumes as an example the sudden production of aortic leakage, his mind's eye at once sees the diastolic back-rush of the blood from the aorta to the left ventricle, the overdistention of that elastic pumping chamber by the opposing currents represented by

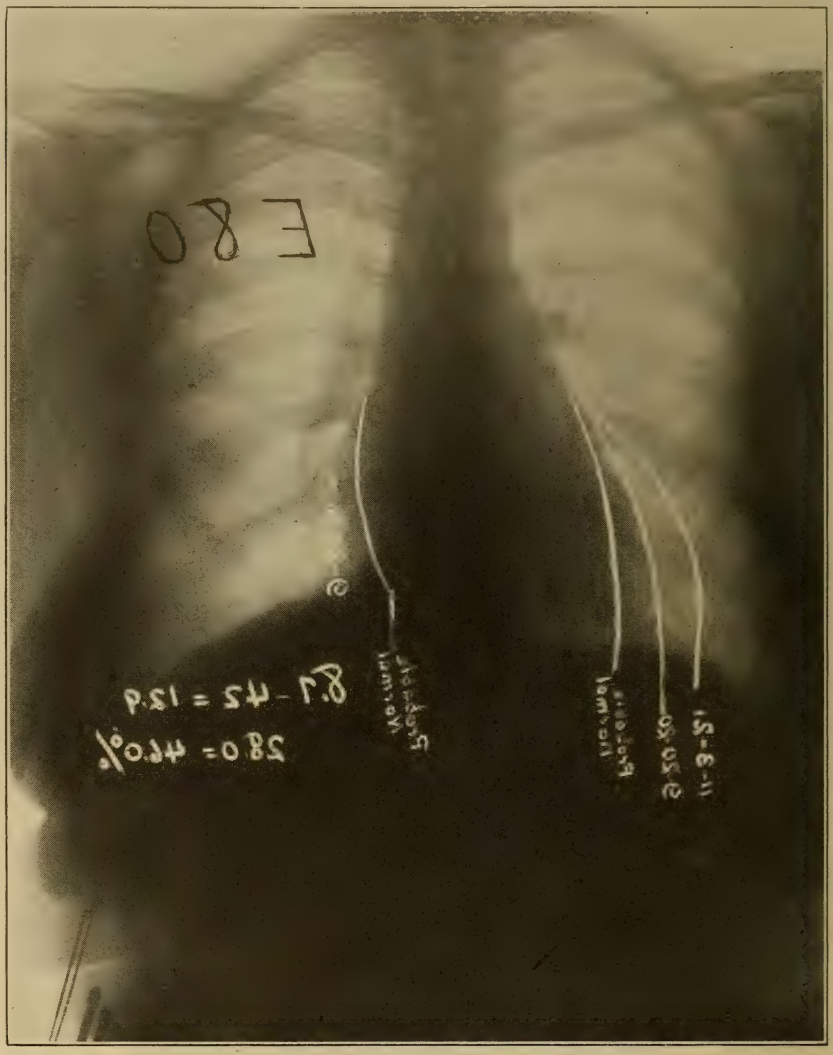


Fig. 329.—(E 80). Same heart as shown in Fig. 330. showing the reduced diameter following a short period of ambulatory treatment. The heart was originally a "drop" heart, marked associated gastroptosis being present. The outer white line shows the passing out of the left border by renewed dilatations after the patient had broken treatment and disregarded all instructions, the result being the recurrence of pulmonary congestion and other signs of minor decompensation.

that pouring through the open mitral valve from the auricular reservoir above and the abnormal inrush from the aorta below.

Dilatation

One finds primarily a *dilatation* of the left ventricle, limited and constantly resisted by muscle tonus and contractility, and usually, by an in-

creased frequency, evidently adapted to lessen the load per heart beat, and yet supply the vitally necessary nutriment to the body tissues and burn their waste products. It is evident that the intermittent shock and overaction now experienced by the aorta and to a less degree by the blood vessel throughout the body cannot but tend gradually to produce degenerative

Arteries.

FIG. 330.—E 80'). Readily dilatable "drop" heart in obese young woman. Decompensation when present manifested chiefly by pulmonary congestion. Exposure shown was made during such a decompensatory period. The total transverse diameter is 14.7 cm. See Fig. 329 for comparison.

arterial changes and a slowly progressive increasing deficiency of the peripheral circulation and of the heart itself.

Men do not often die of chronic valvular disease, per se, but of its inevitable accompaniment, myocardial degeneration.

Acquired equilibrium.

Hypertrophy is gradually superadded to dilatation and after a variable period, in young hearts at least, the increase of muscle and the dilatation limiting tonus establish a circulatory equilibrium backed by a variable amount of "reserve," often wholly adequate to hard labor, and a normal pulse

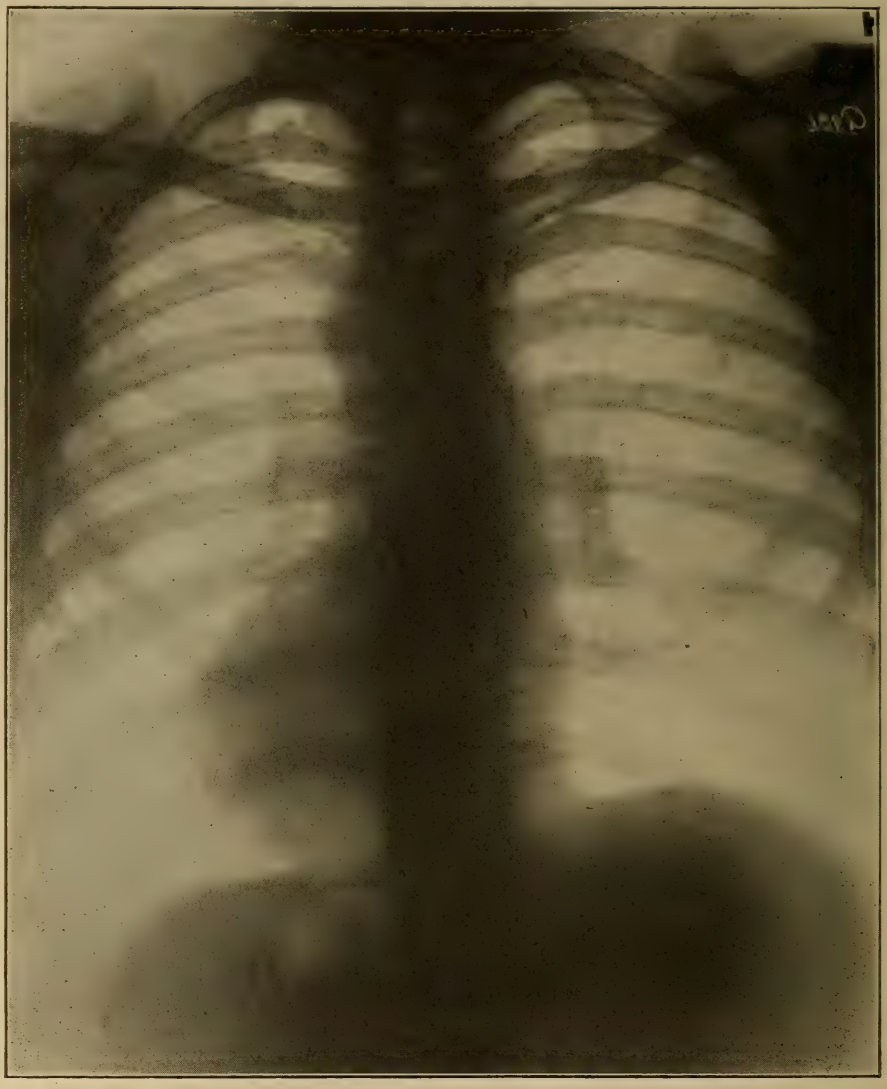


FIG. 331.—A type of cardiac outline frequently observed in high blood-pressure cases. Note underlying "hanging drop" type and extremely small transverse diameter despite the increase of left heart area.

rate. There remains, however, perhaps only in slight and unnoticeable degree, a constant slowly progressing damage in the vascular field. In such cases we inaccurately but conveniently say that "the lesion is compensated."

A Cardiovascular Paradox.—So wonderful is this adaptive change that for years a man may pursue a laborious occupation without any serious break in this circulatory equilibrium, yet he may and often does break down suddenly under some unusual physical strain, no greater, less perhaps, than

May endure for years.

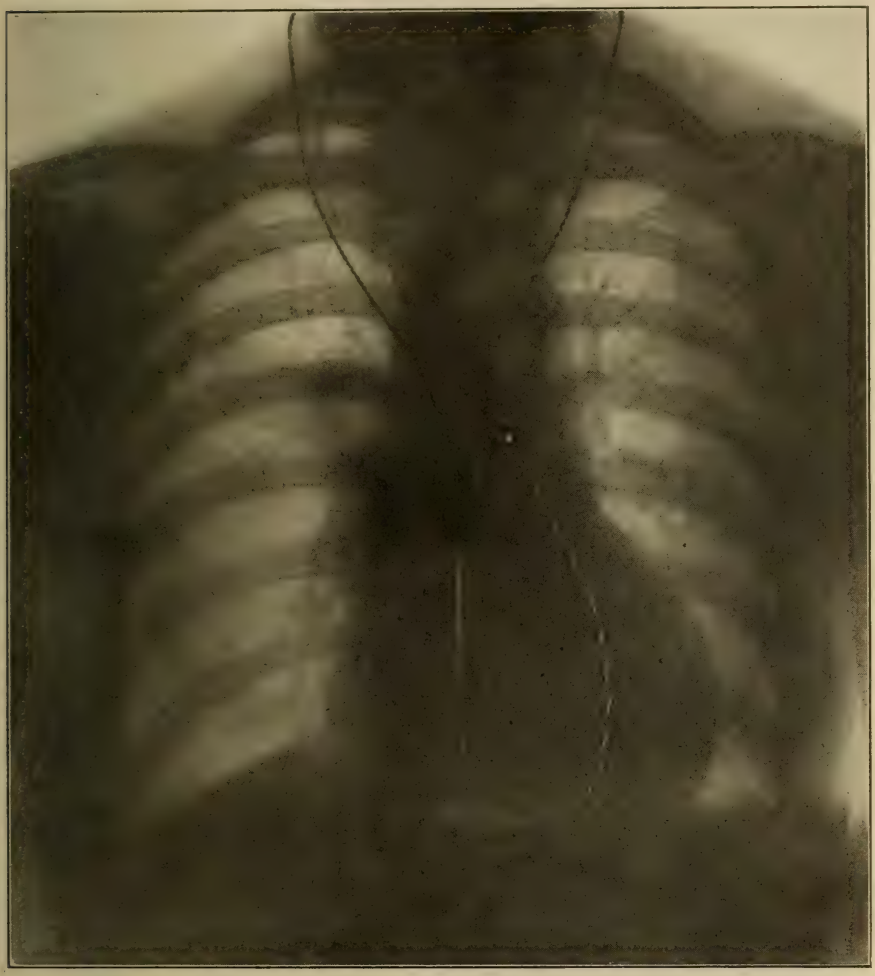


FIG. 332.—A "drop" heart, once typical roentgenographically, which has assumed a modified aortic profile during the past ten years by reason of the development of an interstitial nephritis and sclerotic changes in the aortic valve, associated with systolic and diastolic *bruits*, persistent arterial hypertension and a sustained high diastolic level. Visceroptosis and other stigmata of congenital asthenia typical; patient during many years was typical "neurasthenic" and "nervous dyspeptic;" and bears scars of several futile abdominal operations. Cardiac compensation never more than "fair." Best for several years at the time this exposure was made. Dotted line shows approximate form and position of left border before the period of change. Under author's observation twenty years. *Despite the great relative change in size, the heart measures but* 14 *cm.*, total transverse diameter. (Died of cerebral hemorrhage since the legend was written.)

that of his daily labor, to the peculiar strains and vicissitudes of which cardiac and skeletal muscles alike have become trained and habituated.

Progressive Diminution of Cardiac Reserve.—*Year by year the margin of relative safety for such patients diminishes under the effects of more or less constant myocardial overstrain, the abnormal arterial and venous pressure, the toxins of fatigue, disturbed metabolism, the constantly impaired and ever diminishing coronary blood supply, and the invariably progressive cardiovascular degeneration.*

Potent causes.

FIG. 333.—An interesting example of the dilated and hypertrophied imperfectly compensating "drop" heart. The patient's heart formerly measured about 10 cm. For the past eight years she has carried arterial hypertension 170 to 220 + and passed through a cerebral apoplexy. Weight of patient was 185 lb. but the structure was typically asthenic. The diseased and greatly enlarged heart shows a "*normal*" (13.5 cm.) measurement. An impure mitral first sound or an actual apical bruit has persisted. Slight edema is present usually, but patient is ambulant.

Direct factors.

To these constant factors one must usually add the effects of recurrent infection or toxemia from primary cryptogenetic sources or from without.

The Laboring Heart.—Certain direct decompensatory factors are common to all valvular and myocardial lesions and the chief of these are muscle strain, gradually diminishing tonus with progressive or recurrent increase both of dilatation and dilatability, and progressive susceptibility to fatigue,

FIG. 334.—Widely dilated heart of the "drop" type. Ambulant patient extremely weak and dyspneic. Case of pernicious anemia, erythrocyte count 1,525,000. Heart undoubtedly the seat of fatty degeneration and shows evidence of fatty infiltration in the shape of an illy-defined "tail" at the apex. Heart measurements—Mr., 4.5 cm.; Ml., 11.5 cm.; total transverse, 16 cm. Typical stigmata of congenital asthenia. Death six weeks later.

representing a slow but constant contraction of the field of cardiac response.

The wonderful cardiovascular mechanism can and does overcome unaided its more or less frequently recurring periods of minor insufficiency, though at a constantly increasing cost, but not without flying distress signals other and earlier than the classic symptoms of the later stages of decompensation.

Permits effective therapy.

Early Diagnosis.—We are just learning the importance and value of the early diagnosis of heart disease and of prompt recognition of myocardial insufficiency of lesser grades as prerequisite to timely and effective treatment. The aim and end of diagnosis is effective therapy and no department

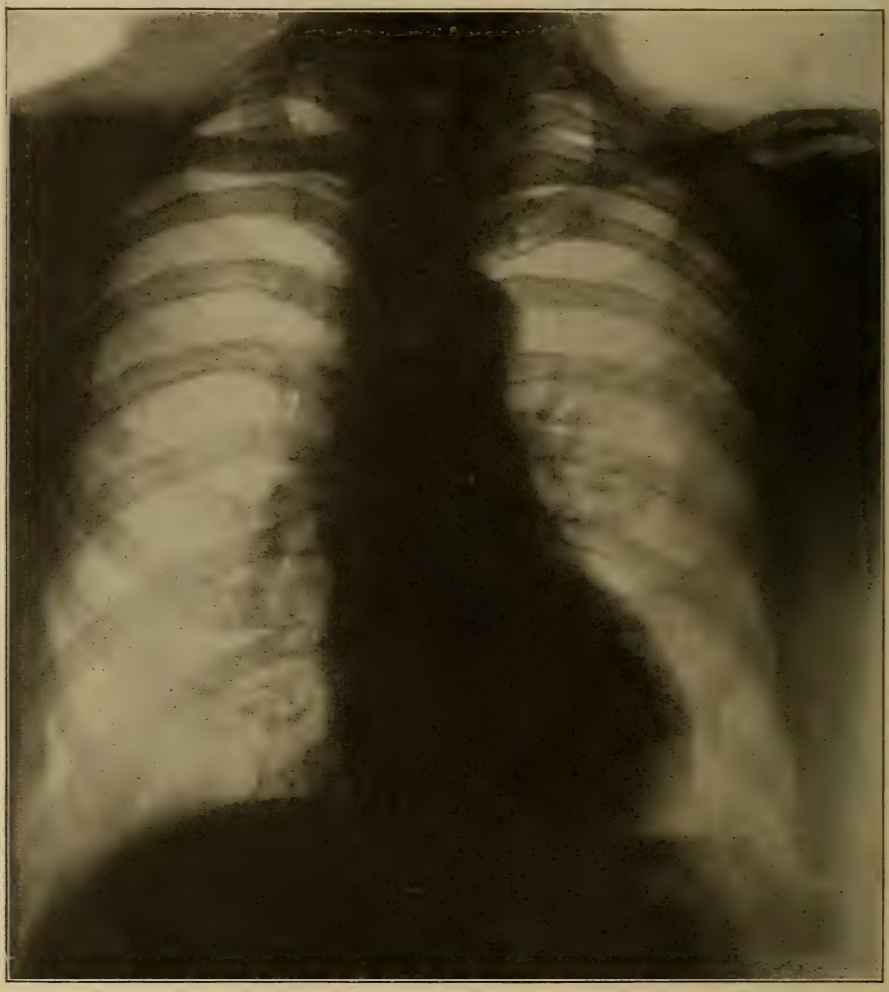


FIG. 335.—Interstitial nephritis with progressive increase of arterial hypertension Effect upon a "drop" heart. Patient under observation twenty years. Change in outline associated with periods of transient dilatation and increased arterial tension five years ago. At present systolic pressure varies from 185 to 200+ and incompensation is manifest in slight diurnal leg edema, slight dyspnea on exertion and persistent subjective fatigue. Heart only 13.8 cm. in total transverse diameter. (Death from acute dilatation and pulmonary edema since legend was written.)

of medicine offers a broader, better, or more fruitful field than that of cardiovascular disease.

A suggestive parallel is to be found between the present neglect of the early evidences of decompensation in the cardiopath and the like failure to recognize

and direct or treat the victim of incipient and early phases of tuberculosis but a few decades ago. Results may be obtained in heart disease through early recognition, wise supervision and direction in minor grades of insufficiency, which will compare most favorably in respect to bestowal of added comfort and

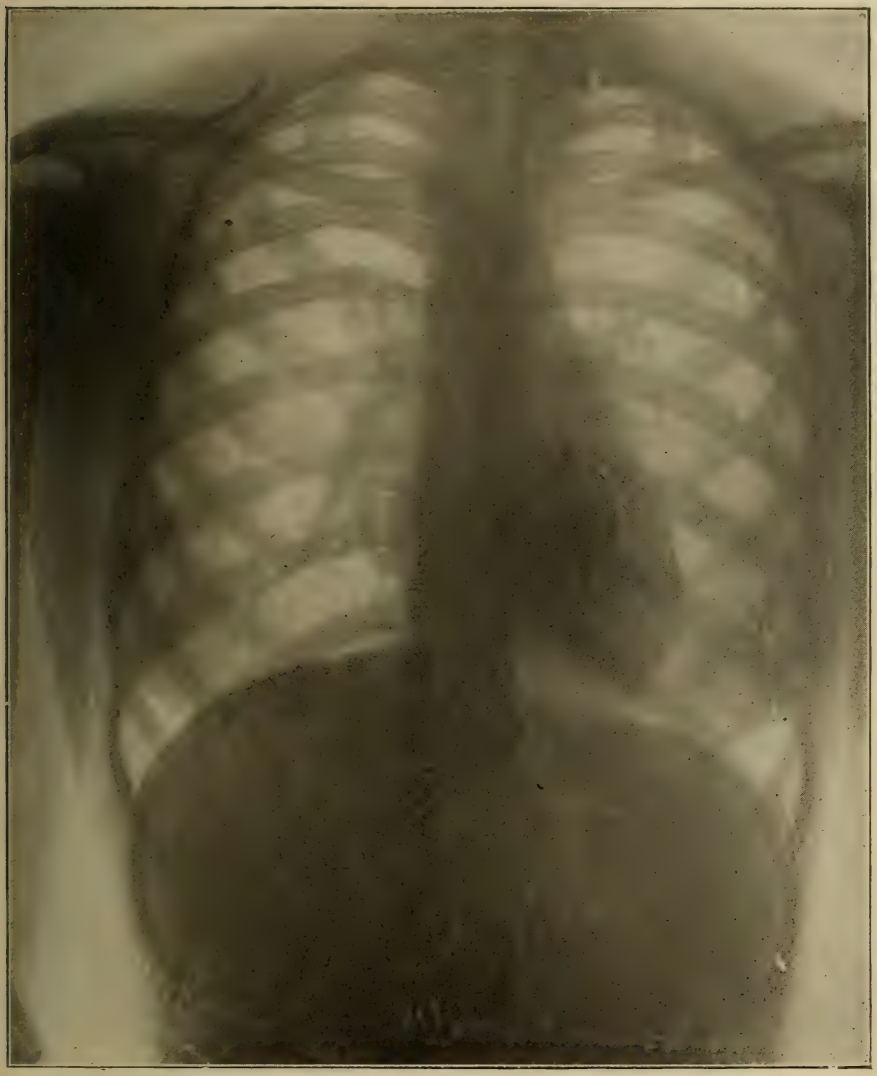


FIG. 336.—Aortic regurgitation and mitral insufficiency superimposed upon a "drop" heart. After several years the heart has attained a transverse diameter of 11.5 cm. Ml. 8.5; Mr., 3 cm; total, 11.5 cm. The "hanging" quality is shown even better in the original negative. The patient's reserve was distinctly impaired.

increase of longevity, with those now obtained in tuberculosis. One cannot but surmise that, if the cardiopath were infective in proportion to the presence or increase of decompensatory signs, he would fare better.

Retardation and Rehabilitation the Primary Need.—As in the case of tuberculosis, so also in heart disease, the effectiveness of its management depends upon the earliest possible recognition of the lesion itself and of its decompensations. Such management involves intelligent oversight, sane counsel and, when any insufficiency is discovered, rational and timely thera-

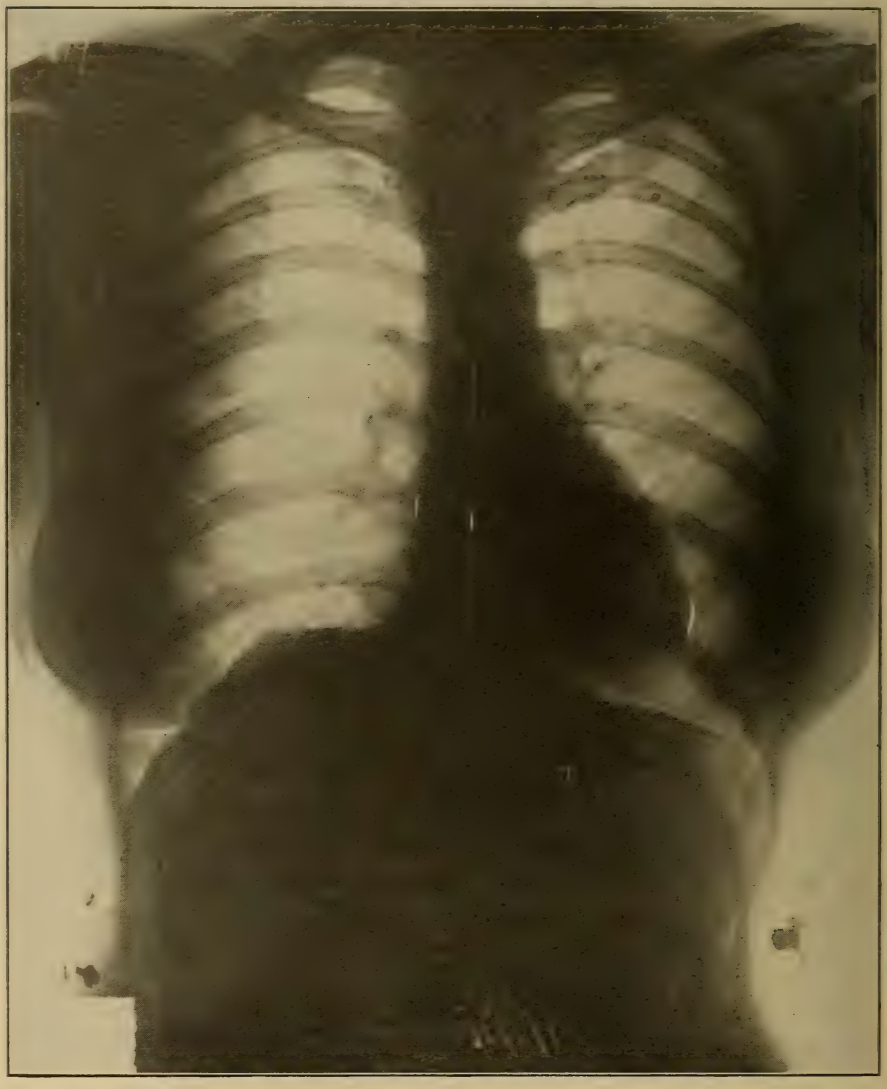


FIG. 337.—Despite obvious enlargement of the left ventricle the total transverse measurement is but 10.5 cm. (See Fig. 338).

peutic aid directed especially to the relief of those lesser decompensatory phases which are inevitable sooner or later, once the lesion is definitely established, though often almost unbelievably slow in reaching a recognizable stage, once the primary insufficiency has been compensated.

MEANS OF EARLY RECOGNITION OF FAILING RESERVE.—It is evident that the means of such prompt recognition of cardiovascular insufficiency deserves first consideration and discussion.

Direct Tests of Cardiac Sufficiency.—*We have sought long, but never yet found a direct, single, simple test of cardiac sufficiency of moderate grades.* Unreliable tests.

Even the wonder-working modern instruments of precision fail to supply

FIG. 338.—Stomach of patient whose heart is shown in Fig. 284. Note decided gastroptosis.

this need in any degree at all commensurate with the time and training necessary to their intelligent and critical use.

The muscle antagonism test of Herz the pulse rate recovery test of Mendel-*

* Flexion of the forearm at the elbow if firmly maintained is supposed to slow the pulse if the heart be weak.

sohn and Graupner or that of Katzenstein,† are alike uncertain and unreliable.*

Various other tests based upon the variations in blood pressure attending changes in posture or stated exercise, and even resort to the deter-

FIG. 330.—Case of universal dilatation of the heart. *No murmurs.* High arterial tension. Cheyne-Stokes breathing, edema of legs and engorgement of liver. Dimensions: right median, 7 cm.; left median, 12 cm.; total, 19 cm.

mination of vital capacity as a test of myocardial integrity, cannot be considered dependable.

* Time required after exercise for the resumption of the original pulse rate.

† Obliterative pressure upon the two femoral arteries should raise the cubital pressure in the case of a sound or fully compensating heart 5 to 15 and 15 to 40 mm., respectively. It is assumed that this effect is lacking in insufficiency or that the cubital pressure will fall.

Many other similar tests are alike indeterminate, but fortunately we may in most instances detect existent cardiac insufficiency by the response of any subjective symptoms present, and, in many instances, of the cardiac percussion area Valuable simple methods.

FIG. 340.—Same heart as shown in Fig. 286, showing remarkable change in outline under treatment and full recovery of compensation. Mr., 5.5 cm.; Ml., 10.5 cm.; total M., 16 cm. A reduction of 3 cm. in transverse diameter is evident, a remarkable figure for a hypertrophied and dilated heart of this type. One has to think of the circumferential reduction obtained to appreciate the full measure of change. Another patient under treatment at the same time (transverse measurement 17 cm.) and showing initially the same conditions less the Cheyne-Stokes breathing, left the hospital rehabilitated, yet showed a shrinkage of but 0.5 cm., an amount too small to be exactly determinable. The cases emphasize the fact that impaired tonus rather than mere relief of dilatation is the fundamental factor.

of the suspect, to a few full and adequate but physiologic doses of a direct cardiac stimulant with or without absolute rest, as the individual case demands.

Neither the normal heart nor the efficiently compensating hypertrophied heart should show a decided change (Fränkel and Schwartz, Cloetta). Helpful fact.

The readiness with which decided dyspnea is produced under exertion doubtless remains the most useful direct rough test of myocardial inadequacy.

Unavoidable errors.

Great Value of Skilful Percussion.—As may readily be shown by X-ray control, serious error in skillfuly applied percussion *by modern methods*, though occasionally unavoidable, and sometimes gross, is far less frequent than is generally believed if one adheres to like conditions on different occasions, outlines both borders, and substitutes the modern rectilinear orthopercussion method, with or without threshold percussion, for the heavy flat-finger technic of former days.*

In the case of acutely or subacutely dilated hearts even though preëxisting hypertrophy is present, the shrinkage of outline is usually marked and the lessened area is often so well maintained under and following the therapeutic test as to be most illuminating, but the most important and constant factor is the rapid amelioration or disappearance of troublesome symptoms oftentimes primarily obscure and remote.

The most dramatic relief of symptoms may result in cases showing a roentgenographic or roentgenoscopic change of cardiac outline so slight as to be useless for purposes of routine clinical comparison and this is especially true of many symptom-producing "drop" hearts and of old lesions with well-established enlargement of the ventricular cavity and hypertrophy of its walls.

This statement applies both to those cases presenting subjective symptoms only, and to such as carry objective signs of the grossest and most obvious type.

One has but to recall the ratio of circumference to the diameter to realize that a very definite and decided shrinkage may fail to diminish the cardiac profile by even one centimeter.

It is evident also that a symptom-producing diminution of tonus may be unaccompanied by a clearly demonstrable extension of the cardiac silhouette.

Know and demand the normal.

Auscultation.—With respect to auscultation our great need is of *an accurate knowledge of normal sounds and the range of permissible variations*, together with a concentration of our attention *primarily* upon the question of their presence or absence in the given case.† One may suggest the need of more careful attention to weak and impure sounds and abnormalities of accent, and

* Treupel found in 97 per cent. of the cases examined by the method of threshold percussion a variation of less than 1 cm. in the position of the *right border* as compared with the orthodiagraphic outline.

In 95 per cent. the left border was established with the same accuracy. Such remarkable figures are not applicable of course to the ordinary routine of a busy practitioner, inasmuch as they represent doubtless examinations made under ideal conditions of time and place.

The author finds accurate determination of both the left and right borders sometimes impossible, *for him*, by any method, when working under ordinary office conditions which embody noise and interruptions. In general he finds that an error by one careful man is shared by those who follow him. Roentgenography and roentgenoscopy long since were adopted therefore as an unvarying part of the routine of his consulting-room work.

† In the section dealing with the examination of the heart the author has referred to the common mistake of seeking primarily the abnormal, rather than the proving of the presence of the normal. The difference is actually vital.

a less exclusive concentration on actual murmurs which are the most obvious of the phenomena of cardiac disease.

Cardinal Factors in Timely Diagnosis.—Five decided steps in advance make this possible, viz.:

(1) *A fuller realization of the importance of the anamnesis and of the cardinal value of the sensations of the patient together with a knowledge of the number and diversity of subjective symptoms of cardiac origin.* For this we must thank chiefly James Mackenzie and Henry Head.

2. *A more accurate standard of "normal" heart measurements.*

3. *A proper valuation of alterations in subjective and objective symptoms and, oftentimes, in the heart outline under tentative therapeutic measures.*

4. *The knowledge that both the wholly normal and the fully compensating though crippled heart fail to show material response to doses of digitalis,* which are clinically effective in decompensation.*

5. *The substantial increase in accuracy afforded by modern percussion methods and the use of the x-ray.*

6. *The use of instruments of precision yielding graphic records of the heart action.*

It is to be hoped that before long one may be able to add—

The abandonment of the flat-finger method in heart percussion.

Proper Valuation of Subjective Symptoms.—The early recognition of cardiac incompensation demands a better understanding and truer valuation of the subjective manifestations of cardiovascular disease, until recently submerged and concealed by our ignorance of their genesis, nature and peculiarities of localization and by a natural but most unfortunate tendency to attribute them nearly always to sources other than the heart.

Why formerly unrecognized.

THE RECOGNITION OF SUBJECTIVE SYMPTOMS.—*The subjective symptoms of heart disease are many, varied, and* **invaluable,** *though few are peculiar to cardiac insufficiency and none are pathognomonic.*

Fundamental factors.

The fundamental factors in interpretation are:

1. *The relation of their occurrence to concurrent or precedent physical exertion, especially if this be of an unusual nature.*

2. *Their occasional association with the lesser demonstrable dilatations.*

3. *Especially in the case of the middle-aged patients and to a less degree in younger persons, their relation to excitement or emotional strain.*

4. *But chiefly their favorable response, and oftentimes that of the cardiac outline, when increased, to cardiac stimulation, with or without physical rest or regulated exercise, as may be found necessary.*

Subjective Dyspnea.—A subjective sensation of dyspnea, diurnal or nocturnal, even of the milder types, is a striking symptom of value, too readily ascribed to hysteria or "neurasthenia." Even a persistently increased respiratory frequency or an inability to hold the breath is frequently over-

Very important.

* Fränkel: Ueber Digitaliswirkung auf gesunden Menschen, Münchener med. Wochenschr., lii, p. 1537, 1905.

Cloetta: Einfluss der chronischen Digitalis behandlung auf das normale and pathologische Herz, Therapic der Gegenwart, vol. xlix, p. 437, 1908.

looked in the absence of any complaint of dyspnea or too hastily credited to the same diagnostic "catch all."*

Another cardinal early symptom of failing reserve is subjective dyspnea initiated by relatively slight, but *unusual* effort.

Purely Subjective Symptoms.—A sense of lassitude, of easily induced physical fatigue, vertigo, unexplained drowsiness during the day time and unrelated to heavy meals,† or insomnia, wakefulness, lack of concentration and sustained application, mental confusion, faintness, actual syncope, heaviness of the legs or the sensation of wading through water, together with disturbed sleep, bad dreams and subjective gastric disturbances of the most varied kinds, are extremely common, helpful and suggestive, though by no means distinctive, symptoms.

Of corroborative value.

One of the most important of all symptoms is an inability to perform without conscious added effort and fatigue everyday tasks formerly easily dispatched.

This represents in most instances the first evidence of a narrowing of the cardiovascular reserve and may be associated with any one or several of the other symptoms mentioned.

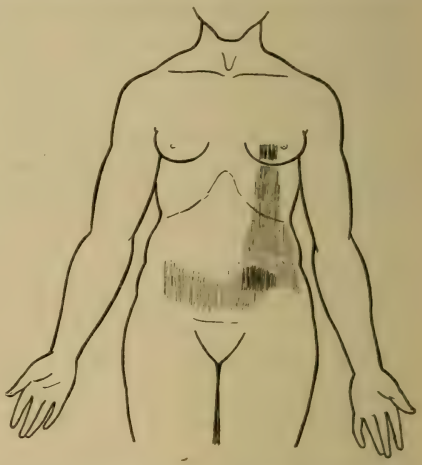


FIG. 341.—Case of mitral leakage and moderate dilatation. Pain during preceding year. Illustrates curious distribution of pain and tenderness, occasionally observed in cardiac insufficiency. Dark areas indicate maximal points of chronically recurring pain; shaded area—its distribution. Patient was to undergo exploratory operation. Entirely free after four days of partial rest and full doses of digipuratum. Has remained free for past two years. (Another case showing this pain reference has been observed since this legend was originally written.)

In cases of insufficiency associated with high arterial tension, numbness and prickling of the lower extremities frequently occur, often being manifested only under a coincident increase of cardiac dilatation and increased arterial pressure and subsiding as these are relieved.

Hypertension.

Pain or Discomfort Due to Muscle Fatigue and Overstrain.—*Any muscle in the body may give rise to manifestations varying from mere subjective discomfort to agonizing cramp-like pain when forced to continue maximal effort under conditions of excessive strain and fatigue.*

Muscle cramp.

We have the report of forced marches, the records of various long-distance contests, as well as the histories of acute heart strain in patients to

* In a number of instances observed by the author the recurrent or paroxysmal dyspnea attending excitement or emotional strain has had all the earmarks of the hysterical form, which doubtless can occur, yet was found to be associated with cardiac insufficiency and arterial hypertension. In such cases not only the emotions, but even slight gastric or intestinal overdistention, or mere corset pressure, may precipitate an attack.

† A very common symptom in the author's experience and one of the most responsive to the therapeutic test if due to circulatory insufficiency.

attest this fact and to explain one of the chief manifestations of chronic cardiac overstrain.

Multiple Factors.—In most heart cases many factors other than actual overexertion diminish the tonus and contractile power of a diseased heart, and in varying degree the other functions of the heart muscle, until accustomed use, and later mere existence, involves serious overstrain.

Intrinsic blood supply.

Sclerosis of the coronary arteries not only may produce a persistent deficit in the intrinsic blood supply of the heart and render its response to increased demand distressing or actually impossible, but may also cause attacks of angina pectoris under conditions of physical rest (see "Angina Pectoris").

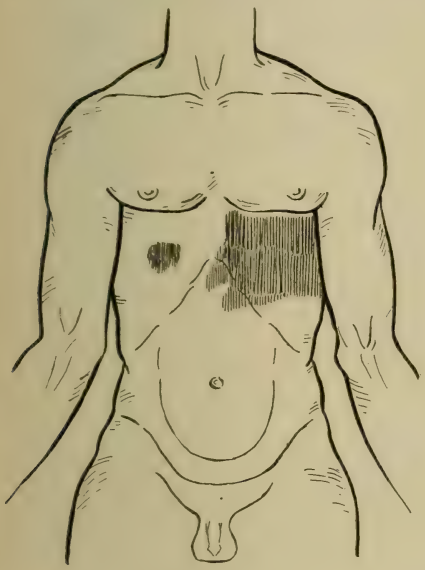


FIG. 342.

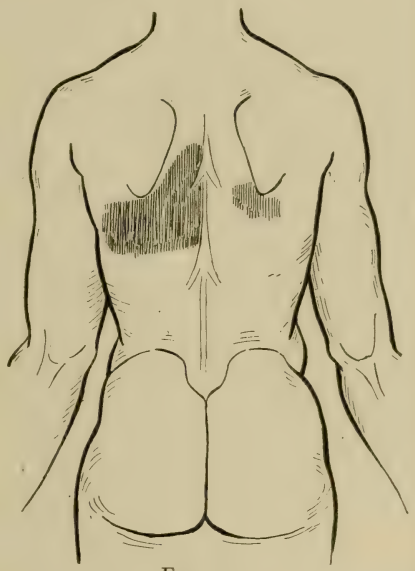


FIG. 343.

FIGS. 342 AND 343.—Interesting areas of pain and residual tenderness associated with certain cases of cardiac decompensation, induced by over-exertion or acute infections. Dull pain or discomfort and more or less marked hyperesthesia over the region of the left heart border are extremely common. Occasionally it is bilateral and in such a case is almost invariably referred to hysteria or "neurasthenia."

Minor Crises.—From time to time, long before the terminal period is reached, the struggling heart cries out for relief. Our clinical interest must, therefore, be directed primarily to the lesser painful or distressing expressions of cardiac fatigue and overstrain and the regions to which these may be referred.

Extracardial Factors in Diagnosis.—*Both subjective and objective symptoms of chronic myocardial insufficiency arise in considerable measure, in both the minor and the grosser insufficiencies, from districts lying outside the precordial area and the heart itself, and this is strikingly true of certain types of pain.*

Deceptive localization.

Referred Pain and Discomfort.—The work of Henry Head, Sherrington, Mackenzie and others shows that despite the relative insensitiveness of the heart parenchyma itself, painful sensations, with or without hyperesthesia,

Referred pains.

arise from cardiac embarrassment, follow the direct embryonic distribution of the primitive cardiac tube and, because of the distortion attending fetal development, may be referred to the chest wall, axilla, neck, shoulders, inner aspect of arm, epigastrium and even the right and left hypochondrium.*

Many types.

With such a wide area of distribution for cardiac pain and granting the logical assumption that various sensations of discomfort may substitute pain in cardiac overstrain, toxemia and fatigue, as in like conditions affecting any other muscle, we see the possible relationships not alone of precordial distress, but of the many axillary, neck, shoulder, thoracic and upper abdominal pains or uncomfortable sensations, such as are usually, *and rightly* given the more obvious interpretation suggested by their surface relation to underlying tissues.

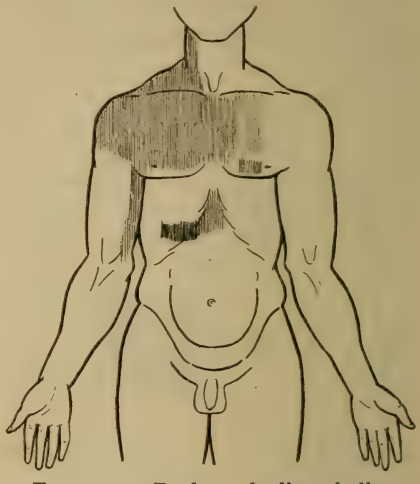


FIG. 344.—Darker shading indicates pain maxima. Lighter shading, its distribution. Recurrent acute major angina. Widely dilated heart. History of acute overstrain (electric-car accident). Operation for gall-stones advised. Prompt relief from rest and digitalis. No recurrence until return of dilatation and signs of major insufficiency several months later.

Often epigastric

Epigastric, Substernal and Precordial Discomfort.—This usually takes the form of precordial or epigastric discomfort and, frequently, both combined; a sense of weight or pressure; a subjective sense of "*crowding*" distention, substernal gripping or constriction, usually mild, but sometimes described as "vise-like," even when unassociated with true angina pectoris.†

These sensations are referred to the stomach more frequently than to the precordium and by the physician are interpreted usually as of gastric origin,‡ or, not infrequently, in nervous women, as hysteria ("globus hystericus," etc.).

The irritable overstrained or degenerated heart is affected greatly by an overdistended stomach or an overloaded portal system.

An Old Rule Reversed.—So frequent is the occurrence of gastric symptoms even in the minor decompensations that it would seem that in the case

* The auricles may refer their pain to the lower axilla and shoulders; the ventricles to the chest wall, epigastrium, inner aspect of the upper arm, ulnar surface of the forearm and wrist or to the little and ring fingers. Pain from the ascending aorta may be referred to the entire neck, including the occiput. Cases of major angina, to which all portions may contribute, are reported in which even the gums were painful and others produce pain and residual hyperesthesia over the entire thorax, neck and upper abdomen.

† Many patients report voluntarily, after successful treatment, even though no marked shrinkage of the cardiac outline is demonstrable, that for the first time in many months or years they feel that their heart is not "crowded" or "too large for the chest."

‡ The number of so-called "dyspepsias" relieved promptly by digitalis, with or without rest, is surprising.

of the elderly patient, especially, we should reverse the traditional rule, and instead of considering first the stomach when he complains of his heart, look rather to the arteries, blood pressure and kidneys primarily and the stomach secondarily when he complains of either the heart or stomach.

"Indigestion" the common complaint.

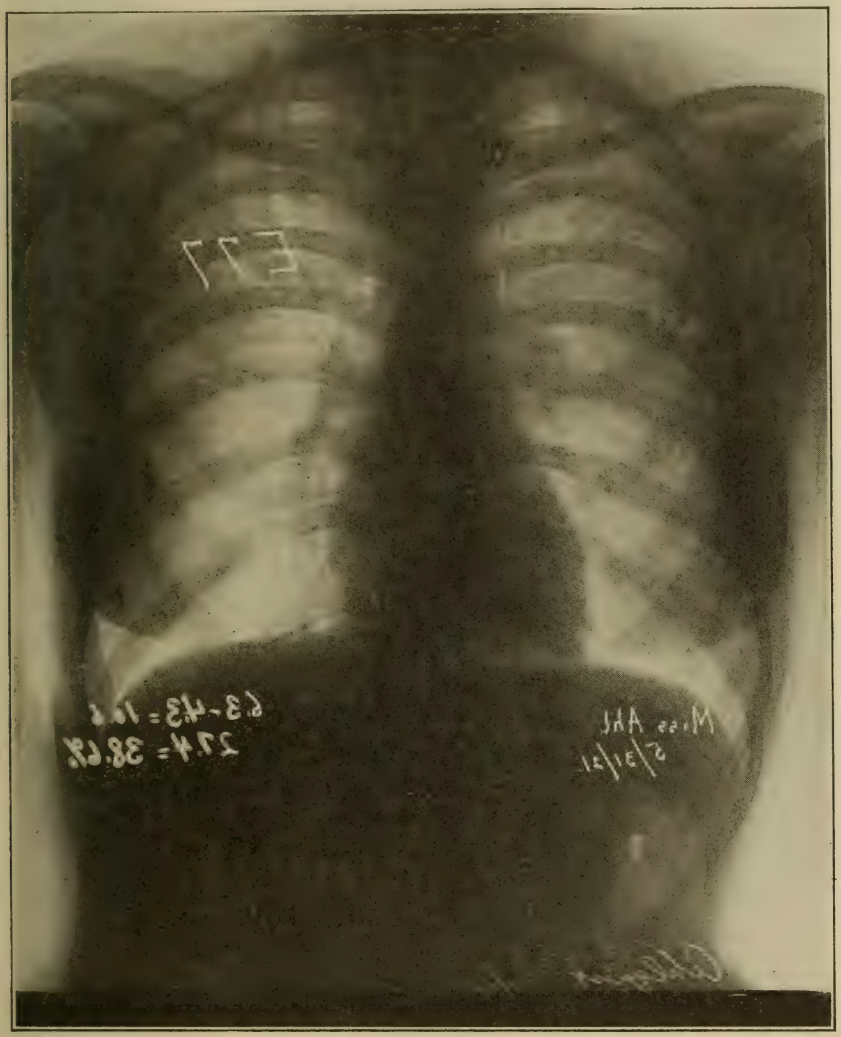


FIG. 345.—(E 77). Decompensated and dilated heart of the "drop" type associated with dyspeptic symptoms which dominate the clinical picture. The most pronounced dyspeptic symptoms appear during the latter part of the day or following, either directly or after a short period, unusual physical exertion. Vertigo and fatigability were also present. Exertion dyspnea was slight and there was no edema of the extremities. This is of the type commonly miscalled the "small" heart. (See Fig. 346.—(E 77′). for comparison.)

Simulation of Urgent Abdominal Lesions.—Occasionally a patient with chronic myocarditis or coronary sclerosis and a *dilated laboring heart* is confidently but vainly explored for gall-stones or gastric ulcer under a not

Simulation of surgical lesions.

unnatural misinterpretation of referred cardiac pain and hyperesthesia. Such errors may be avoided usually if the radiation of the pain with reference to the precordium, upper thorax, neck and arms is studied. Though such pain may be maximal over the upper abdomen, it usually

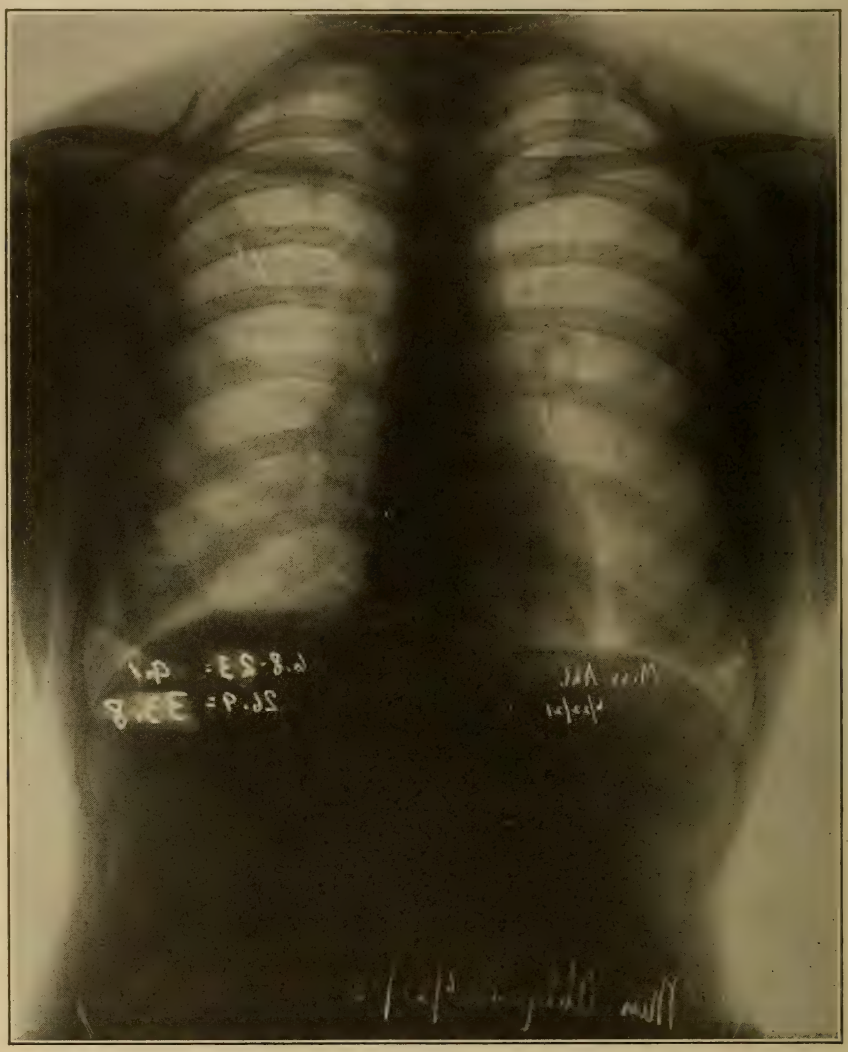


FIG. 346.—(E 77′). Same heart as shown in Fig. 345 showing the reduced diameter following a short period of ambulatory treatment. The heart was originally a "drop" heart, marked associated gastroptosis being present. The outer white line shows the passing out of the left border by renewed dilatations after the patient had broken treatment and disregarded all instructions, the result being the recurrence of pulmonary congestion and other signs of minor decompensation.

shows a tendency to involve the other regions in part at least. Careful examination of the heart, and blood-pressure estimations, usually reveal an adequate cause.

A Word of Warning.—*It must be remembered that gastric, duodenal,*

FIG. 347.—Toxic heart. Patient had suffered from gall-stones 10 years previously and temporary relief had been afforded by the passage of a large number of small biliary calculi. Previous to, and from that time, recurrent severe precordial and epigastric pain and increasing disability had been present, all being charged against the gall bladder, though no operation was attempted. When seen by the author in 1914 she carried decided edema of the legs, some hepatic engorgement, and marked enlargement of the heart, with auricular fibrillation. A history of recurrent typical attacks of cardiac angina combined with epigastric distress was obtained and the nature of some of the abdominal pain distinctly *suggested* the participation of the diseased gall-bladder, although no local tenderness or fever was present. The heart measured 15.5 cm. in total transverse diameter and decided diffuse enlargement of the transverse and descending portion of the aortic arch was evident in the original negative. The patient was placed at rest and under digitalis with the result that all symptoms were relieved promptly and the heart rhythm became extrasystolic. The outline of the heart at the end of this period is shown in Fig. 348. Aside from a slight inconstant and untransmitted mitral bruit or "murmurishness" *the heart was silent.*

She had been told repeatedly that her heart was wholly normal.

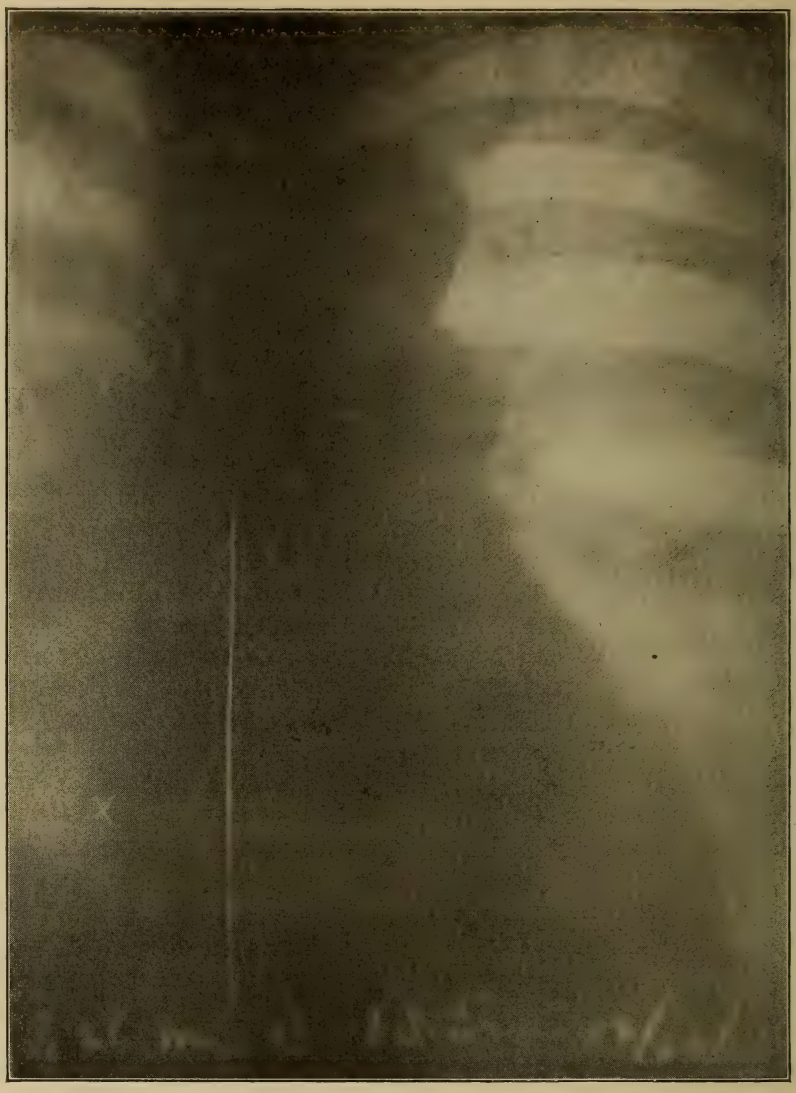


FIG. 348.—Toxic heart after treatment. The total transverse measurement is reduced from 15.5 cm. to 13.5 cm., a normal area, *apparently*, for so large a woman (weight 185+) but actually a greatly enlarged heart primarily of the "drop" or modified "drop" type, in a very small-boned individual. It will be noted that with the subsidence of the fibrillation the right border is lost behind the sternum and the left has lost its mitral configuration above, though in this printed reproduction the prominence of the left *hilus* shadow obscures the true outline obtained. Despite all warnings the patient so imposed upon the immunity from suffering obtained as to create from time to time through rash overactivity a recurrence of fibrillation and intervals of diurnal edema of the extremities. Finally, while on a visit certain purely abdominal seizures arose which were apparently due to gall-stones alone. Upon her return it was evident that definite fibrillation and other decompensatory signs were present. The patient was put to bed, but after a brief period of improvement a definite attack of cholecystitis occurred. The evident hazard involved in an operation upon one with so slight a margin of reserve led the surgeons to decline interference, but after 48 hours, at the patient's urgent request and by reason of the threatening nature of the symptoms, an operation was attempted and expeditiously performed. The patient never reacted. The heart dilated acutely and she died 12 hours after removal from the table. *It will be noted that the existence of a highly toxic, greatly enlarged silent heart had been wholly unsuspected up to* 1914. Yet so far had the process advanced as to render periods of fibrillation frequent and maintain a decided edema for many years.

appendiceal, and cholecystic conditions may be coexistent with and sometimes the basic cause of a toxic heart (see Figs. 342, 343 and 344).

Moderate or trivial pain of a slight, vague character or in the form of sharp "*stitches*" may occur over the heart itself, often extending upward to the left or behind the sternum.

Tenderness.—Not infrequently one encounters tenderness just within the apex and for a few centimeters up the left border of the heart. This curious hyperesthesia is often mistaken for the hysterical breast stigma when occurring in nervous women, but is common in the chronic tonus defects, moderate dilatation and chronic overstrain of myocardial degeneration or mere asthenia, as well as in acute toxic dilatation and mitral stenosis.* Commonly misinterpreted.

It sometimes shifts its maximal point with the recession of the left border as a toxic widely dilated heart contracts.

Major Anginal Pain.—Intense pain of the severe paroxysmal type may occur, associated with fear of impending death, pallor and cyanosis, cold perspiration, orthopnea and tense immobility on the part of the patient, shallow breathing, weak, rapid or unduly slow pulse and high arterial tension. Pain may extend to the left arm, wrist, and fingers up into the neck, or involve the arm and neck of both sides. In rare instances such *paroxysmal* pain of cardiovascular origin may be maximal over the epigastrium and gall-bladder area and be preceded or followed by nausea or vomiting.†

Miniature replicas of such attacks have been observed and reported by the author, oftener fragmentary clinical pictures, and a marked and widespread hyperesthesia may follow the severe attacks. Replicas in miniature.

Paroxysmal Pain of the Sciatic Type.—Sciatic pain, lameness, quickly induced leg-fatigue or sudden temporary loss of power, in the lower extremities or even the arm, may attend attempts to put the affected extremities to accustomed use ("*intermittierende hinken*"), and in many such cases the pulse of the affected member will be weak or absent, the accessible arteries sclerotic, and the heart degenerated. Arterial spasm.

When the sciatic pain is marked, an erroneous primary diagnosis is common.

OBJECTIVE SYMPTOMS OF DECIDED CARDIOVASCULAR INSUFFICIENCY.—Subjective dyspnea has been described elsewhere. Objective dyspnea may be present only on exertion or in one position of the body, or be constant, paroxysmal, of the type of "air hunger," Cheyne-Stokes, or a replica of spasmodic asthma. Occasionally it is no more than an evident *inability to hold the breath* but in some cases it will abruptly enforce the sitting posture or even force the patient to stand suddenly erect (see "Dyspnea").

* "Stitch in the side" is, of course, common and unimportant in the absence of other signs, but occurring in the precordium, and especially in middle-aged individuals, often represents a minor angina.

† The author has seen a number of such cases in one of which a very large aneurysm of the transverse portion of the arch was present and ruptured during the early period of general anesthesia incident to an attempted operation for gall-stones.

Stasis and edema.

The Lungs.—Recurrent acute or subacute bronchitis, chronic bronchitis, blood-stained sputum, with the pigmented ("heart") cells, actual hemoptysis, or pulmonary edema, may occur as the result of passive congestion of the lungs or pulmonary infarct, in the failing heart of certain valvular lesions or primary myocardial degenerations.

Crepitations *at the lung bases, persisting after the first deep inspiration* indicate right heart weakness and become of importance in the diagnosis of myocardial insufficiency and its treatment. They may be more marked on one side or present only as *persistent* râles. In such instances one finds usually that the patient lies upon that side by preference or has so lain for a considerable period.

Portal congestion.

Gastrointestinal Tract.—Misleading referred cardiac pain or discomfort and gastric disturbances more or less directly attributable to passive congestion occur frequently together with constipation, diarrhea, anorexia, excessive flatulence or nausea and vomiting.

Usually all these symptoms are significantly relieved or ameliorated by rest and cardiac stimulation if they are of cardiac origin.

Engorgement.

The Liver.—The liver is frequently enlarged in the myocardial degenerations attended by gross decompensation, especially when these are associated with valvular lesions involving right heart stasis (mitral or tricuspid lesions especially).

Variations.

Its variations in size may roughly reflect the degree of decided incompensation present at a given time. Systolic venous pulsation, palpable or shown by instrumental registration is not uncommon and the liver may become

Pulsation.

permanently indurated ("*nutmeg liver*"), though in many instances that which may have seemed a permanent change wholly or almost wholly disappears under proper, prolonged treatment.

Ominous if persistent.

Jaundice of slight or even marked degree is not uncommonly seen in advanced tricuspid lesions, whether primary or secondary, in association with long-established passive hepatic congestion.

Important sign.

Kidneys.—*Chronic passive congestion is one* of the manifestations of advanced incompensation in primary myocardial degenerations and mitral or tricuspid lesions.

Varying output.

The amount of urine is more or less reduced and diurnal variations in the amount **roughly** *measure the variations of severe incompensation if the fluid intake is maintained at a uniform level and no sweating or purging occurs.*

Laboratory findings.

The color and specific gravity are high. Albumin is present in small amount and hyaline with occasional granular casts may be found.

Obscures existing nephritis.

If other forms of nephritis co-exist, their typical urinary signs will be obscured, intensified, or modified by any existing passive renal congestion and uncovered or rendered clearer by an improvement in the heart strength.

EDEMA.—*This, when pronounced, is the most strikingly objective of the commoner gross symptoms and the most marked of any save pronounced dyspnea or the profound cyanosis of right heart stasis.*

In its slighter and localized forms it is very likely to be overlooked, espe-

cially if it occurs only in unusual locations. In a number of cases the author has found it present over the calf of the leg and absent at the ankle.

Usually seeks dependent parts.

Slight, equal, bilateral edema is almost invariably overlooked in life insurance examinations and, very generally, in the ordinary office or hospital procedure.

The patient carrying it is often unaware of its presence or thinks so lightly of it as to fail to report it.

It may exist for years before the graver symptoms of decompensation manifest themselves and is a symptom of cardinal importance.

Cardiac edema appears as the right heart fails,* seeks primarily the dependent portions of the body, is markedly affected by decubitus, and, in advanced cases with primary or secondary right heart weakness, is pronounced, slowly progressive in the absence of effective treatment, and tends to superadd to the already obstructed blood flow and the laboring heart behind it (see "Edema"), the adverse pressure effects of effusion into the peritoneal, pleural, or even the pericardial cavities.

Embarrassing transudates.

Rationale of Cardiac Edema.—Under conditions of rapidly induced or persistent venous congestion and consequent increased pressure and slowing of the blood stream in the capillaries, a great excess of fluid, poor in albumin, is poured out and distends the connective-tissue spaces. The general circulation is thus further slowed, the distension and loss of tonus of the connective-tissue spaces invites further invasion, and the pressure of collected fluid tends further to block the flow of blood and establish a vicious circle.

Another vicious circle.

The loss of tonus in the connective tissue is sufficiently proven by the persistent indentations left by finger-pressure.

Pitting

Weigh Cardiac and Renal Cases.—In both renal and cardiac cases showing a tendency to edema it is of great importance to weigh the patient from time to time. Any sudden increase in weight should suggest the possibility of an exudate either into the connective tissue or the enclosed cavities of the body and the pleuræ should be watched with especial care.

Weight often varies with edema.

Cyanosis.—*Any and every type of cyanosis may be encountered in cardiac insufficiency according to the anatomic seat and chief effect of the dominant lesion, the degree of involvement and the grade of incompensation present.*

All cardiac incompensations or pulmonary diseases weakening the right heart, directly or indirectly, and hindering or preventing free oxygen and CO_2 exchanges, produce cyanosis.

Diminished oxygen and CO_2 exchanges.

In most forms of congenital heart disease it is so obtrusive and extreme and so evidently related to infancy in its inception as to be an almost pathognomonic sign.

In the least rare type of congenital insufficiency (*pulmonary stenosis or atresia*) the patient is almost invariably blue and, on exertion or in paroxysmal seizures, deep purple or even black.

Blue babies.

* Edema may be absent throughout the entire course of an aortic stenosis or even a regurgitant lesion, for the patient may and often does die before the *right heart* is rendered markedly insufficient, the burden in either instance being carried wholly or in large measure by the left ventricle until the terminal stages are reached.

Absent in pure aortic cases.

In aortic stenosis or regurgitation it may never be present to any marked degree until the mitral yields or becomes involved structurally in the disease process, and throws back its burden in part upon the pulmonary circuit and the right heart.

Relative insufficiencies.

Cyanosis is decided in incompensated mitral lesions and still more so if secondary tricuspid insufficiency or the far rarer pulmonary leakage ensues.

Heart Sounds.—Absolutely normal heart sounds are always absent in primary or secondary valvular disease and murmurs are usually present or more or less readily elicited under exertion, change of posture, properly regulated stethoscopic pressure, rest, or direct cardiac stimulation.

In myocarditis, acute or chronic, or in the myocardial degenerations of marked degree, there is a tendency for murmurs to develop, especially in the mitral area, as the disease advances, the valvular deficiency being due either to an extension of the disease to the valve itself or to weakening of the valvular ring.

Abnormalities of accent, sharpness, undue brevity, and heightened pitch of heart sounds, no less than impurity, "murmurishness," or "muffled" tones, are also to be noted.

Arrhythmias.—Any and every variety of abnormal rhythm may be encountered (see "Arrhythmias").

CARDIAC OUTLINE.—*As previously stated, the cardiac outline as determined by the more modern percussion methods with or without the reënforcement of the fluoroscope is one of the chief means of detecting otherwise dubious or indefinite lesions.*

Almost invariably an enlarged profile due to coincident increase of dilatation is present in the case of a grossly incompensating heart and this yields oftentimes a significant recession in response to full doses of digitalis and rest, both of which measures are without effect upon a normal heart or one fully compensating any existing lesion.

Therapeutic test invaluable.

The cardinal sign is, however, a decided and demonstrable amelioration or relief of symptoms under such therapeutic procedures.

Summary.—(*a*) *The severity or degree of all symptoms of cardiac insufficiency depends upon the nature and location of the lesion and the stage and duration of the incompensatory period.*

Factors determining severity of symptoms.

(*b*) *The frequency of damagingly reduced tonus, lacking the coarser and more obtrusive incompensatory signs, is as striking as is the relief of its vague and often misleading symptoms under a brief period of treatment and proper instruction.*

(*c*) *Such hearts occur with especial frequency in patients who have entered* the *fifth or sixth decennium and in younger patients as well, if they are undernourished and carry the stigmata of chronic congenital asthenia and its movable, pendant, low-lying, unstable and, oftentimes, readily dilatable heart.*

Age.

Congenital asthenia.

Truly Effective Therapy.—The larger, most effective and most permanent part of an effective therapy lies in the regulation of occupation, habits, physical and mental exertion, diet, *and above all an intelligent appreciation*

Avoid mere meddling.

of the fact that neither cardiac murmurs nor cardiac arrhythmia, in and of themselves, demand active treatment.

Neither Drugs nor Rest, Whether Alone or Combined, are Adequate.—One of the most important factors in the therapy of cardiac disorders consists in the use of *carefully and definitely graduated* and conservatively increased exercise, *following* the correction of any acute or chronic insufficiency requiring primary limitation of activity or an insistence upon a sufficiently *prolonged period* of absolute rest.

Value of regulated exercise.

Reiteration.—The student should carefully consider the following points in review:

1. *An early diagnosis of myocardial insufficiency is essential to the best welfare and maximal longevity of the cardiopath.*

2. *Such early diagnosis necessitates an acquaintance with symptomatic expressions of the* **subjective type,** *and the determination of the presence and symptomatic relationships of any existing cardiac dilatation.*

3. A large group of structurally deficient individuals many of whom are commonly classed as "neurasthenics," while, possibly, comparatively free from organic heart disease of the valvular or luetic type or that taking the form of primary myocardial or vascular degenerations, are peculiarly lacking in resistance and prone to undernutrition, to acute infections, and recurrent or persisting deficiencies in heart muscle tonus, and possess ptotic, readily dilatable, symptom-producing hearts as a part of their fundamental and usually congenital defects in general body structure and function.*

The heart of congenital asthenia.

The author's more recent experience has convinced him that such hearts are in no sense immune to infection whether of the juvenile degenerative or luetic type, but that in the past we have failed to recognize the "drop" heart when transformed by valvular or primary degenerative disease. If, as the author hopes, he has found the key in associated gastroptosis and more or less definite peculiarities of outline, much more light upon this subject may be anticipated.

4. Such hearts, in individuals of this type, are narrow when undilated, mobile, and even when pathologically enlarged show a *transverse* measurement which is usually less than that of the undilated heart of the non-asthenic individual.

5. In many patients of this type a relative cardiac insufficiency may play a large part in their disability and be accountable for many symptoms, usually referred to such syndromes as "neurasthenia" and "nervous dyspepsia," which, for the greater part, represent, in all probability, mere symptom-groupings of various maxima of localization, of the same general, congenital, constitutional and structural defect (see "Congenital Asthenia").

Periods of minor insufficiency.

* In opposition to the views of Prof. Stiller relative to the asserted immunity of this type of individual to organic heart disease, the author believes that the females of the asthenic group furnish the greater number at least of those cases of the pure funnel type, mitral stenosis, of the youthful non-rheumatic and non-infectious form described by Duroziez as a special form of that ailment. Every example of these peculiarly long-enduring cases seen by the author during the last decade have shown distinctively the stigma of congenital asthenia.

6. **Adaptation vs. "Perfect Compensation."**—Though patients with organic heart lesions may go for years without serious symptoms after adaptive hypertrophy is established, the pathologic events in such cases make it evident that there is a constant, more or less gradual but progressive limitation of the field of cardiac response. Long before the onset of gross or emergent symptoms, periods of more or less decided minor insufficiency appear and recur from time to time with increasing frequency. Appropriate therapeutic measures will then support and aid the embarrassed and laboring heart and prolong the life of its possessor.

7. **Causative Agents and Portals of Infection.**—The recent studies of the causative agents and portals of infection in acute rheumatism, a better knowledge of the effects of prostrating acute infection, and chronic or recurrent cryptogenetic sepsis upon the heart, the better understanding of the nature and means of detection of syphilitic infection and the introduction of new agencies and better methods for the early and efficient treatment of lues, indicate the possibility of limiting considerably the large group of myocardial, pericardial and valvular lesions of which they are the causative factors.

8. *The mere existence of a heart lesion is no justification for active treatment by the exhibition of drugs, though a fully compensating heart neither is damaged nor influenced by test doses of digitalis properly administered and controlled.*

Active treatment of any considerable duration is necessary and justifiable only when symptoms of cardio vascular *insufficiency* are clearly established and the use of heart stimulants should be limited if possible, (*a*) to test doses, which should be freely used,* (*b*) prophylactic small doses during convalescence from acute prostrating ailments, (*c*) to the more or less prolonged periods or recurrent intervals of actual stress or genuine emergency.

Responsive to treatment.

9. *The importance of early diagnosis is emphasized by the fact that chronic heart disease, though incurable, is on the whole, wonderfully responsive to intelligent and properly timed treatment, and always benefited by such proper supervision and control as may be indicated in the individual case and obtainable only through early diagnosis, tactful disclosure and a judiciously tempered optimism.*

Lost opportunities.

The "Abandoned" Heart Case.—An unfortunate tendency exists to the practical or actual abandonment of advanced or apparently hopeless cases of heart disease.

Practical abandonment may take the form of a failure to establish such control of the patient's activities and such therapy as will, on the one hand, give the greatest possible amount of rest to the laboring heart muscle and, on the other, assist it directly to regain in some important degree its waning tonus.

Total abandonment is usually represented by an attitude of utter hope-

* If one were to try the experiment of substituting them initially for the "tonic" or "digestant," so universally used in obscure cases of "nervous debility" and "dyspepsia," he might be greatly surprised by the frequency of decided favorable effects.

lessness and a disinclination to do more than smooth the victim's pathway to the grave.

Keep fighting.

In mitral cases, especially and, to a lesser degree, in other common cardiovascular lesions, the patient may go to an apparent utter cardiac breakdown with renal congestion, hepatic and pulmonary engorgement and general anasarca and still respond to treatment and live for years.

Absolute rest is but too seldom enforced and rationally maintained. The patient is up and down, even though mostly down, and if improvement occurs is not held to a schedule demanding a gradual increase of activity, such as is needed in the generality of cases if improvement is to be had.

In far too many instances in which absolute rest *is* enforced it is maintained for too long a period and almost immediately thereafter the patient is permitted to resume his or her activity.

Too abrupt a transition from absolute rest to physical activity is oftentimes less effective and more dangerous than ambulant treatment.

Abundant time indispensable.

Above everything in cardiac therapy must be placed the element of time when one deals with its severer types or the cases of the nutritionally depressed asthenic type. These latter demand treatment which, as elsewhere stated, is practically identical with that of so-called "neurasthenia."

Greatly shortens life.

The After Lifetime of the Cardiopath.—*Cardiovascular disease greatly reduces the life expectancy of its "group,"* but the duration of life, in the case of any individual, involves a host of factors among which are the nature of the lesion, its cause, the constitutional peculiarities of the individual, his environment, habits, occupation and, above all, his own intelligence and common sense, and the same qualities in his physician.

Individualization of cases imperative.

Wonderful individual resistance.

Futile Forecasts.—Despite adverse conditions and oftentimes despite the demands of an active, useful and even laborious life, certain individuals may and do carry the cardiac lesions of youth into a ripe old age and nothing can be more elusive and uncertain than prognosis in cardiovascular disease or more futile than the attempt on the part of the physician to set a date for the *exitus* of any patient not patently moribund at the time.

Futility of prophecy.

Promise of added days.

With early recognition and prompt medical intervention in cases of actual insufficiencies, not only the life expectancy of the individual but that of cardiopaths as a group will be greatly extended.

MYOCARDIAL OVERSTRAIN

Acute Cardiac Overstrain

Basic factors.

Fundamental Factors.—Physical overstrain may affect the heart in different degrees dependent upon: (*a*) *the congenital structural peculiarities or deficiencies of the individual;* (*b*) *his age, general physical condition and the actual state of his heart and blood vessels at the time of exposure;* (*c*) *the severity, abruptness and duration of the strain imposed;* (*d*) *the habituation or non-habituation of the individual both to physical exertion of the degree attempted, and to such specific coördination of certain neuro-muscular units as is involved*

in the particular form of effort put forth, and, finally and chiefly, the question of toxemia, proximate, actually present, or recurrent.

Physical Fitness and Habituation.—Physical fitness is an important factor, one of the commonest causes of acute overstrain being the attempted performance of tasks suited only to the trained athlete or the frontiersman by individuals who are distinctly out of condition, actually convalescing from an illness, or congenitally unfit.*

Misleading cases.

The author has encountered it repeatedly in well-fed men, outwardly appearing to possess a powerful build, but proving to be of asthenic stock and to carry inherited deficiencies, especially such as involve the heart, blood vessels and lungs. Also in individuals possessing a past replete with athletic prowess, but at the time distinctly out of condition and unfit.

In the well-conditioned athlete the heart is actually slightly smaller after or during some endurance test than at its beginning, being fully able to meet the demand by a combination of increased rate and driving power, this being in direct contrast to the condition of dilatation produced in untrained or structurally deficient individuals.†

* A brief summary of a typical case out of the great number encountered by the author would read thus: Male. Sedentary occupation and habit, aet. forty-seven, weight 200, complains of indigestion (as most of them do) chiefly manifested by dull pain over pit of stomach. Pain usually late in day (*after day's activities*), sometimes seems to follow exertion. Can't do his work as easily as he did formerly. Gets tired quickly when out shooting. Is often drowsy during the latter part of the day. This often passes off after dinner. No definite relation of pain to meal hours, but is always worse when he is constipated or has "gas on the stomach." This patient's heart was found to measure 16 cm. in total transverse diameter and a soft systolic murmur was present, limited to the apex; pulmonary second sound about equal to the aortic tone. All findings negative save as to heart.

His pain had begun three years previously while duck hunting, but he had not related to us the events because of the misleading epigastric localization. On that day he had shot both morning and evening standing in the water and deep mud of a shallow North Dakota slough lake. Shooting was good and when darkness fell and he waded about through the deep clinging mud to gather his ducks the increasing load was hard to carry. After a long wade to shore he felt "all in." Had a great deal of pain in the chest, struggled to shore, felt faint, vomited, and lost consciousness. Finally struggled to his feet and went to camp to lie down, leaving ducks, shells and game behind him. "Felt pretty well" next day but now recalled the fact that his "indigestion" had dated from that day.

Under treatment the pain disappeared wholly in three or four days. He remained under treatment for four weeks. The cardiac outline shrank to 13 cm. and the murmur wholly disappeared.

He has now remained perfectly well during five years.

This represented an acute dilatation occurring in a particularly well-built robust but middle-aged man, leading a sedentary life and wholly out of training. Anyone who has repeated his hunting experience knows well the extreme effort required under the particular conditions present. It is not at all unlikely that some transient antecedent infection may have played a part. The "drop" hearts or those of individuals in poor health dilate far more easily and with less dramatic symptoms in most instances.

† The author has been struck by the frequency with which he has encountered cases of distinct physical impairment in men of athletic records who have suddenly been translated to sedentary pursuits but still seek to pursue sporadically and fitfully athletic pursuits of an extremely strenuous sort.

The Commonest Cause of Minor Heart Strain.—The placid state of physical "unpreparedness" represented by middle age, a sedentary occupation, and a daily life in which the accustomed exercise is represented at best by a round of golf and a walk to the office, is often disturbed by an abrupt autumnal

Middle age.

Indian summer overstrains.

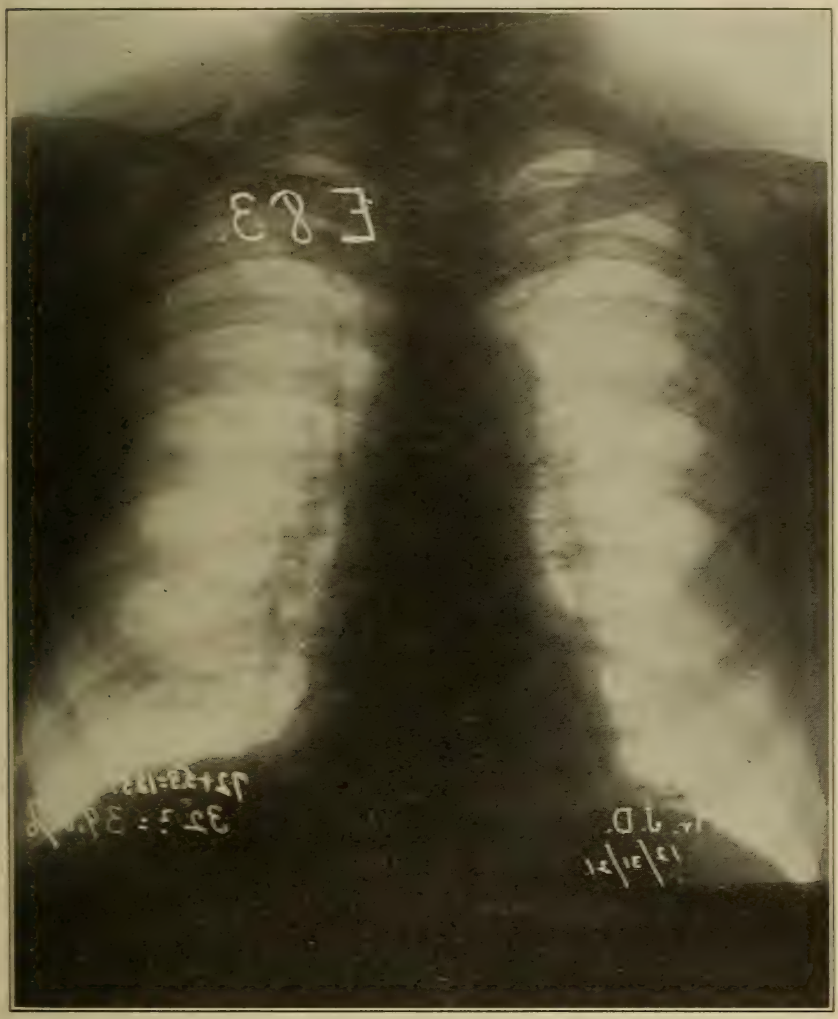


FIG. 349.—Plate made nineteen days after acute dilatation of a supposedly normal heart. Fibrillation was present during eighteen hours after attack. Some precordial distress still present when exposure was made. Total transverse diameter of heart 12.5 cm. (See also Figs. 350 and 351.)

rejuvenescence taking the form of a tendency to play tennis with mistaken strenuosity and perseverance; to climb mountains; take an oar or a paddle "to show them," or hunt the deer over heavy going; all these plunges being taken *without sensible moderation or previous preparation*. Such unwisdom results in a host of cardiac overstrains, for the most part, spontaneously

readjusted, but in some instances working great damage if undetected and unaided.

The Asthenic Heart.—*Considerable attention has already been paid to the peculiar heart of the congenital asthenic ("drop" heart) and one need only*

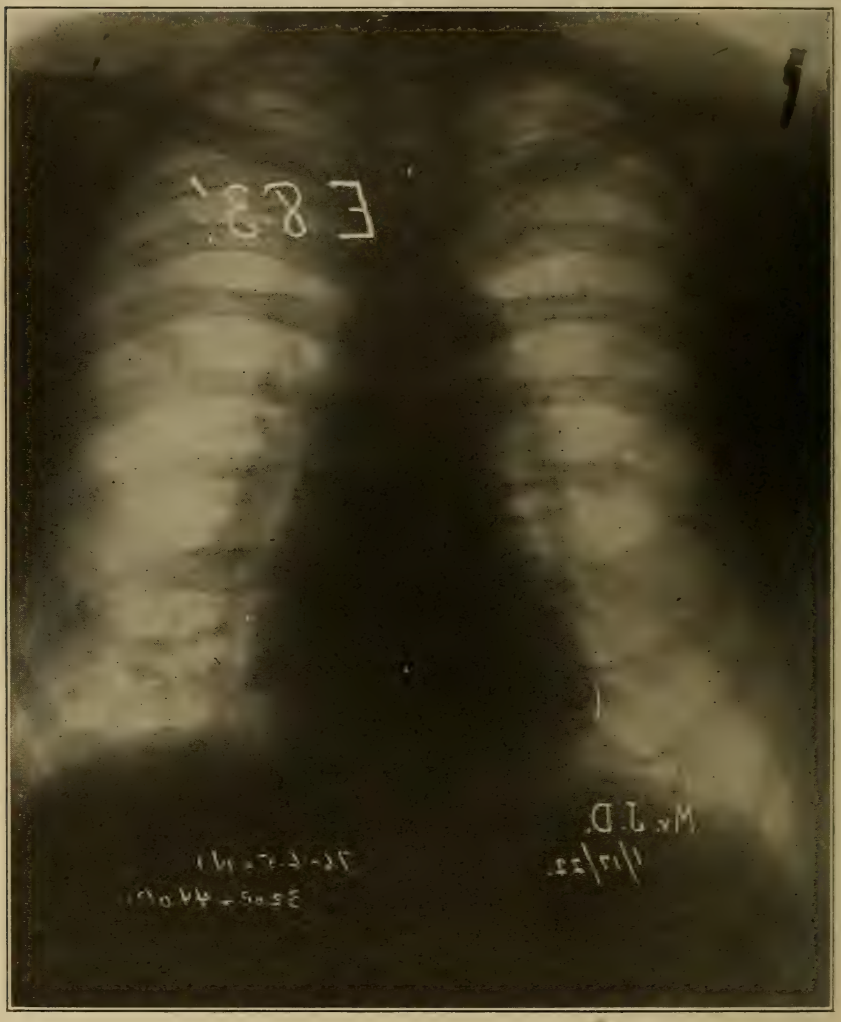


Fig. 350.—Same case of acute dilatation as is shown in Fig. 349. Patient, when apparently fully recovered, slipped and fell on the ice severely wrenching himself. A second attack of acute dilatation followed. Heart is still dilated from second seizure. Total transverse diameter 14.1 cm. Both right and left borders are extended outward. (See Fig. 351.)

repeat in this connection, that such hearts are among the commonest of clinical findings. They are peculiarly prone to transient or persistent loss of tonus or actual minor dilatation, and, to the production of subjective expressions of insufficiency, often remote from the heart itself.

As stated previously, such hearts, even when both overstrained and decidedly

dilated are, at present, almost universally disregarded, because of the fact that even when greatly enlarged they need not equal in transverse diameter the area of the normal heart of a non-asthenic individual and further because of their unfortunate tendency to present the clinical features of so-called "neurasthenia" or the equally dubious syndrome "nervous dyspepsia."

Constantly overlooked.

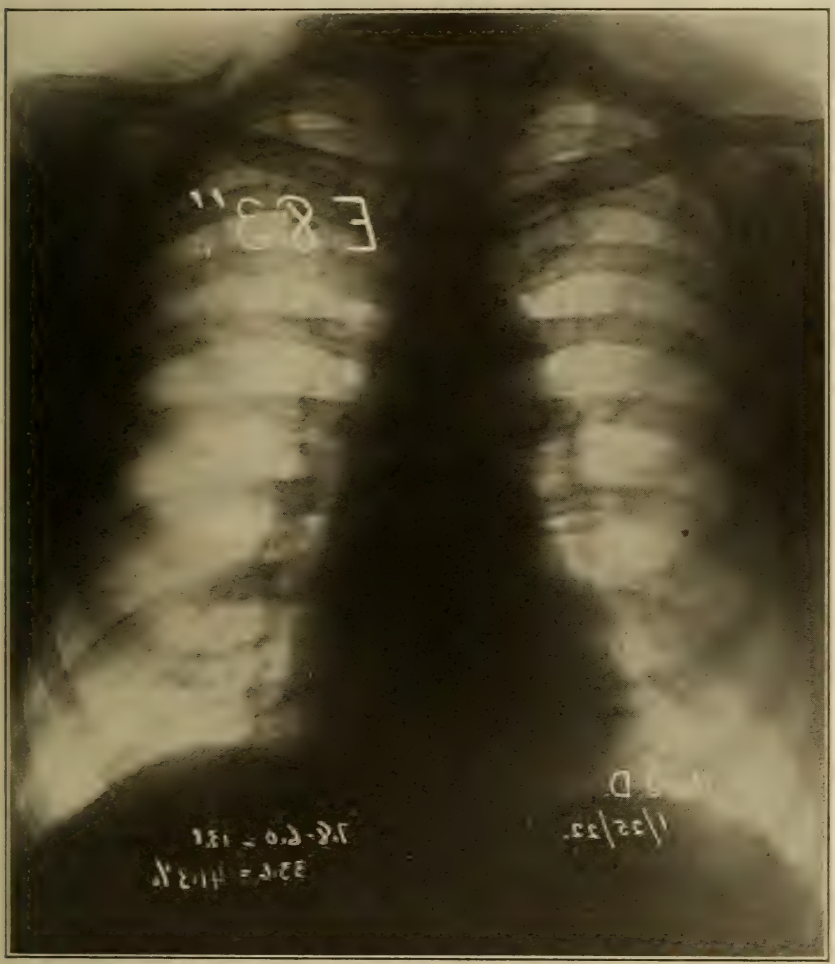


FIG. 351.—Same case. Acute dilatation of the heart. Plate taken nine days later, showing shrinkage of the heart after second attack of dilatation. Heart is still dilated as compared with Fig. 349.

A Common Cause of Overstrain.—*One of the commonest sources of acute or sustained cardiac overstrain lies in the too early resumption of exercise after an exhausting surgical operation or prostrating illness, or even in hasty unconsidered exertion during the actual illness.*

Myocardial toxemia.

We too often forget that for one case of recognized acute myocardial disease there must be a multitude in which a similar though less pronounced and advanced process is present, but undiscovered and undiscoverable save

during periods of impaired tonicity and overstrain. In all severe acute prostrating infections, even though of short duration, the heart is peculiarly liable to suffer from myocardial toxemia of varying degrees and in acute rheumatism affecting the already damaged heart, as also in true influenza, we are peculiarly likely to have larval and obscure or definite and recognizable insufficiency. The former type is almost universally overlooked at the present time during and following such an illness.

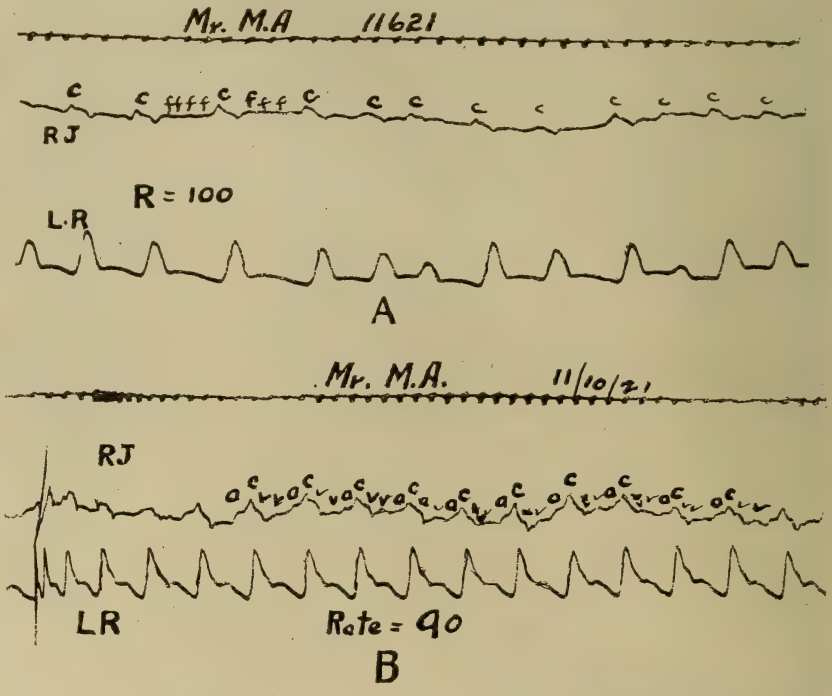


FIG. 352.—Mr. M. A. (*A*) 11–6–21. Temporary fibrillation of the auricles following acute overstrain, and a readily demonstrable dilatation in a supposedly normal "drop" heart. (*B*) 11–10–21. Record taken four days later showing a normal sinus rhythm. Clinically the normal sinus rhythm was reestablished in 24 hours. LR = Left radial. RJ = right jugular.

Subjective Weakness.—In the absence of frankly objective cardiac symptoms the best guide often proves to be the therapeutic test, which unmasks a slight dilatation or loss of tonus, and the evident or merely subjective weakness of the patient. It may be demonstrated easily in many instances that despite a striking loss in endurance and in the capacity for symptomless performance of the day's work or ordinary physical activities the strength of the skeletal muscles is well preserved.

Hurried convalescence.

Many patients themselves date a persistent myocardial weakness and dilatation from an illness during which neither myocarditis nor endocarditis was recognized, but following which there was a forced resumption of activity in the face of a persistent bodily weakness.

Patients should be guarded with special care during and after prostrating tonsillitis, acute rheumatism, influenza, diphtheria, scarlet fever and typhoid; *and their own sensations, properly interpreted, as a rule constitute the best*

Important infections.

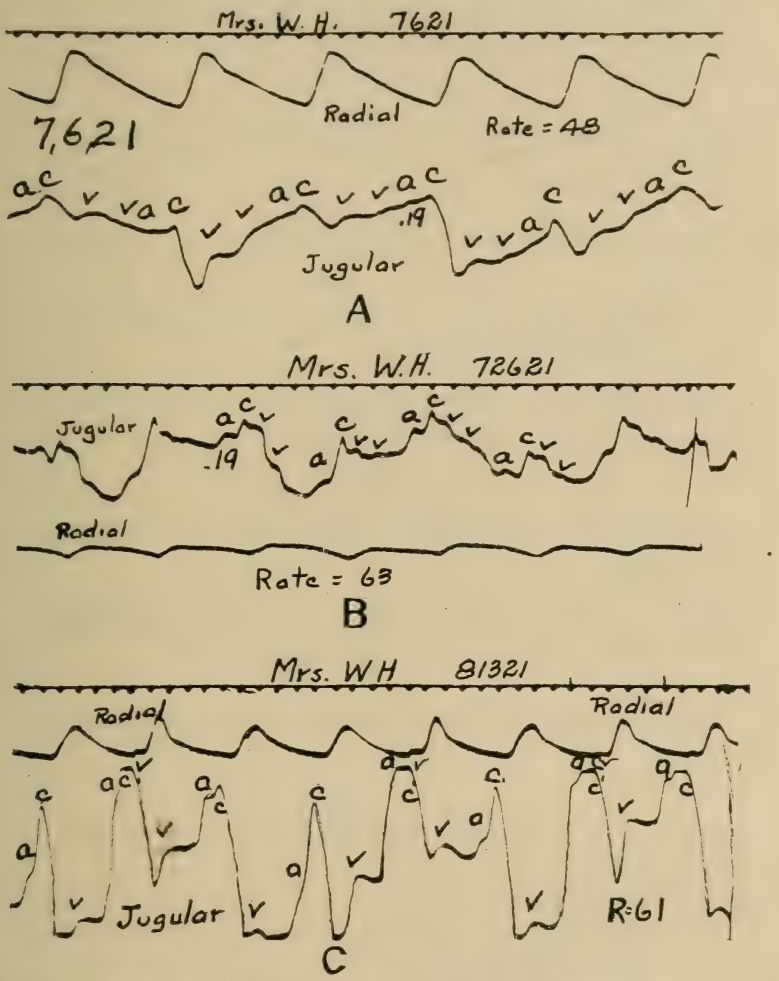


Fig. 353.—Mrs. W. H. Variability in myocardial strength as indicated by polygraphic records. (*A*) 7–6–21. Record taken before operation showing high radial thrust and indicative of good myocardial strength and tone. (*B*) 7–26–21. Record taken 6 days after laparotomy under local anesthesia for supravaginal hysterectomy with removal of both appendages. Note marked flattening of radial as compared with that in (*A*), indicating a decided loss of myocardial tone and strength after operation. (*C*) 8–13–21. Record taken 24 days after operation. Compare radial with that of (*A*) and (*B*) and note that even then the record indicates that myocardial tone and strength have not yet been completely regained. Patient in hospital under ideal conditions.

general guide for the physician, but do not replace a physical examination. Nor should they be allowed to drop out of sight wholly for months afterward, for both primary myocardial damage and actual endocarditis may delay

May initiate decompensation.

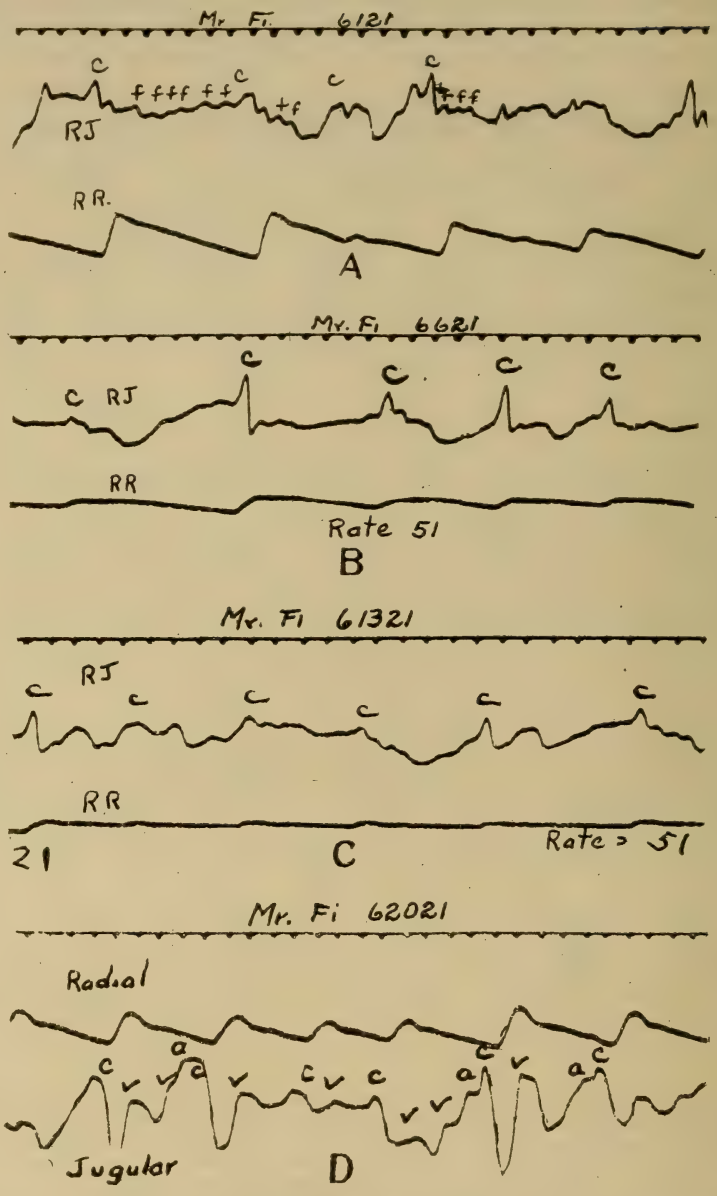


FIG. 354.—Mr. Fi. Series of four polygraphic records in a case of fibrillation during hospital regime, showing marked variation in myocardial strength and tone as indicated by the radial record. (*A*) Note the high radial thrust indicating good myocardial strength and tone. (*B*) and (*C*) show a progressive diminution of myocardial strength and tone, while (*D*) shows that myocardial strength is only partially regained. The myocardial weakening was due to a very slight cardiac overstrain as a result of a breach of instruction as to graduated exercise. RR = right radial. RJ = right jugular.

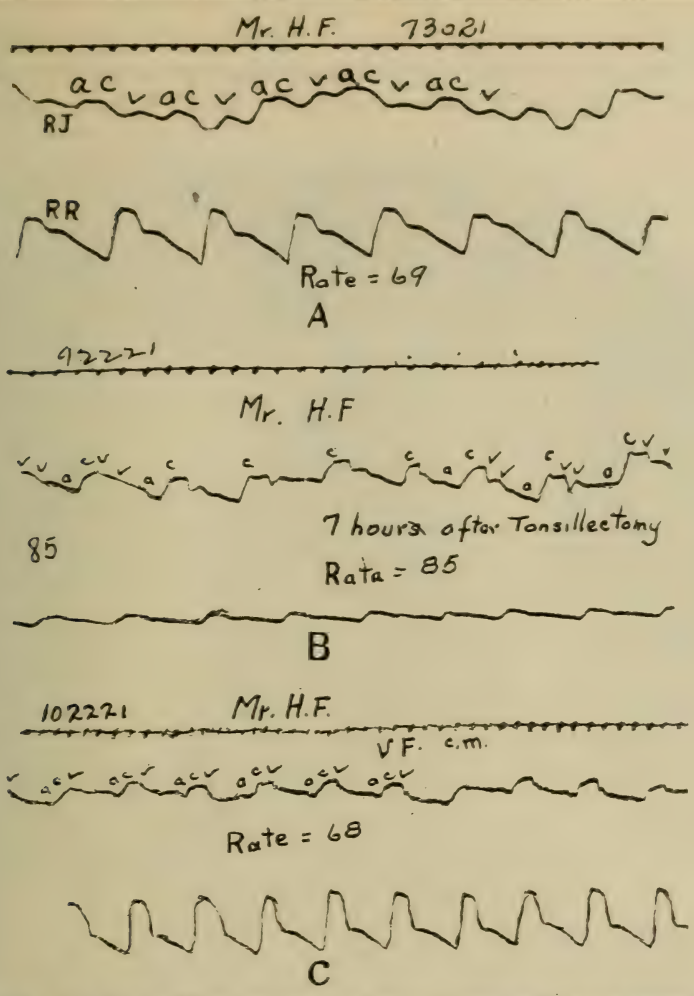


FIG. 355.—Mr. H. F. Three polygraphic records showing effect on myocardial tone and strength of a so-called "simple" tonsillectomy. Patient in hospital under ideal conditions. Compensated myocardial defect. (*A*) Record taken before operation. Note height of radial thrust. (*B*) Record taken 7 hours after tonsillectomy. Compare height of radial with that of (*A*). (*C*) record taken 30 days later. Note that height of radial thrust indicates that myocardial tone and strength have been regained.

their manifestations for several weeks or months after apparent recovery from the primary disease.*

* It is the failure to guard patients in convalescence and examine them at intervals afterward which makes our early recognition of certain chronic endocarditic valvular lesions so rare an achievement (mitral stenosis, aortic stenosis).

For the patient, early detection of his heart lesion is quite as desirable and necessary as the early detection of pulmonary tuberculosis. Most of the tuberculous undergo a spontaneous recovery and the same is true of, and the same favorable issue attends, a host of instances of myocardial involvement during acute illness, yet in the case of both, early recognition and proper regimen, wise counsel and judiciously timed treatment are invaluable, for in either disease the cases recognized and treated only when gross and obtrusive signs are present, represent almost hopeless conditions, though, in each, life may be greatly prolonged.

Careful percussion by modern methods, having in mind the peculiarly small hearts of patients bearing the asthenic stigmata and the narrowed normal limits represented by the table of Cohn, reveals the relative frequency of minor dila-*

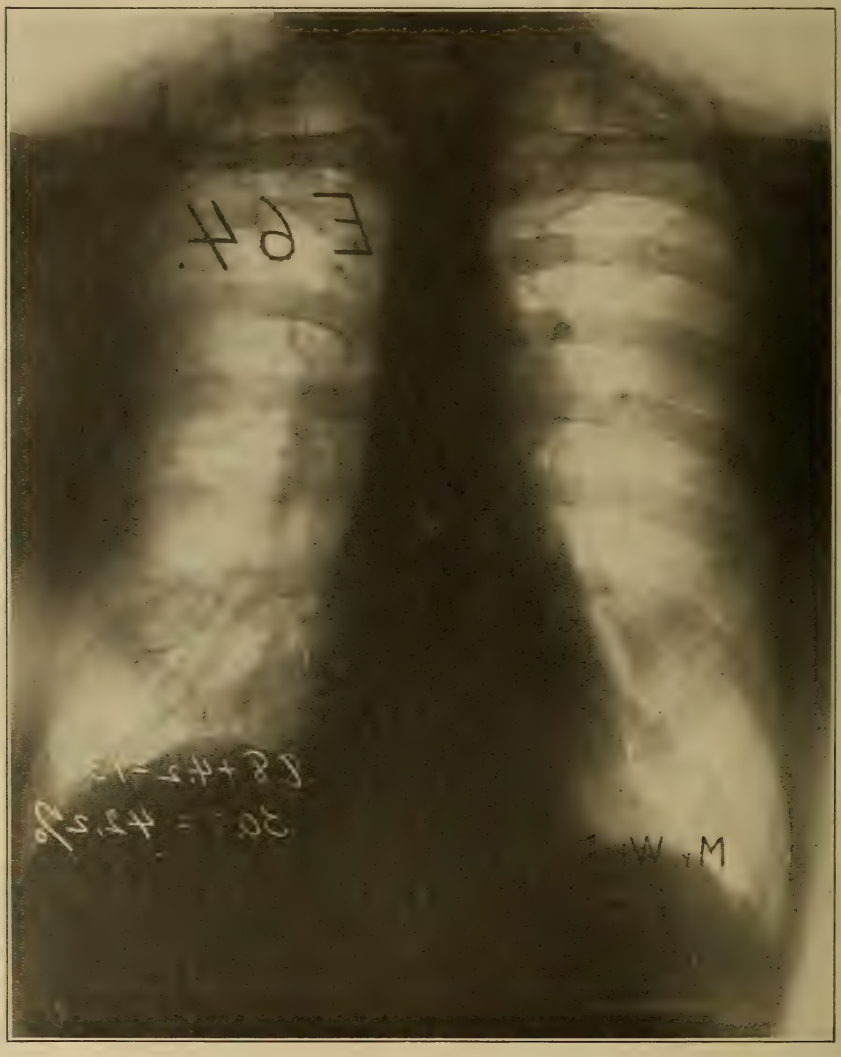


FIG. 356.—Ready dilatability of the "drop" heart. This figure represents an exposure made during a period of continuing overstrain and shows a heart diameter of 13.0 cm. This patient has passed through three attacks of acute dilatation, two of which threatened his life and were slowly recovered from. The third produced effects lasting acutely only a few hours although the dilatation was pronounced. The two attacks of massive dilatation were associated with major surgical operations; the third with simple physical overstrain. Compare this exposure with that shown in Fig. 357.

tations following acute infections. The promptness of amelioration or disappearance of symptoms, and sometimes of gross border recession, under rest and full therapeutic test doses of digitalis is oftentimes astonishing.

*See opening pages of heart section in this volume.

The Split Second Sound.—As previously stated, the author during several years has been studying a split second sound, audible only in the third left interspace, *in its association with past or persisting cardiac overstrain and associated minor dilatation, often accompanied by more or less obscure and,*

A sign of interest.

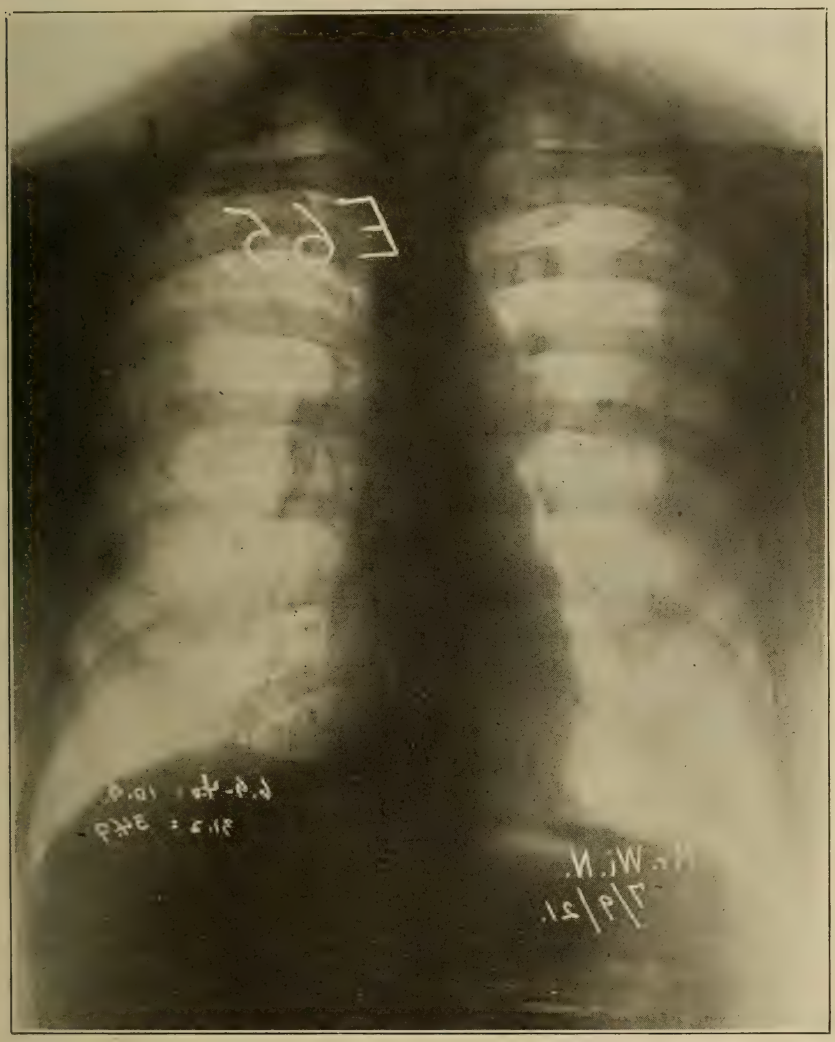


Fig. 357.—Same heart as shown in Fig. 356. Under treatment the cardiac diameter has become reduced to 10.9 cm., and all symptoms of decompensation have disappeared. Nevertheless this patient's reserve is definitely and permanently impaired although sufficient remains to enable him to carry on all of the necessary activities of a business life and a reasonable amount of recreational pursuits.

oftentimes, referred symptoms of cardiac discomfort or primarily baffling subjective manifestations. It is usually present in middle-aged individuals, but is not uncommon in young people and, when persisting after the dilatation is relieved, usually assumes the form of a short diastolic, aortic murmur.

It is almost invariably associated with a capillary pulse and, in its later stages* with an imperfect *pulsus celer* and increased pulse pressure.

Signs of Acute Extreme Overstrain.—*Extreme precordial oppression, marked dyspnea, pallor, dizziness, perhaps cyanosis and, not infrequently, severe or agonizing anginal pain and actual syncope are the symptoms observed in extreme cases.*

The heart is usually distinctly enlarged, the pulse rapid, weak and frequently irregular. Recovery may be extraordinarily prompt if the victim is in good physical trim and, in well-trained men, may pass off promptly and leave no visible effects behind. In others, and especially such as are already carrying recognized or unrecognized myocardial lesions, and in those structurally and nutritionally deficient or wholly out of training, the bad results may be prolonged or permanent or even promptly fatal.

Preëxistent Lesions.—The effect of acute overstrain upon the illy or even fully compensated heart carrying some preëxistent lesion is such as will often initiate a fatal decompensatory process and this is especially true of instances in which the excessive effort is one lying outside of the habitual activities of the individual. The man with the double aortic and mitral lesion may perform the arduous duties of the locomotive fireman for years only to encounter his breakdown or actual *exitus* in the overexertion of a baseball game or the effort of cranking a refractory motor car.

Habituation important.

The diseased heart, even though fully compensating, is very susceptible to acute infections, notably to acute rheumatism and to influenza; many serious overstrains occurring during convalescence from such diseases.

Overstrains of Adolescence.—Cases of acute cardiac overstrain are especially frequent in young boys at or about the age of puberty who are growing rapidly and seem in many instances to attain a body mass somewhat disproportionate to the strength and size of the heart, this being especially true of such as carry the congenital drop heart.

Deficient horse-power.

In many, if not most, instances the initial impairment of compensation dates probably from some past infection and any such case demands a careful search for hidden septic foci.

In some instances of this type coming under the author's observation dilatation and recurring overstrain have produced more or less frequent attacks of breathlessness, vertigo and syncope which in several instances have been interpreted and treated as *petit mal*. A short course of treatment directed to the heart, or the mere avoidance of *overexertion*, have resulted in prompt and permanent relief, and future growth and development balances the defect in most instances.

Seldom serious.

The Heart in Laborious Occupations.—With better diagnostic methods, the use of the sphygmomanometer, improved case-taking and an appreciation of the intimate relationship between the size of the heart and the body weight and musculature, no less than a wide knowledge of pathologic associations and etiologic factors, we must consign nearly all cases of cardiac disease, supposedly due to occupation itself, to the scrap heap.

Work alone rarely a cause.

* As yet no cases have been watched for more than a decade.

It is **conceivable** *that certain occupations of an excessively laborious sort, involving extreme fatigue and constantly recurring maximal physical effort, may cause disease of the heart in the case of those structurally unfit, of bad habits, or weakened by illness.*

Other factors always present.

On the other hand, it would seem proven that the effort involved in the most arduous occupations can be fully met by the normal heart which is habituated to its burdens.

In the cases of congenital "drop" heart, temporary overstrain and chronic inadequacy often force its possessor to abandon heavy labor, but an astonishing response to demand is seen in most instances.

In nearly all cases of heart overstrain fundamental defects of physical structure or in the habits of the individual are the more prominent factors and, in nearly every instance, *past disease, chronic latent infections*, or other similar conditions recognized as potent causes, play a chief part.

ACUTE PARENCHYMATOUS MYOCARDIAL DEGENERATION

Acute Myocarditis.—This, the common form of acute myocardial disease, in varying degrees is commonly associated with *major* acute infections and especially with true severe acute rheumatism, diphtheria, influenza, scarlet fever, pneumonia, typhoid or typhus, and smallpox,* but can and occasionally does accompany or follow tonsillitis, severe measles and other acute infections including gonorrhea, and post-partum sepsis. One should bear in mind also the toxic effect of the excessive use of alcohol upon the myocardium and the special susceptibility of alcoholics to myocardial complications.

Causative ailments.

Morbid Anatomy.—It is characterized by diffuse, cloudy swelling, and granular and oftentimes fatty degeneration or even hyaline or waxy and calcareous changes of the muscle fibers, and the heart muscle is flabby, pale and easily torn.

"Mushroom heart."

The degenerative process may proceed from the epicardial or endocardial surface into the interior or extend along the connective-tissue framework. Aschoff and Tawara believe that certain perivascular aggregations of nucleated round or oval basophile cells lying in the perivascular connective tissue are especially characteristic of or peculiar to *rheumatic* myocarditis. Such a process in an interstitial form would be peculiarly likely to affect conduction through the bundle of His.

As will be seen readily, it is a composite process, as are all the myocardial degenerations, whatever the type.

Silent Transient or Permanent Forms.—These are doubtless extremely common and are usually and often unavoidably overlooked, much to the damage of the patient. They are not uncommon in a transient form in toxic "drop" hearts and afford most interesting near-replicas, in miniature, of the severe form in many such cases.

Masked cases.

* The order given is based upon the author's personal experience and is somewhat different from that usually given. In the old pre-antitoxin days diphtheria would have largely exceeded all other causes of *fatal* myocarditis.

The flabby myocardium of the asthenic seems peculiarly susceptible to, yet capable of recovery from, the toxemia of the commoner acute infections. Its endothelium has been considered to be *relatively* immune, but, as stated elsewhere, since he has had an opportunity to study the modifications of form assumed by the "drop" heart in the presence of endocardial and myocardial involvement the author believes such assumed immunity improbable.

Septic Form of Myocarditis.—Septic emboli may block the terminal coronary branches and form miliary or pea-sized abscesses often yielding positive cultures of the causative organisms. Occasionally large abscesses form and rupture into the heart, or they may undergo absorption and inspissation leaving areas of scar tissue behind them.

This is especially true of rheumatic, typhoidal, and influenzal cases in which the lymph spaces surrounding the vessels of this blocked area are filled with cells which in rheumatism and typhoid usually prove to be mononuclear.

SYMPTOMS OF ACUTE SIMPLE MYOCARDITIS.—It must be remembered that in all severe acute infections the heart muscle is likely to suffer in greater or less degree without producing obvious or obtrusive symptoms or, of necessity, inducing permanent lesions.

Extremely important.

Subjective Weakness.—*This sense of exhaustion the author regards as the most important early indication of myocardial toxemia, especially when coming on somewhat rapidly, and it is especially notable in influenza which in its true form attacks the heart muscle with peculiar severity and relative constancy.*

Easily overlooked.

Subjective vs. Apparent Dyspnea.—*Subjective dyspnea is also an early symptom of great importance, often disregarded or overlooked, and with it might be included certain vague sensations of precordial oppression or discomfort, frequently discovered only upon direct inquiry. An inability to hold the breath, a tendency to occasional sighing respirations or a visible air hunger, temporary or persistent, may be the only evidences of dyspnea.*

On the other hand, it may be decided or even extreme in frank, severe cases.

Facies.—Marked pallor, pale lividity or moderate cyanosis may be present or appear abruptly and constitute signs of great value and significance in connection with other findings.

Unsuspected fatal cases.

Increase in Cardiac Area.—With a more general appreciation of the true normal heart boundaries and better methods of percussion many more acute myocardial lesions will be recognized and fewer instances encountered of a dramatic *exitus*, wholly unanticipated by the physician.

Minor cases.

Minor dilatation is not at all uncommon in the various everyday infections of a severe type and not infrequently becomes persistent and troublesome later if the patient is not protected against the too early resumption of physical activity.

Massive Dilatation.—*In severe cases the dilatation may be enormous and usually affects the left ventricle chiefly, but, to some degree, the right also.*

Abnormalities of tone.

Heart Sounds.—To him who is accustomed to demand normal heart sounds and is therefore familiar with their qualities and variations, the weak-

ness, impurities, soft murmurs, "murmurishness," sharp brevity, or association with soft murmurs, so frequent in myocardial degeneration and inflammation, will be apparent.

Gross, distinct, clearly defined murmurs are often wholly absent even in the most serious cases as they may be in certain of the excessively weak and widely dilated hearts of terminal, chronic myocardial degeneration, and for the same reason.

The pulse may be merely rapid, extremely fast, or, distinctly slowed from true heart block or vagus involvement and resulting excessive inhibition.

The rhythm is often, though by no means always, more or less markedly disturbed and any form of arrhythmia may be present. Increase of rate, especially marked on slight exertion, is a common symptom and an important one according to its degree. So-called sinus arrhythmia is especially common during and following acute infections in the young.

Gross Decompensation Signs.—Any or all of the signs already described under cardiac insufficiency may appear in severe cases and these may be those of left or right heart type or both together. Any form may be present.

One has only to review the disastrous series of degenerative changes constituting the pathologic picture of myocarditis to realize the extraordinary ease with which dilatation and severe or extreme decompensation may be brought about by even slight exertion in acute myocarditis. Among the results are pulmonary or hepatic engorgement, reduction in the amount of urine, and other evidences of passive congestion, and multiple infarctions due to detached particles of clots, formed in the auricles or ventricles which may bombard the tissues supplied by the systemic and pulmonary arteries.

Pain.—Acute pain is unusual though the most extreme angina has been present in a few of the author's cases.

Epigastric Distress.—*A subjective sense of distension, epigastric, precordial or distinctly substernal, or its opposite, a sense of constriction, may or may not be observed in various degrees of severity. Vomiting and excruciating epigastric pain may accompany any* **abrupt increase** *of dilatation and, as they tend to increase the heart weakness, these often prove forerunners of death.* An ominous sign.

Vasomotor Symptoms.—These are frequently present in the form of cold hands or feet, blueness of the extremities, nose, ears, and, usually, as a most suggestive associated sign, a markedly lowered blood pressure. Low blood pressure.

Urinary Signs.—A sudden drop in quantity or a persistent undersecretion may be most significant though the febrile urine itself is scant and due allowance must be made if fever of any considerable degree is present. Abrupt diminution.

Vigilance Demanded.—One must never forget the danger attending exertion or excitement and the absolute necessity for close oversight.

Cases presenting few symptoms and but slight dilatation may go into fatal collapse without warning, and dilatation of the most extreme type may occur with appalling rapidity.

Although many cases are silent, general symptoms in others may be most severe and, in the septic case, strongly resemble those of malignant endocar-

ditis, septic symptoms predominating and positive cultures being usually obtainable from the blood.

Pancarditis.

It should also be remembered that a *pancarditis* may be present, especially in children, in which event the endo-, peri-, and myocarditis are practically coincident.

Care in convalescence.

If any decided evidence of acute myocarditis has been present in a given case, the utmost caution should be observed with respect to his resumption of activity. Many cases of chronic myocardial degeneration owe their inception to the neglect of this rule and sudden death has occurred on many occasions. Especial care is necessary in convalescence from influenza, diphtheria, acute rheumatism and scarlet fever.

THE CHRONIC MYOCARDIAL DEGENERATIONS

CHRONIC RESIDUAL MYOCARDITIS.—A Transition Stage.—It is wholly rational to assume that after an attack of myocarditis or as the result of recurrent cryptogenetic sepsis, one often deals with a residual chronic myocarditis which still carries as its predominant characteristic the changes incident to the antecedent acute toxic process, but will be gradually transformed into one of the chronic degenerative forms of the disease.

Bridges acute stages and chronic degeneration.

In this phase one still finds round-cell infiltration of tissue and parenchymatous degeneration of the muscle fibers.

Cardiosclerosis.

Sclerotic Changes.—Coincidently there will be a tendency to diffuse sclerotic changes foreshadowing the probable future dominance of a myofibrosis, or *cardiosclerosis, the commonest type of chronic myocardial degeneration.*

Softening of the heart.

Myomalacia Cordis.—Should the local embolic processes have been especially marked, replacement of muscle fibers by scar tissue will be evident and if extensive may yield the picture of *myomalacia cordis* and subject the patient to the risk of cardiac aneurysms, rupture and sudden death.

In certain cases, all active *inflammatory* changes may subside; in others, there is a tendency to renewed infection and myocarditic activity, perhaps many times repeated. On the other hand, it usually takes on one of the other forms of extremely gradual chronic degeneration associated with a more or less effective (adaptive and defensive) hypertrophy.

No characteristic symptoms.

Diagnosis.—There are no specific symptoms of *actual* "chronic myocarditis" aside from those of cardiac insufficiency and its presence is merely an assumption based upon probabilities having special reference to known acute infection in the past.

FATTY OVERGROWTH.—*An Important Distinction.*—Even though in many cases the two conditions are associated, "fatty heart," signifying fatty overgrowth and infiltration, as a specific pathologic process, should be sharply distinguished from "*fatty degeneration*" of the heart, a far more serious condition.

Conditions inviting fatty deposit.

Obesity and Sedentary Pursuits.—Fatty infiltration or overgrowth of various degrees is of especially frequent occurrence in obese individuals who

are heavy eaters, heavy consumers of alcohol, or both, and who lead an indolent or sedentary life.

Pathology.—At first it is merely an exaggeration of the normal epicardial fat, such as is present normally in varying degrees beneath the endothelial layer of the visceral pericardium within the sulci, and it may so remain; but in certain persons the deposit becomes greatly increased and spreads over and even invades the underlying myocardium, much as does the connective tissue in a case of "interfascicular fibrosis." Pathologic exaggeration of the normal deposit.

The amount thus accumulated may be enormous, may envelop the whole heart and even equal or exceed the weight of that organ.

In itself it seldom exercises any fatal degree of adverse influence, but it does superadd a certain amount of interference with the integrity and functional adequacy of the cardiac musculature, favors the increase of any coexisting degeneration of the myocardium, and, to some extent, mechanically embarrasses the heart's action and affects the intrinsic circulation. Its adverse effect, though usually relatively slight, is intensified by any concurrent obesity and general muscular flabbiness existing in its possessor. Effects.

Obesity and Cardiac Overstrain.—In this connection the adverse influence of an overplus of body fat, when allied to relative or absolute muscular weakness should be strongly emphasized.

The individual, who in the course of a few months or years adds 75 or 100 pounds or more to his weight, owes it to his heart to keep his muscles in trim for, however well "packed" the adipose tissue may be, he is, nevertheless, carrying constantly a heavy load and, if his muscles are not coincidently strengthened, this must of necessity constitute a chronic overload most evident under physical stress. His longevity in great measure depends, therefore, upon a direct reduction of this burden by any proper means or such exercise and outdoor living as will serve to keep him in training for the inevitable persistent increase of physical effort required. Ratio of heart strength to body weight. Constant overload.

Insurability.—Life insurance companies justly fear the flabby-muscled, big-bellied man whose weight exceeds, to any marked degree, the tabular figures and this fear is intensified if a history of rheumatism or other severe infections in the past, sedentary habits, overeating and indulgence in alcoholics, are also in evidence. Big-bellied overweights.

Common Associated Pathologic Conditions.—There are three conditions, aside from actual fatty degeneration, which more or less commonly may be found associated with disabling cases of fatty overgrowth of the heart.

The most interesting of these is an atrophy which may be wholly obscured by an increase in the apparent size of the organ due to fatty deposit and dilatation. The second and most important is sclerosis of the coronary arteries; the third, cardiosclerosis. Submerged atrophy.

Other forms of degeneration may of course exist and almost any one of them *may* constitute the most potent factor in any major cardiac insufficiency present in the individual case.

"*Fatty hearts*" *rarely hypertrophy to any considerable degree* and the obstacles which the fatty bond offers to that full freedom of diastolic relaxation and True hypertrophy rare.

Coronary deficit.

attendant coronary flushing of the heart muscle which is so vital to myocardial nutrition, makes it easy to understand the handicaps ultimately imposed. It is a well-known fact that any obstruction to the descent of the diaphragm or persistent upward pressure upon it greatly embarrasses the work of the heart and Wenckebach has called attention to the high position of the heart frequently demonstrable in large-bellied individuals in the sitting posture and especially in those who wear tight belts or habitually adopt attitudes which brings upward pressure to bear.

High diaphragm.

Temporary exertion-dyspnea.

Symptoms and Physical Signs.—*The shortness of breath on exertion so often observed in the obese does not* **necessarily** *mean a degenerative disease of the heart and often wholly disappears when circumstances lead to a proper amount of daily exercise on the part of the individual; yet it constitutes a factor of importance in connection with other symptoms or when persisting in a decided degree.*

General symptoms and signs.

Aside from this the symptoms in most instances are merely those of cardiac insufficiency, modified by the type of the dominant pathologic myocardial lesion, and emphasized usually by its occurrence in persons who show a manifest discrepancy between body weight and heart mass, as inferred from their general contour contrasted with their skeletal musculature and its tonus.

The Cardiac Outline.—The cardiac outline is often decidedly misleading, the heart being apparently enlarged to right and left even though dilatation is not present. The heart sounds and any murmurs of relative insufficiency may be relatively indistinct, the apex-beat faint or absent and the superficial cardiac dulness usually less flat in tone because of the underlying fat.*

Secondary signs often extreme.

Obscure Advanced Cases.—Cases of this type associated with atrophy or a silent dilatation are frequently encountered in the stage of decided incompensation made evident by hepatic engorgement, moderate edema of the feet and ankles, decided dyspnea on exertion, signs of passive pulmonary congestion and renal stasis.

The combination of these frank symptoms with an almost silent heart forms a striking, and, to the uninitiated, a puzzling combination.

Worse than primary degenerations.

Prognosis.—*The disease is usually manifested during or after the fourth decade of life and only slight symptoms of cardiac weakness may be present for years, but if gross decompensation is present, the outlook is distinctly worse even than in primary, uncomplicated, degenerative, myocardial lesions.*

Peculiar combination.

Many cases are associated with an interstitial nephritis and arterial hypertension which makes a peculiarly unfortunate combination.

Furthermore, if cardiac atrophy or any marked degree of true fatty degeneration is present, the cases are peculiarly unresponsive to treatment and especially so to *large initial test doses of digitalis or its congeners.*

Tendency to right heart failure.

Such cases usually lose weight as the terminal stages approach and present a peculiar combination of anemia, flaccid muscles and weak heart. Curiously enough failure is earlier and more marked in the right heart.

* In the absence of distinct signs of emphysema the presence of such a modification of cardiac flatness in a stout individual is distinctly suggestive of decided fatty overgrowth.

THE CARDIAC INSUFFICIENCY OF THE GLUTTON AND THE SOT.—In general this condition is merely that of "fatty heart" or a greatly disturbed ratio of body weight to heart mass and vigor plus a myocardium weakened by alcohol and the toxic products of an abused metabolism, occurring in notorious gluttons and habitually heavy drinkers or drunkards. Beer-heart.

Special mention should be made of the so-called "hypertrophic beer heart," encountered in large numbers in Munich, Bavaria, but by no means infrequent in other parts of the world.

Brewery Drivers and Ice Cart Men.—In America it is more frequently seen in persons who combine an excessive consumption of beer and resulting plethora, with an excessively laborious occupation. It is best exemplified in ice cart men and drivers of brewery wagons, both of whom often combine lifting and the carrying of heavy burdens with frequent potations.* In these cases it is supposed that the large amount of fluid introduced, by creating hypertension, plays a considerable part in the cardiac decompensation. Excessive drinking and laborious occupation. Victims of interested hospitality.

Human Vats.—In Munich it is said to occur in relatively young persons who drink beer in prodigious quantities "day in, day out," with only relatively short pauses.

The author's patients have almost wholly been in the "forties." The decompensatory signs have been especially those of right heart weakness, though the left shows marked hypertrophy and dilatation. Common pathologic type.

Extreme dilatation with frank symptoms of tricuspid insufficiency constitute the common finding and the patients show, as a rule, a decided and relatively prompt response to a diversion of the flooding tides of beer, rest and cardiac stimulants. On the other hand, relapses are common and prompt unless the occupation and the habits alike are changed. Relapses common.

FATTY DEGENERATION.—*This serious pathologic change, though often concurrent with fatty heart in the extreme forms of that lesion, is of itself absolutely and entirely different from fatty overgrowth or infiltration. It is associated in some degree with almost every form of myocardial degeneration and, when the dominant degenerative process, constitutes one of the most serious and intractable of myocardial lesions.* Serious if dominant.

It is an actual destructive fatty degeneration of the muscle fibers affected, which undergo finally complete loss of structure and function. True fiber destroyer.

It is especially common in association with various forms of severe chronic anemia, primary, secondary, or post-hemorrhagic, constitutes one of the characteristic features of acute phosphorous poisoning, and is a not uncommon post-mortem finding in deaths from exhausting chronic disease.

Symptoms.—*As a dominant process, it is especially common in middle age and its symptoms and physical signs in no wise differ from those described under the later stages of fatty infiltration, being merely those of chronic, progressive, and relatively intractable cardiac insufficiency occurring, however, in fat*

* Ice cart drivers are so frequently "treated" to beer, on their rounds, not only in saloons but in private kitchens as well, as to make them heavy drinkers in many instances. The head of a large corporation informed the author that a thorough investigation of the habits of his own men revealed the fact that 80 per cent. were steady drinkers.

and thin alike, and, not infrequently, associated with a sallow countenance and a special resistance to and intolerance for drugs of the digitalis group.

ENDOCARDIAL LESIONS

Always secondary.

ENDOCARDITIS.—Definition.—An inflammation of the endocardium, which may be acute, subacute, or chronic; is always secondary, and may assume a benign, malignantly septic, or septically recurrent form.

It affects by preference the left heart and, chiefly, the opposed surfaces of the valves; more rarely, and oftenest in the malignant, septic and chronic

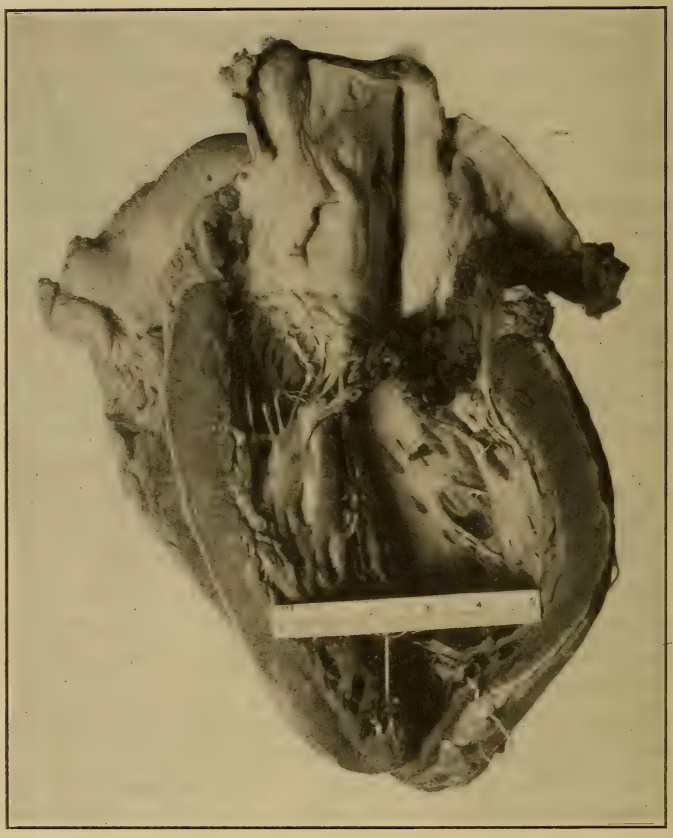


FIG. 358.—Acute vegetative mitral endocarditis. The right side of the photograph shows the cauliflower-like mass of vegetations almost replacing the valve and running up on the endocardium of the left auricle. (Viridans type.) Case of woman, age 28, who "never had rheumatism"(?). At 22 had jaundice and enlargement of liver. Married at 23. After birth of child was never well. About 2 years before death had right-sided hemiplegia. Slowly recovered for a year and then had left hemiplegia. Infarcts of both brain and kidneys were found at post-mortem. (*Courtesy of H. E. Robertson.*)

recurrent ("*streptococcus viridans*") cases, the adjacent mural endocardium. It tends to produce either transient or permanent vegetations or deformities, is overwhelmingly chief in the production of chronic valvular lesions *of children and young adults;* in its acute and recurrent malignant types is al-

most invariably fatal, and in any of its severer forms seriously affects valvular function.

The degree of primary inflammation and the occurrence, depth and subsequent behavior of the necrotic process depends upon the condition of the myocardium, the virulence of the causative organism and the resistance of the patient.

In the non-malignant forms the primary effect may be slight and, probably, *transient* as is shown in certain experimental work and suggested by clinical experiences. Ordinarily vegetations upon the valves result from the formation of bacterial emboli and a reactive fibrinous exudate. Valve emboli.

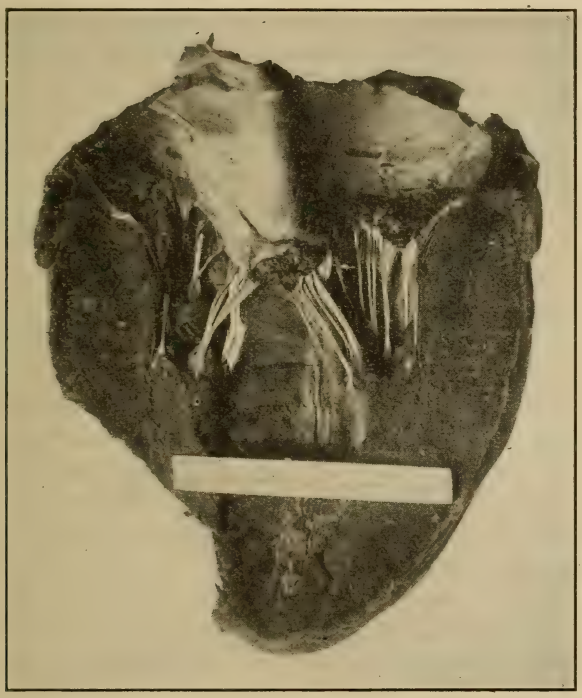


FIG. 359.—Acute vegetative mitral endocarditis. A ridge of soft reddish thrombi is located on the auricular surface of the line of closure.

Etiology and Pathology.—No microörganism can be charged with the responsibility for acute endocarditis. The pneumococcus, staphylococcus, streptococcus, and more rarely the colon bacillus have been found in endocardial lesions. Acute rheumatism,* the acute forms of septic infection, and the toxins of various acute infectious diseases are associated with both the benign and ulcerative cases, and the true sclerotic forms are caused by the same factors that are active in arteriosclerosis. It is believed that an endo- Associated ailments.

* Acute rheumatism is responsible for about 90 per cent. of the chronic valvular lesions encountered *in children and young adults*. *Valvular lesions* of a pronounced type *developing after* the age of 30 are overwhelmingly syphilitic in origin, though rheumatism is even then a decided factor in those of the mitral type.

carditis of some degree exists in nearly every severe case of acute rheumatism (its most common cause) and that many spontaneous recoveries from transient unrecognized lesions occur. This can be true when the inflammation is extremely slight and fleeting, and in mitral stenosis the classical symptoms of that lesion may be so long postponed as to cause over-hasty primary conclusions as to the patient's escape from valvular involvement.

Acute rheumatism.

Children are especially liable to this acute complication of severe acute rheumatism, 60 to 80 per cent. of those cases occurring between the ages of ten and fifteen presenting demonstrable lesions, and an astonishing number a pancarditis. In adults the demonstrable percentage by careful methods is probably between 25 and 40 per cent., varying with age differences; the young adults being the more often attacked and old age being almost immune. In choreic cases endocarditis is extremely frequent judged by *post-mortem* records, many murmurs are present which may prove ultimately to be functional or accidental. Endocarditis occasionally complicates other acute infections and, rarely, mild forms may occur in general sepsis, ulcerating internal malignant growths, and *tuberculosis*.*

Any of the exanthemata and especially scarlatina and diphtheria may be complicated by it and gonorrhea in the adult is sometimes associated with the ulcerative form.

Acute rheumatism and its recurrences doubtless account for from 65 to 70 per cent. of all cases of acute endocarditis, and syphilis is now held accountable for the majority of the sclerotic cases developing in adult life and for long periods latent and undemonstrable. We now have excellent proof in the work of Poynton and Paine and E. C. Rosenow, especially, that acute rheumatism and the various forms of endocarditis are caused by various more or less specific strains of hemolytic streptococci which most frequently find their nidus in the tonsils, which when chronically infected offer an ideal point for incubation under the varied conditions of oxygen supply and nutrition which Rosenow found necessary to artificially reproduce in the test-tube in developing the different streptococcus strains.

It is impossible to clearly separate the acute benign from the acute ulcerative form save on clinical grounds and chronic endocarditis of any type may follow an acute attack and in both the acute and chronic forms, and especially in children, associated myocardial changes always must be assumed to be present.

The peculiar structure of the vegetations in verrucose endocarditis makes it possible in virulent cases for fragments to be swept away by the blood stream and produce emboli in the brain, lungs or other viscera, and this may occur in the acute and subacute forms though to a less degree than in the malignant types.

In simple verrucose cases these do not appear often and the ultimate change is one of healing and transformation of the vegetations into connective tissue with resultant deformity and disability of the valve affected.

* A low white blood count is present usually in such cases, but is not wholly distinctive, although leucocytosis is the rule in acute and malignant cases.

Varieties.—*In general there are three classes of chronic endocarditis:*

1. **Verrucose,** in which the valves are the seat of transformed vegetations.

2. **The ulcerative form,** characterized by superaddition and predominance of a rapidly extending necrotic process, present only to a slight degree in the verrucose form, the clinical symptoms of the former being largely those of acute or recurrent septic intoxication, associated with a valvular lesion and myocardial insufficiency.

3. **The sclerotic form** characterized by its slow development, its extreme chronicity and its tendency to deform gradually the valvular structures.

The previous occurrence of tonsillitis may alone explain a mysterious endo-, myo-, peri-, or, pancarditis, in children especially, for in them the actual rheumatic manifestations may be very slight. The author has seen several cases of this kind in young children in which an actual malignant endocarditis followed a tonsillitis apparently after a period of seven to twelve days of apparent health. The joints in such cases may be no more than slightly tender and any history of acute arthritis lacking.

Symptoms of Simple Endocarditis.—*Early symptoms are likely to be almost wholly subjective,* any change in the temperature being in most cases obscured by the febrile manifestations of the primary ailment.

It should be stated with emphasis that in the greater proportion of cases of endocarditis there is absolutely no complaint of cardiac symptoms on the part of the patient whose attention is wholly absorbed by the pain of arthritis or the discomfort of his sweats. A point of value.

His responses to the physician's inquiries usually furnish the only source of information with respect to the most important subjective manifestations of acute valvular involvement.

Such patients, however, often exhibit increased pallor, cyanosis, subjective weakness, precordial oppression and subjective dyspnea, the last being sometimes objective as well, but seldom marked. Important signs.

Acceleration and disturbances in cardiac rhythm occur as in myocarditis, and, perhaps, because of the concurrence of that lesion. Arrhythmia may be wholly absent or, if present, may vary from sinus irregularity of no clinical significance, to extrasystoles and varying degrees of heart block, as revealed by polygraphic or electrocardiographic tests. Arrhythmia.

The importance of an acceleration out of correspondence with the degree of fever is only less important than the development of a definite arrhythmia. And even if no murmur is present, cases that show such signs should be followed with especial care through, and for several months after, convalescence.

If the heart sounds have been carefully watched, an impurity, sharpness, or change of accent is usually detected, due probably more to the accompanying myocardial toxemia than to the valvular lesion itself, but nevertheless suggestive. Multiple systolic murmurs may be present over the heart which defy accurate classification, but usually prove to be temporary, this statement being almost wholly true of systolic murmurs maximal at the base. Precordial oppression.

In any event the sounds will have undergone a marked change since the last visit, in most instances, if in the meantime endocarditis has developed.

According to the virulence of the infection, the intensity of the reactive changes and the valve involved, the occurrence of actual organic murmurs will be early or delayed.

Mitral regurgitant lesions are by far the most common and easiest to recognize (80 per cent.).

Aortic regurgitant bruits combined with a mitral come next in frequency and may be frank or, at first, but an apparent blurring or splitting of the second sound in the third left interspace, followed later by a short diastolic bruit or the typical long-drawn murmur.

Isolated aortic regurgitation.

Aortic regurgitation occurring as an *isolated* lesion is relatively rare (3 per cent.) in acute endocarditis. It is usually a luetic lesion as ordinarily encountered in practice.

Aortic stenosis is relatively rare as a result of acute endocarditis and difficult to place during the febrile stage.

Mitral stenosis usually affords no definite signs until long after the primary and causative illness.

If the mitral valve is the seat of the endocarditis, enlargement of the right ventricle is likely to be the primary change. This is by no means an absolute rule, however, both ventricles being affected in many instances, whatever the lesion. This is doubtless in a large measure due to generalized myocardial weakness.

Silent cases.

In many of the subacute or slowly developed cases, the change may first be noticeable when a resumption of activity is attempted in convalescence.

The author knows of no means of differentiating silent cases from acute or subacute myocarditis until their silence is broken. Indeed an accompanying myocardial toxemia of greater or less degree is present undoubtedly in every case and the reader should refer to the discussion of this subject on another page.

Subacute Endocarditis.—This form presents few symptoms, other than a murmur, and fever may pass unnoticed unless it appear in an afebrile interval of the primary disease. Palpitation and inconstant symptoms of dyspnea, or more often a sense of precordial oppression, are usually present, though often overlooked.

Common and Serious Error.—The frequency with which a chronic endocarditis is found in patients whose condition is wholly unsuspected by the family physician is largely the result of three serious *errors* of omission.

One is the failure to watch the heart during all acute infections, but especially tonsillitis and rheumatism; the second is the failure to guard the patient in convalescence; the third, the almost universal neglect of the heart after such an illness is passed.

Delayed signs.

Following every case of acute rheumatism the patient's heart should be watched and examined at intervals over a period of several months. Even a mitral or aortic regurgitation *may* not become clearly recognizable for six or eight weeks after recovery and mitral stenosis is rarely demonstrable at all or even suspected during the illness. The physician will add many years to

the life of cardiopaths and save himself from legitimate criticism if he does his plain duty along these lines. Protection at the stages of early development may decidedly affect the after-course of the ailment by limiting or retarding the associated processes of myocardial degeneration.

Recurrences of Acute Simple Endocarditis.—*The liability to recurrence is decided, especially if, as is usual, these crippled hearts are not protected under proper and extended observation and judicious therapy, and the usual cryptogenetic foci (tonsils, peridental abscesses, chronically infected accessory sinuses and the like) are allowed to remain active.*

MALIGNANT ENDOCARDITIS.—In this form of endocarditis the symptoms are essentially those of (*a*) the *typhoid* state, or (*b*) of *pyemia*, and are usually definite, though masked and afebrile cases occur.

Chills or chilliness, high or moderate, intermittent or markedly irregular, fever and sweats, associated with a valvular murmur, often considerably delayed in its appearance and changing from day to day as the emboli are thrown off the affected valves or new formation occurs, are the more characteristic features, but the disease may very closely simulate typhoid fever, malaria and meningitis, and cerebral emboli may further confuse the picture.

Offers many difficulties.

Petechiæ are usually present and of the utmost significance as representing an embolic process; multiple abscess formation due to septic emboli are not uncommon; splenic tumefaction, often sudden, from infarct, may occur; gastrointestinal symptoms may be predominant, and, rarely, a septic arthritis develops.

Septic phenomena.

Petechiæ.

A systolic murmur in the tricuspid area is a prominent feature but is usually relative and secondary. Optic neuritis is a common finding in the later stages and in some instances retinal hemorrhages are demonstrable.

Diagnosis.—*The diagnosis depends largely upon the presence or the development of the signs of a valvular lesion, together with septic and usually embolic manifestations and the results of blood cultures, which are usually positive in this form. The disease in rare instances may run a prolonged fatal course with absolutely no fever.*

Fundamental factors.

The blood culture is negative for typhoid and the blood smears equally so for malaria unless the latter disease co-exists, as may happen in certain districts. The Widal reaction fails unless the patient has had typhoid within six months or a year. The leukopenia of typhoid is absent and, usually, one finds a polymorphonuclear leucocytosis of varying degree.

The pyemic form, especially when developing without a previous known infection, and in the absence of evidence of any preëxisting valvular disease, is extremely difficult to differentiate from pyemia of the common type.

Murmurs are less reliable guides in such instances than the early or even initial appearance of cardiac weakness and change in outline of the heart due to associated myocardial toxemia. The multiple cutaneous hemorrhages, due to the showers of superficial infarcts, are extremely helpful when present.

Emboli may attack every portion of the body and vary from the tiniest of terminal artery plugs, to those sufficient to block a femoral artery. The skin emboli are characterized by a pale, yellow or white center and definitely

hemorrhagic border and sometimes appear as pemphigus-like or gangrenous areas. The embolic manifestations are limited only (*a*) by the boundaries of the systemic arterial distribution, being confined to the pulmonary circuit in about 10 per cent. of all cases.

Multiple abscess formation, if it occurs in either form of malignant endocarditis, is usually the result of staphylococcic rather than the commoner streptococcic infection.

RECURRENT MALIGNANT SEPTIC ENDOCARDITIS.—*This is essentially a chronic secondary recurrent infection of the septic type, caused by the streptococcus viridans (Schottmüller), and presents the symptoms of recurrent fever of variable intensity, but of a septic type, with signs of established valvulitis and, usually, a preëxisting hypertrophy and dilatation.*

Streptococcus viridans.

It is often mistaken for malaria, the irregular estivo-autumnal forms of which it strongly resembles, both in the type of fever and in the ability of its possessor to keep about during the actual attacks, if these be mild.

Resembles malaria

In other instances it is often mistaken for tuberculosis because of accidental concurrence or the peculiarities of the fever curve.*

A common error.

As contrasted *clinically* with the simple form this malignant type may be cryptogenetic and show no history of an acute antecedent illness. Three-fourths of the cases develop upon a preëxisting valvular lesion and furnish good examples of the diminished resistance to infection associated with myocardial overstrain and diseased valves, the former undoubtedly playing the chief part.

About 90 per cent. of the cases involve the left heart alone and limit their characteristic showers of septic emboli to the systemic arteries.

It is probable that any aortic valve involvement is usually pneumococcic.

Aside from the history of previous attacks, but four features of any importance can be emphasized as aiding differential diagnosis:

1. *The presence of recognizable and usually ancient cardiac changes.*
2. *A leucocytosis during febrile periods.*
3. *The recovery of the streptococcus viridans from the blood cultures, of which several may be necessary.*
4. *The transient, red, raised, painful, cutaneous nodes seen in many, if not in most cases, on the hands and feet, and almost pathognomonic.*

Prognosis.—Few cases recover but the intermittent febrile periods may continue during many months.

MITRAL INSUFFICIENCY

(Mitral Regurgitation or Incompetence)

THE TYPICAL CASE.—The following summarizes the findings in typical *endocarditic* cases only (see "*Rationale*" for variations).

* In a case recently observed by the author the patient had a "*cor bovis*" with mitral and double aortic lesions, showed unmistakable physical signs at both apices, had recently returned from the tropics and carried the estivo-autumnal organisms. Blood cultures were positive, however, and he met his death within a few weeks. A diagnosis of tuberculosis (actually arrested) had first been made, then one of malaria, and finally the true one. His fever had been present at intervals for two years.

THE MURMUR.—Quality.—A blowing sound clearly heard but usually of only moderate intensity.

Blowing systolic murmur.

Time.—*Systolic, i.e.*, coincident with, modifying, obscuring or entirely replacing the first heart sound at the apex.*

Maximum of Audibility.—At apex of heart.

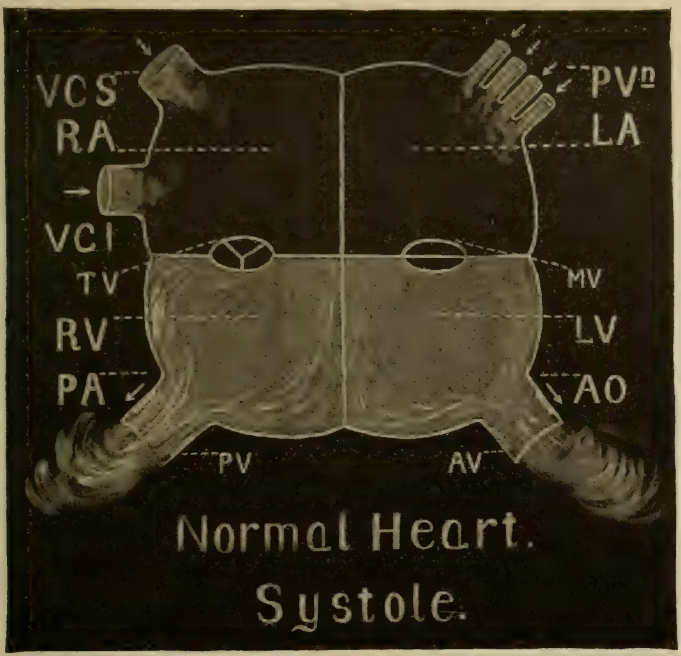


FIG. 360.—The normal heart in systole. The full ventricles are contracting, the blood flows freely from them into the pulmonary artery and aorta; the mitral and tricuspid valves are tightly closed; the auricles are refilling. MV. Mitral valve. TV. Tricuspid valve. AV. Aortic valve. PV. Pulmonary valve. LA. Left auricle. RA. Right auricle. LV. Left ventricle. RV. Right ventricle. VCS. Vena cava superior. VCI. Vena cava inferior. PVn. Pulmonary veins. AO. Aorta. PA. Pulmonary artery. (*Schematic Outline.*)

Areas of Maximum Transmission.—To and through the axilla; at the back, just within and somewhat above the inferior angle of scapula; usually not lost between these points.

Cardiac Outline.—The relative cardiac dulness is increased both to left and right and the left border line rises somewhat upward and to the left and extends outward.

Right and left heart affected.

The superficial cardiac area is significantly extended to the right and the apex-beat lies further to the left than normal and slightly or somewhat markedly lower, though never to the extent characterizing the aortic lesions.

Pulse.—The pulse is not characteristic though usually soft, *however forcible may be the apex-beat*, and it for years may show no marked variation in force or rhythm. So many factors enter into its quality as affected by the many conditions with which mitral regurgitation is associated that no one type of pulse may be regarded as peculiar to the lesion.

* Certain *bruits* closely follow the first sound and are called "postsystolic" and if further removed mesosystolic.

On the other hand, one may encounter excessive pulse-weakness during periods of serious decompensation and any form of arrhythmia may be encountered.

The auricles then show the same tendency to fibrillation or rarely flutter as is present in mitral stenosis, though in lesser degree.

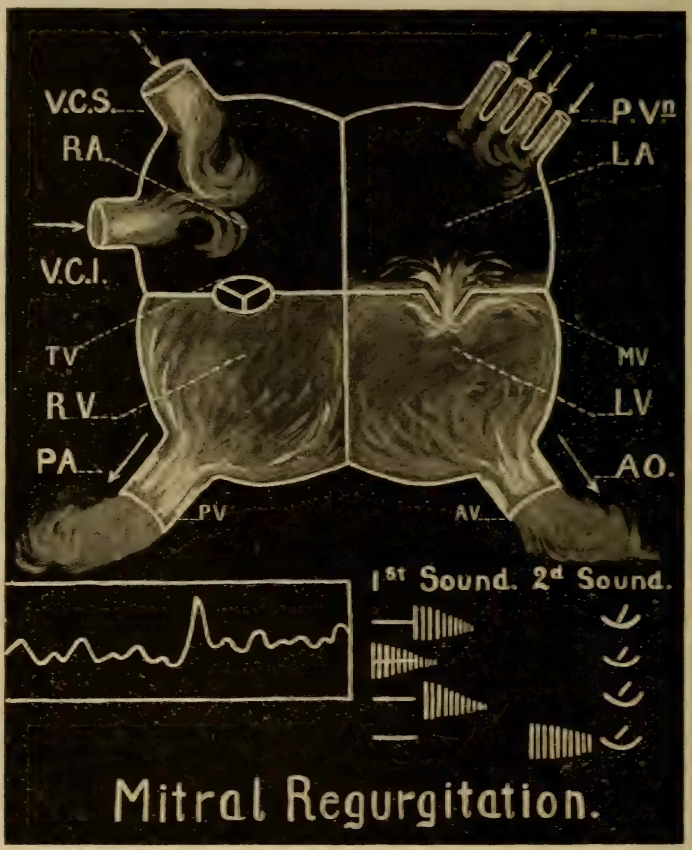


FIG. 361.—Mitral regurgitation. Four varieties of the murmur of mitral regurgitation are shown graphically, the first two of which represent the type. The heart in systole, mitral leakage evident. The contracting ventricles are forcing the blood through the open aortic and pulmonary valves; the tricuspid, tightly closed, prevents regurgitation into right auricle. The leaky mitral allows backflow into the left auricle already filling from the pulmonary veins above. *Results.*—A systolic murmur, dilatation of left auricle, pulmonary congestion, and consequent ultimate enlargement of right ventricle. MV. Mitral valve. TV. Tricuspid valve. AV. Aortic valve. PV. Pulmonary valve. LA. Left auricle. RA. Right auricle. LV. Left ventricle. RV. Right ventricle. V.C.S. Vena cava superior. V.C.I. Vena cava inferior. P.Vn. Pulmonary veins. PA. Pulmonary artery. AO. Aorta. (*Schematic outline.*)

Blood pressure varies slightly in the same manner but the tendency in compensated cases is toward a normal pressure.

Associated Signs.—*The pulmonary second sound in the second left interspace is sharply accentuated, until the right heart weakens* or actual tricuspid regurgitation is established.

Thrill.—In about 20 per cent. of well-marked and established *endocarditic* cases a systolic thrill may be palpated at the apex.

Fundamental Differential Characteristics.—*A murmur of maximal audibility at the apex, beginning accurately and exactly with or wholly replacing the first heart sound, is almost invariably due to mitral regurgitation. If the*

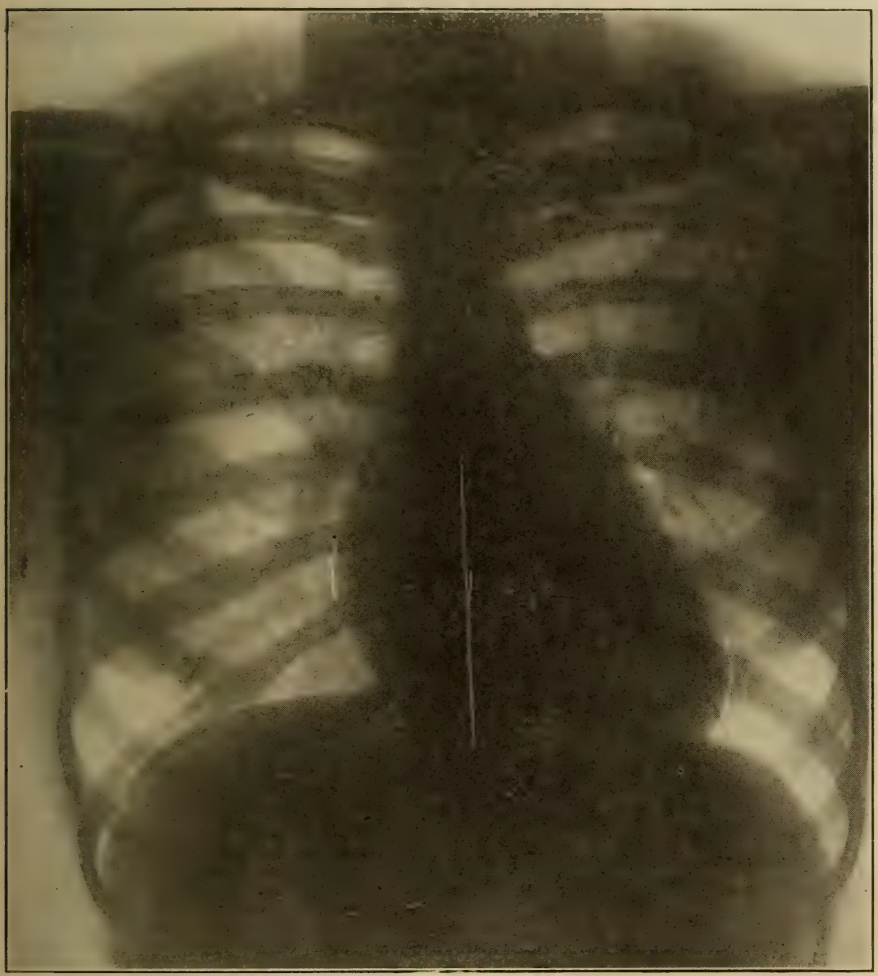


FIG. 362.—Mitral regurgitation. Note enlargement both to right and left. Measurements: M.R., 5.5; M.L., 9.5; total, 15 cm. Comparison with Fig. 308 is interesting. This picture lacks the excessive auricular bulge, although the median and inferior notches are filled out. Left ventricular hypertrophy with the mitral curve above is well shown

lesion is solitary and long-established, the cardiac area is quite characteristic and of great value in confirmation of the diagnosis.

RATIONALE.—*Mitral insufficiency is at once the most common, most tractable, and most elusive, of the commoner valvular deficiencies; easy of recognition in typical cases, but oftentimes extremely difficult to identify and properly place in others, because of the multitude of systolic murmurs of the so called*

"accidental" type and the great variations in intensity which even the endocarditic regurgitant murmur displays.

Juvenile type.

Etiology.—In the young, *when typical*, it is almost invariably (85 to 90 per cent.) the result of endocarditis resulting from acute rheumatism and in such cases is usually typical in its manifestations. Many soft, poorly-transmitted systolic or post-systolic *bruits* maximal at the apex are associated with muscle-tonus deficiency and leakage. These are not without significance or importance, though the *valves* themselves may be wholly free from disease, and are discussed fully elsewhere. (See signs and symptoms of minor myocardial insufficiency.) In older persons it is more often a manifestation of sclerosis, the relative insufficiency of myocardial degenerations, or a leakage secondary to aortic defects (relative or secondary insufficiency), or, associated with a preëxistent or coincident mitral stenosis.

Degenerative.

Secondary Effects.—In all these latter conditions it runs much less "true to form" in its symptomatic expression than in those originating in the acute infections of the first two or three decades of life, but in its typical forms it offers the simplest of problems both as to the mechanics and the secondary effects.

Relative insufficiency.

The left ventricle has one inlet and one outlet and, when acting normally, has but a single *open* orifice during each major phase of its cycle, one, the aortic, for outflow in its systole; one, the mitral, for inflow in its diastole.

It is thus protected from overdistention and embarrassment, whether from currents from two sources in its diastole or failure to deliver its full load in its systole through the one proper channel. Normally this self-protection of the ventricle itself is extended to the auricular reservoir which supplies it, and hence also to the entire pulmonary area, the right ventricle, right auricle, and the systemic circuit which these drain.

Triple effect.

It is obvious that in established gross mitral leakage the left ventricle fails to prevent backflow into the auricle during systole. As an *immediate* result, the left ventricle, left auricle, the entire pulmonary field and the right ventricle are affected.

The left ventricle, *by reason of the extraordinary and exhausting demands upon emergency reserve, almost invariably impaired by the causative toxemia or the antecedent effects of a primary myocardial degeneration or aortic valve lesion, must fail at first to deliver its full load to the aorta.*

The Left Auricle.—The auricle receives not only the flow of blood from the pulmonary veins, but also a concurrent abnormal backflow from the ventricle and must undergo primary dilatation.

Increased pulmonary pressure.

The effects of auricular engorgement are at once reflected in the pulmonary vascular field which it directly drains.

Pulmonary Circuit.—It is evident that stasis of the pulmonary circulation threatens and that the right ventricle is called upon for extra work.

Auricular dilatation and hypertrophy.

Obviously, the most serious direct primary effect is that exerted upon that thin-walled reservoir, the left auricle, and this chamber must dilate and to the best of its limited ability hypertrophy; for, like the lungs, it is

placed between two opposing forces, *i.e.*, that of systolic regurgitation from the left ventricle and that represented by an attempt at systolic compensatory overaction on the part of the right ventricle. A measure of relief must be found in an increase of rate and of cubic capacity (dilatation) on the part of

Between two fires.

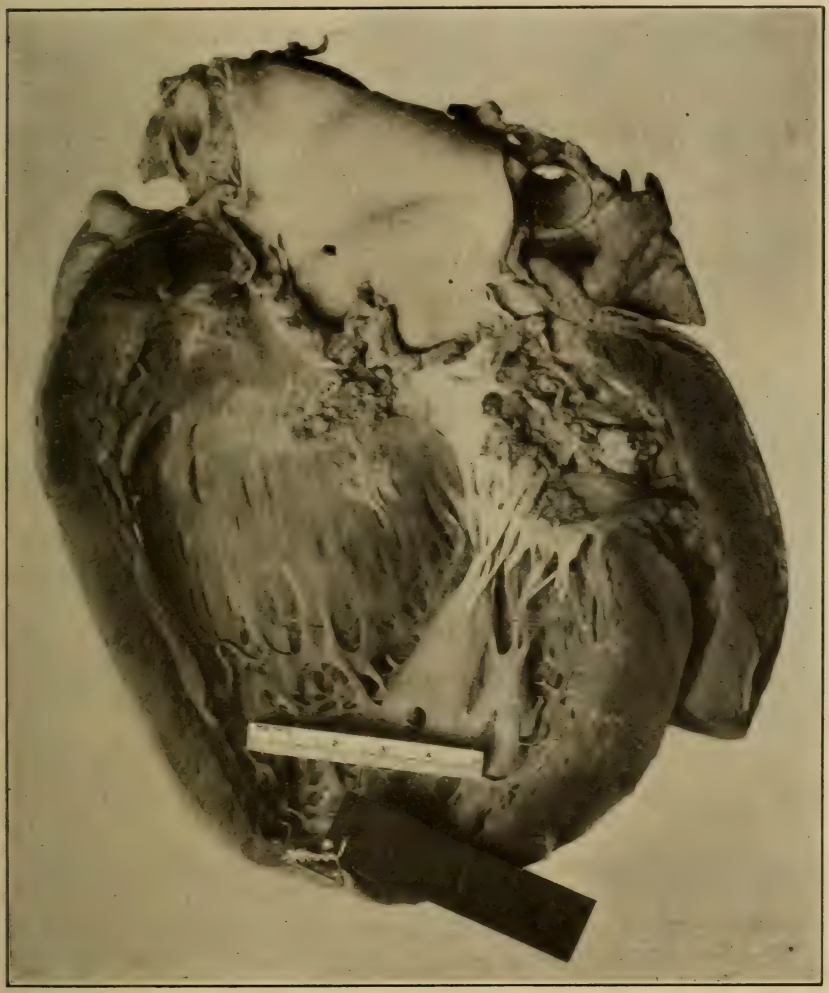


FIG. 363.—Chronic vegetative mitral and aortic endocarditis with partial destruction of leaflets, thickening and retraction of margins and consequent insufficiency and involvement of the endocardium by vegetations and fibrous nodules over an area in the left ventricle below the aortic ring. The heart is hypertrophied and the cavities dilated. Case at University Hospital (Hosp. No. 3605) of man, age 33, who had rheumatism a few years before his attack of shortness of breath. Admitted because of edema of extremities and dyspnea. At autopsy heart weighed 640 grams. Infarctions of spleen and kidneys were present. (*Dept. of Pathology, Univ. of Minn. Courtesy of H. E. Robertson.*)

the left ventricle, and this latter in turn must be limited and reënforced by hypertrophy of the musculature.

Compensation.—*Obviously the attainment of "compensation" in a typical case of free mitral regurgitation must represent* (*a*) *dilatation and hypertrophy*

Chief elements in compensation.

of the left auricle itself; (b) dilatation and hypertrophy of the left ventricle; and, (c), hypertrophy of the right ventricle, which, of the three chambers involved, is primarily least directly or violently affected because of the interposed arterio-capillary area of the lungs.

Great Variation in Lesions.—The smaller the lesion and the more gradual its production or increase, the more insignificant will be the primary changes and, as a matter of fact, true regurgitation may be either temporary or permanent, so free as to abolish the murmur in weakened hearts when the stasis pressure within the left auricle may actually block the regurgitation mechanically, or so slight or fully met by slight adaptive hypertrophy as for long periods to add little to the burden of the chambers and present almost nothing in the way of physical signs save the murmur itself.

Absent murmur.

Variations in Quality, Pitch and Duration of Murmur.—*The quality, pitch and duration of the murmur will vary with the auricular vigor and capacity and*

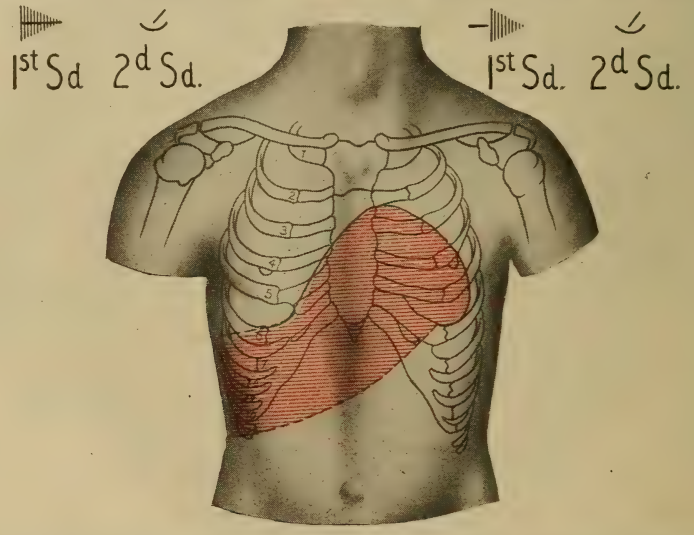


FIG. 364.—Compare this area, obtained by flat-finger percussion, with the roentgenographic silhouettes, which represent very closely the outlines obtainable by modern methods.

yet more with right and left ventricular heart strength and intraventricular head of pressure.

The size and shape of the opening, abnormalities of consistence and surface, the exact nature of the structures chiefly implicated in the valvular or myocardial disease-process, and the condition of the lungs are one and all important factors.

A few general deductions are permissible if their limitation and fallibility are recognized and held in mind.

If decided signs of stasis are absent and only the signs of a mitral leakage present, a short murmur usually indicates a small leakage; a long one a larger leakage though pitch is also a factor. In general the greater the degree of left ventricular hypertrophy, the larger the leak.

A decided heave over the epigastrium usually means a gross hyper-

trophy of the right chamber, hence a big mitral leak, a weak left heart, or both combined and its association with a pulmonary accentuation indicates good *right ventricular* strength.

Variations in Time of Murmur.—*Its time must obviously be systolic, and in typical cases, it must modify or even replace the first sound of the heart*, for it occurs at the moment when ventricular contraction "slams" shut the auricular-ventricular trapdoor and drives the blood through the open aortic orifice into the aorta itself.*

Murmurs of Relative Insufficiency.—In certain instances and especially those of relative insufficiency due to myocardial weakness and an unsupported septum and ring or to weakness of the papillary muscles, the mitral curtain seems to yield after primary closure or even late in systole and thus a proto- (early), meso- (midway), or post- (late) systolic murmur appears. These variations, especially the meso- and post-systolic variety, always suggest myocardial toxemia or an overstrained heart and demand careful investigation and especially accurate localization.

Proto-, meso- and post-systolic murmurs.

Such murmurs are likely to lack the definite transmission usually present in the case of true endocarditic mitral regurgitation.

Weakness or entire loss of the murmur may be evident in event of failure or decided weakness of the right ventricle and resulting extreme intra-auricular pressure or the pushing aside of the left apex by a dilated right heart; in the last instance it *may*, nevertheless, be heard over the back.†

Fig. 365.—Mitral regurgitation. Typical maximum intensity and transmission. (*Arthur Sansom.*)

Extreme weakness of the left ventricle and an excessively large leak constitute obvious causes.

Temporary Absences of Murmur.—The murmur sometimes varies greatly with the different heart beats. It must necessarily do so if produced at all by extrasystoles, the weaker beats of *delirium cordis* or those of decided alternation; and the same statement holds true during longer or shorter periods of actual paroxysmal tachycardia or auricular flutter (see "Arrhythmias").

Irregularly missing beats and murmurs occur in heart block when, by chance, the auricular and ventricular systoles coincide and regurgitation is minimized.

Variation in Intensity.—It is usually a soft blowing murmur but may be faint, temporarily inaudible, a mere impurity or blurring of the first sound or loud, harsh, sibilant, musical or raucous.

* At the apex, *i. e.*, in the mitral area.

† In several instances occurring in the author's series, it has been audible primarily at the back only. All have occured in slender, thin women.

Peculiarities of transmission.

Transmission of the Murmur.—Its transmission is peculiar and with respect to the apex area, to which it is too often limited and at which it is almost invariably maximal, to the author's mind is best explained by Hirschfelder's suggestion* that the murmur as heard anteriorly is usually transmitted by the chordæ tendineæ along lines represented by the papillary muscle bases. It is brought thus directly to the chest wall at the point represented by the apex-beat, which is the tip of the left ventricle save in excessive right ventricular dilatation. The murmur is also heard at the back if the left auricle is greatly or even markedly dilated and usually in the axilla, and in the case of soft *bruits* the apparent continuity of transmission through the axillary region to the inferior scapular area, is doubtless due in part to the left ventricle (axillary transmission) and in part to a dilated left auricle (scapular transmission). The latter lies almost wholly posterior, *but sometimes transmits the mitral systolic bruit to the left second interspace.*

Papillary muscle transmission

Apparent continuity of transmission.

Loud murmurs are of course heard over wide areas but the maxima are as stated.

Transmission may be markedly affected by excessive right heart dilatation which, as before stated, may actually displace the left ventricle and cut off the *anterior* field of transmission.

Transmission to back.

Cardiac Area.—The changes in the cardiac area are quite characteristic and logical, the left border being carried outward, *and the area of right heart dulness increased.* This is indicated especially by the extension of *superficial and relative dulness* to the right and only to be determined with any accuracy by the modern percussion methods and the X-ray, because of the peculiar relation of the right heart to the corresponding chest area and lung.

Associated Signs.—The associated signs are of extreme interest and importance.

Accentuation of the Pulmonary Second Sound.—That the pulmonic second sound should in most cases be louder than the aortic second or exceed its own normal intensity is self-evident, for the primary effect of the heightened pressure in the lesser (pulmonary) circulation must of course produce the conditions following: (*a*) *Left auricular overload;* (*b*) *heightened pressure in the pulmonary veins, capillaries, and pulmonary artery; and* (*c*) *overaction of the right ventricle. These combine to produce an abnormally brisk and violent closure of the pulmonic valve.*

Precisely the same accentuation of the second pulmonic tone is evident in any other case of lung stasis or circulatory obstruction unless obscured by emphysema or adhesions, and, in such instances, failing accentuation may be of diagnostic and prognostic value exactly as in the case of the heart, indicating in acute congestion or pneumonia a failing right heart or a clearing up of the lung ("resolution").

Pulmonary Stasis.—In decided mitral insufficiency during decompensatory or incompensatory periods the tendency to actual passive congestion of the lungs is strong and often finds its extreme expression in chronic bronchitis, transient congestion, hemoptysis, a blood-streaked sputum contain-

Striking symptoms.

* "Heart and Blood Vessels."

ing heart disease cells or an actual hypostatic and circulatory pneumonia or terminal edema.

In the tendency to pulmonary stasis, diminished oxygen and CO_2 exchanges and asphyxiation of the respiratory center, we find the explanation of dyspnea on exertion, cough, cyanosis and even the spasmodic dyspnea, occasionally, though not often, present in mitral hearts.

Diminished Pulmonary Accentuation.—The numerous instances in which this sharp accentuation fails are, in the main, easily understood in the light of the statement following, viz.: *First, a roomy, hypertrophied, strongly acting left auricle and left ventricle will greatly diminish over long periods of time the tendency to stasis. Second, slight leakages, or those originally small and but slowly progressive, may for a long period cause little circulatory embarrassment and few symptoms. Conceivably, overstrain of the capacity of the left heart is not always necessary to keep its chambers and those of its fellow on the right reasonably clear and unobstructed. Third, obviously, a diminution of any preëxistent accentuation of the pulmonic second sound is likely to occur in the event of excessive right heart exhaustion, free tricuspid regurgitation or the rarer event, secondary pulmonary insufficiency.*

A good sign.

A bad sign.

Subjective Symptoms.—With the slow advance of decompensatory changes and the gradual but inexorable and progressive limitation of the field of myocardial response, the extremely valuable, much-neglected and so often misinterpreted "subjective symptoms," already described elsewhere, demand recognition.

Early signs of insufficiency.

The second period of slight objective, plus more decided subjective, expressions follows and finally, the transient periods of decided objective dyspnea, pulmonary congestion, hepatic engorgement, renal congestion, and ankle edema arise and tend to become more and more frequently recurrent, resistant or persistent.

Definite evidence.

The limitations of activity may then become more and more definitely defined until general anasarca, pulmonary edema, coma, cerebral embolus, mere cardiac overload, or, as happens frequently, some intercurrent complication terminates the case.

Terminal symptoms.

It must never be forgotten that in the case of this lesion especially even the most severe symptoms may disappear under proper management.

Prognosis.—*In general*, long-enduring, lasting sometimes for a lifetime and even then surrendering oftentimes the long-poised sword to some other executioner, the tendency in mitral regurgitation is nevertheless toward a progressive diminution of cardiac "reserve" and a great reduction in the "life expectancy of its *group*.

Shortens life.

MITRAL STENOSIS

A Lesion of Extraordinary Interest and Many Peculiarities.—This common valvular disease possesses many striking peculiarities relating to its etiology, pathology, symptoms and course. It is predominatingly, though not exclusively, a lesion of the young and of the female sex, and it is almost invariably due to a distinct endocarditis of infectious origin.

Young females chiefly affected.

Follows acute endocarditis.

Usually due to rheumatism.

Nearly or quite three-fourths of all cases follow acute rheumatic arthritis and the remainder are almost entirely chargeable to other acute infections, chiefly of the youthful types. The primary arteriosclerotic form is rare; the congenital case, a clinical curiosity.

Associated frregurgitation equent.

Associations.—The stenotic lesion is seldom unassociated with regurgitation but, when advanced, usually overshadows and dominates its associate, more or less completely, in symptoms and physical signs alike.

Duroziez's disease.

Seldom Isolated.—Rarely it occurs as an isolated lesion and then chiefly in persons bearing the stigmata of "universal congenital asthenia,"* in whom its manifestations are so peculiar as apparently to have mislead Duroziez into describing it as a distinct ailment (Duroziez's disease).

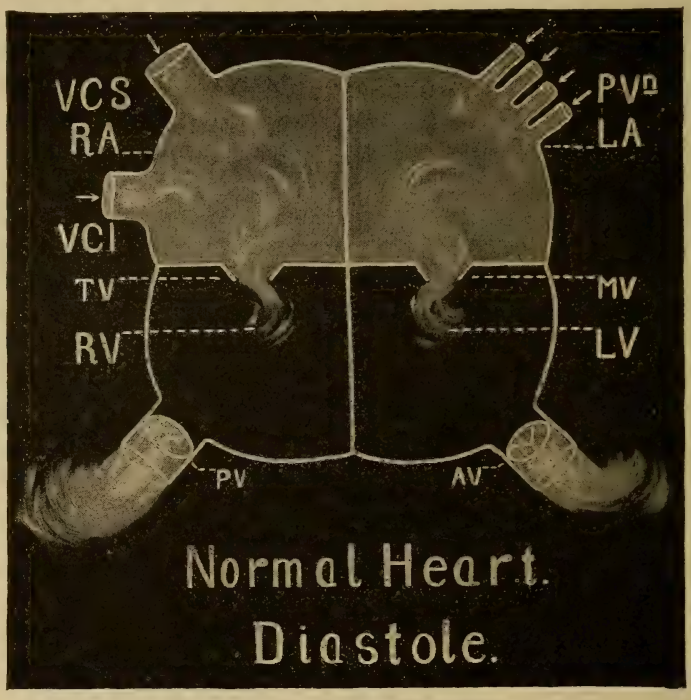


Fig. 366.—Normal heart at the beginning of diastole.

Rarely or never recognizable at onset.

Delayed Manifestations.—Existing alone it is seldom or never recognizable in its true form during the attack of endocarditis which causes it and, aside from marked irregularities in rhythm, *as an isolated lesion* its *decompensatory* manifestations may not appear in any marked degree until the valve has lost two-thirds of its normal caliber. *Decided physical signs may be manifest at a much earlier period.*

Decompensation long delayed.

In almost every case an associated mitral regurgitation or, much less commonly, an aortic leakage hastens the decompensatory stages.

Persistently Progressive.—*Its unremitting course once established, stenosi of the mitral valve almost invariably is slowly but persistently and inexorably*

* According to the author's personal observations.

*progressive though its victims may live one, two or even three decades after its inception.**

Anatomic Types.—Post-mortem, the disease presents two distinct types, viz.: *First*, the common "button-hole mitral" in which the cusps appear short, thick and adherent, and by reason of slowly progressive chronic inflammation and calcareous deposit, often form a solid mass surrounding the slit-like

Button-hole mitral.

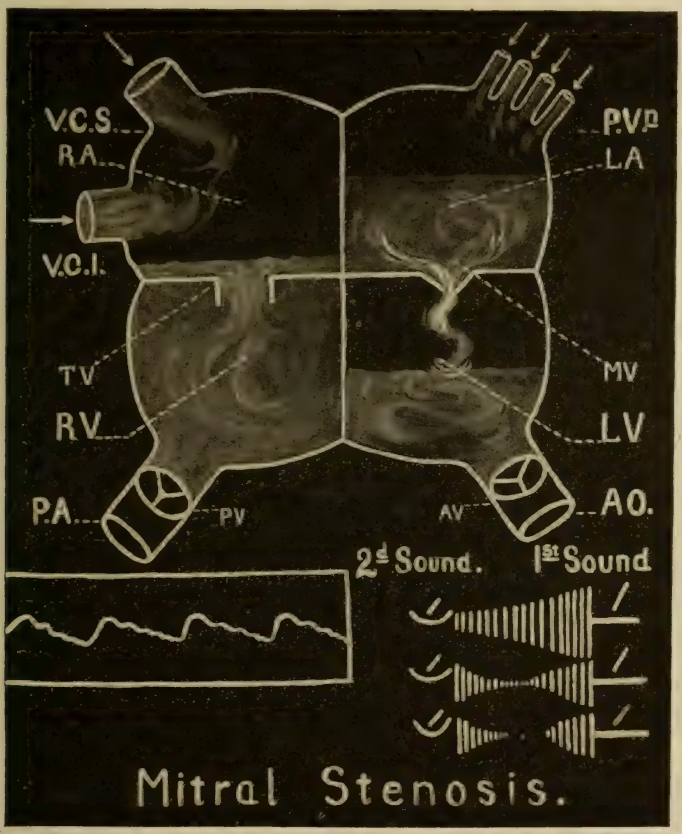


FIG. 367.—Graphic representation of three varieties of the murmur of mitral obstruction. Heart at moment of auricular contraction immediately before systole (presystole); mitral obstruction evident; aortic and pulmonary valves closed; tricuspid freely opened; *right* auricle nearly empty; right ventricle filled; *left auricle but partly emptied; left ventricle barely half full. Result.*—Presystolic or diastolic murmur, dilatation of left auricle, congestion of lungs, consecutive enlargement of right heart. MV. Mitral valve. TV. Tricuspid valve. AV. Aortic valve. PV. Pulmonary valve. LA. Left auricle. RA. Right auricle. LV. Left ventricle. RV. Right ventricle. V.C.S. Vena cava superior. V.C.I. Vena cava inferior. P.Vn. Pulmonary veins. P.A. Pulmonary artery. AO. Aorta.

opening. *Second*, the "funnel form" which, resulting from less violent and destructive inflammation and less marked degenerative processes, shows adhesions of the valve segments at their borders, the body of the cusp remaining

The funnel form

* It is quite possible that the radical treatment of concealed septic foci now so much in evidence may cause us to admit exceptions to the rule herein stated, with respect to the inexorable nature of the *anatomic lesion* itself.

comparatively unaffected. This results in a peculiar funnel-shaped opening and is preëminently the form encountered in cases of the type described by Duroziez.

Two phases.

THE TYPICAL CASE.—Two distinct phases of this lesion are encountered clinically and this fact necessitates a division of the murmurs commonly heard into two chief types.

Murmurs.—*First,* **the diastolic-presystolic bruit** *occurring exactly with or immediately following, the second sound of the heart, diminishing in intensity or entirely lost in mid-diastole, but recurring with increasing intensity just before the first sound of the heart, which seems to terminate it abruptly.*

FIG. 368.—Chronic mitral and tricuspid endocarditis viewed from the auricular surfaces. The mitral opening on the left is further reduced by soft grayish-red vegetations. (Each opening measures 4.5 cm. in circumference, a reduction of about 59 per cent. in the case of the mitral and 64 per cent. in the case of the tricuspid.) Both valve curtains are thickened, practically immobile and their chordæ almost obliterated by shortening, the papillary muscles being attached almost directly to the margins. (*Dept. of Pathology, Univ. of Minnesota. Courtesy of H. E. Robertson.*)

This represents the murmur as it is heard in cases possessing an auricle capable of presystolic contraction and is that commonly encountered in compensated mitral stenosis.

Second, **a pure diastolic form commonly associated with incompensated lesion and lacking wholly the presystolic crescendo** of auricular systole by reason of established fibrillation of the muscle of the auricle.

Quality of the Murmur.—*It is described variously as "harsh," "blubbering," "rumbling," "purring," "thrilling"* or *"vibratory," the three latter terms applying best to the typical presystolic element associated with an actively contracting auricle.*

Characteristic First Sound at Apex.—*The peculiar, loud, sharp, slamming quality of the first heart tone as heard over and near the apex is peculiarly characteristic and usually maintains its unique quality even if the auricle is in fibrillation and the typical presystolic murmur absent in consequence.*

A constant feature.

This sound abruptly terminates any mitral, presystolic bruit and the apex beat yields to the palpating finger and sometimes to the eye, the same sharp, short impact that is revealed to the ear by auscultation.

Fig. 369.—Chronic mitral endocarditis with recent vegetations on the line of closure. (The circumference of the narrowed opening measures 6.5 cm., a reduction of about 40 per cent.) The thickened chordæ tendineæ are not much shortened. The valve curtain is very thick and before section showed a "fish-mouth" opening. The muscle wall of the left ventricle and the papillary muscles are hypertrophied. Tricuspid valve irregularly thickened and insufficient. Case of (J. N. at University Hospital, No. 989) man, age 51, who had rheumatism and epilepsy at varying intervals over a period of 16 years. At post-mortem, heart weighed 573 grams. The aortic leaflets showed chronic changes and fresh vegetations. The right lung showed multiple infarctions. There was also infarction of left kidney. (*Dept. of Pathology, Univ. of Minn. Courtesy of H. E. Robertson, Director.*)

Thrill.—*A presystolic thrill (resembling that felt by the palpating fingers when laid upon the chest of a purring cat) when present at or near the heart apex is practically pathognomonic of mitral stenosis.*

Peculiar variation.

It is most marked, and often of an exquisite quality and distinctness in most of the cases carrying a well-defined presystolic bruit, less perfect or wholly absent in the instances of auricular fibrillation, at times present when no bruit can be heard, and requiring for its best appreciation a somewhat delicate light palpation.

Furthermore, it is extremely limited in its area of diffusion and easily overlooked.

The Pulmonary Second Sound.—The second heart tone as heard in the second left intercostal space is markedly *accentuated*, but this increase of intensity may be diminished or absent if secondary dilatation of the right heart and associated myocardial weakness become extreme or a secondary or co-existent tricuspid leakage is present.

FIG. 370.—Mitral stenosis with tricuspid involvement. Mural thrombus in *right auricular* appendage. The mass of adherent blood clot appears on the right side and opposite the prominent fossa ovalis. *The tricuspid valve* curtain is irregularly thickened and increased in width. (*Dept. of Pathology, Univ. of Minn. Courtesy of H. E. Robertson, Director.*)

Limited area of audibility.

Area of Maximum Audibility and Transmission.—Mitral stenosis murmurs are heard and its thrills are palpable over only a small area the size of a silver dollar lying at, near, or just within, the apex-beat of the heart in most instances. Transmission is quite as circumscribed save in rare instances when it may be diffused for a short distance into the axilla and very slightly to the right.

Easily obliterated.

Caution.—The utmost care must be observed in connection with the auscultation of this murmur to make no more pressure upon the chest than is necessary to secure complete contact of the rim of the stethoscopic chest

piece. Even a typical murmur may be rendered atypical or wholly lost if heavy pressure be applied.*

Effect of Posture and Exertion.—As a rule both the systolic and presystolic elements of this murmur are intensified when the patient assumes the sitting posture or undertakes some slight exertion, yet it is occasionally better marked in recumbency or, more frequently, in the left lateral decubitus and serves well to emphasize the importance and value of changes of posture in the elicitation of obscure baffling bruits. Useful maneuvers.

Changes in the Cardiac Outline.—Obviously, these must be chiefly those due to enlargement of the left auricle and consecutive increase in the size of the right ventricle and ultimately perhaps the right auricle as well. *In* Left auricle and right heart.

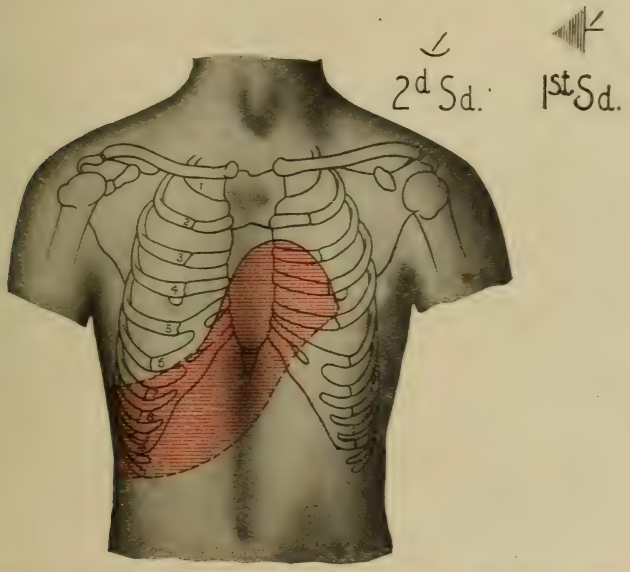


FIG. 371.—Mitral stenosis. Flat-finger percussion area of pure mitral stenosis (type of Duroziez). The left ventricle is not enlarged, but the region of the pulmonary artery, conus and left auricle is extended outward and upward, and the right heart has undergone enlargement both to right and left, indicating a lesion of considerable duration. This is further shown by the enlarged hepatic area, which indicates more or less persistent decompensation.

isolated mitral stenotic lesions the left ventricle either remains unaffected or undergoes a certain degree of atrophy. Increase of superficial percussion dulness over the right ventricle and especially the extension of this decided cardiac dulness well to the right of the left sternal border is the most marked characteristic, and if right auricular dilatation takes place, a marked enlargement is manifest to the right of the sternum. A dilated left auricle may be detected by its shadow in a radiograph or by fluoroscopy but as its position is almost wholly posterior it yields ordinarily little evidence to percussion.

* Disregard of this precaution is extremely common in the case of students, and unless one's ear is trained to an appreciation of the peculiar, characteristic, almost pathognomonic quality of the first tone at the apex, also much modified by excessive pressure, he is likely to miss many mitral stenosis murmurs.

Seldom isolated.

Associated Bruits.—Few cases of mitral stenosis *are* absolutely pure, an overwhelming majority representing combined mitral stenosis and regurgitation. *In such instances either the systolic or diastolic murmur may dominate the auscultatory picture.* Associated aortic lesions are far from uncommon.

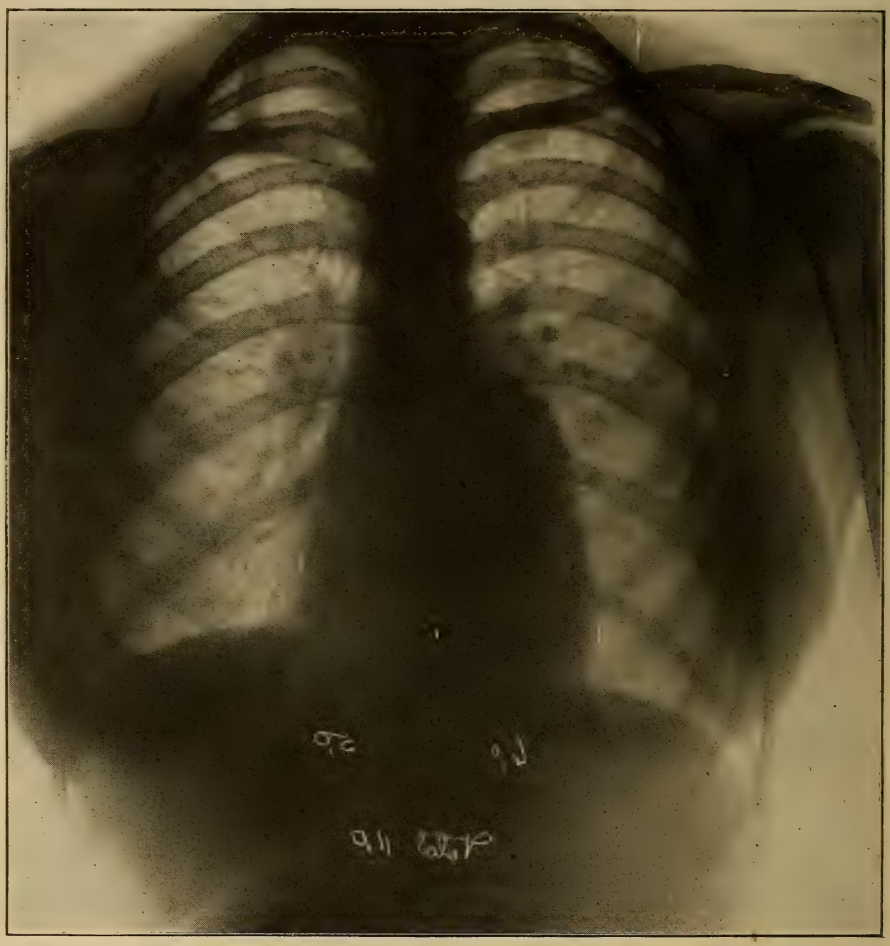


FIG. 372.—Mitral stenosis. The heart of a girl of 19 carrying a pure mitral stenosis. The lesion probably dates from childhood and several periods of definite, moderate, decompensation have been survived. Compensation good when plate was made. Evidence of great left auricular enlargement appears on the plate and in the relatively faint, yet distinct, *atypical transmission of a presystolic murmur to the back* with one break at the posterior axillary line. The left ventricle is small as is the aorta. The right is enlarged, yielding a M.R. semi-diameter of 5 cm. as against 6 only, for the M.L. semi-diameter. Total transverse measurement but 11 cm. The case is one illustrating mitral stenosis in a "drop" heart. Gastroptosis of moderate degree is present and signs of tuberculosis are visible in the original plate, although no activity is clinically manifest.

RATIONALE.—The Murmur of Mitral Stenosis.—*This is one of the most shifty and variable of heart bruits, being readily obliterated or obscured by stethoscopic pressure and markedly affected by the patient's posture, capable of presenting three forms of murmur within the diastolic period and of shifting from*

one to another even during an examination because of variations in the contractile power of the left auricle.

Both murmur and thrill are simulated closely by a rapidly beating "drop" heart.

In slight lesions especially the presystolic element may be wholly absent even with no fibrillation present and the proto-diastolic extremely faint.

Having the patient sit or stand or make almost any form of physical effort increases ventricular activity and increases that contraction of the papillary muscles and the sphincter muscle band which just precedes or really initiates ventricular systole. This increase occurs at the time that the presystolic drive of the auricle propels the last of the blood through the mitral orifice and is probably responsible for the abrupt terminal crescendo of the presystolic murmur.

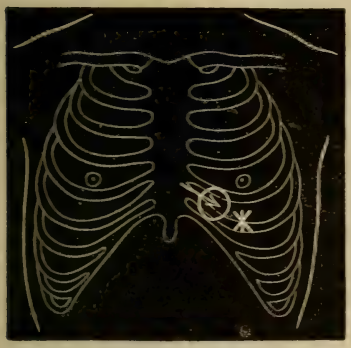


FIG. 373.—Mitral Stenosis. Point of maximum intensity and transmission in pure mitral stenosis. The murmurs may be heard quite clearly at the left back and outward from the apex beat into mid-axilla in certain unusual instances, if the murmur is loud and the left auricle greatly enlarged. Even these do not often carry completely through the axillary space but suffer loss of audibility at the posterior axilla. (*Sansom-Bramwell.*)

FIG. 374.—Mitral Stenosis *Upper shading.*—Area of audibility of simulated doubling of second sound. *Lower shading.*—Unusual area of murmur transmission. See author's note under Fig. 308. (*Sansom-Bramwell.*)

The murmur is at times *completely lacking*, yet the presence of the lesion is often definitely suggested in silent cases by the peculiar accent and quality of the first mitral tone alone.

It has been suggested that inhalations of nitrite of amyl may be used to develop a latent concealed bruit of mitral stenosis and the procedure is not without value if carefully applied.

The mode of production, shifting quality and varying maximal phases of the murmur of mitral stenosis rest upon a simple and logical basis.

With the second sound of the heart, initiating the period of diastole, the mitral valves open, and instantly the overfilled dilated auricle seeks relief from increasing intra-auricular pressure by pouring its blood through the opened gateway.

Audible eddies.

Whenever a narrowed mitral opening sufficiently obstructs this current from auricle to ventricle, every phase of normally silent flow becomes vibratingly or blubberingly noisy from the whorls and eddies created by the unnatural barrier.

Periods of accelerated flow.

Two chief periods of rapid flow appear, the one representing the *initial acceleration* due to the height of accumulated intra-auricular pressure diminishing as the initial pressure is lessened; the other *late diastolic*, active, expulsive and propulsive contraction of the auricle immediately before the next succeeding ventricular systolic contraction.

Maxima.

The initial rate of flow maximal at its beginning, diminishes rapidly as urgent intra-auricular pressure is relieved, and in case the overload of the dilated auricle is excessive the overflow may remain relatively but diminishingly rapid until presystolic (late diastolic) auricular contraction again suddenly accelerates it and it is abruptly shut off by the sharp, instant closure of the mitral valve.

Diastolic *diminuendo*.

Hence the *diminuendo* (first or diastolic phase of the bruit) which would be wholly absent only with failure of the right heart to drive an adequate amount of blood through the pulmonary circuit to the auricle or in cases where the contraction of the mitral orifice was insufficient to create audible eddies at the rate of flow initially achieved. Neither auricular paralysis nor fibrillation alone would completely obliterate it.

The degree of stenosis, blood volume, and pressure in the auricle, would determine the maintenance or nonmaintenance of a rate of flow sufficient to carry the murmur uninterrupted, but *diminuendo* from the second sound (initial flow) to the period of abrupt presystolic *crescendo*, intensification. If the auricle is fibrillating it is incapable of presystolic contraction and that element of the murmur disappears.

It is obvious that in relatively slight degrees of narrowing and with an actively functionating auricle, only the presystolic element may be audible.

It is evident also that inasmuch as a thrill is a *palpable vibration*, usually audible as well, its occurrence is similarly conditioned.

The speed of flow in proto-diastole is insufficient to produce the exquisite rapidly vibratory or purring sound of the typical presystolic element, but is sufficient ordinarily to yield a peculiar rumble or "blubber" whether the auricle is capable or incapable of contraction.

The tendency to undersupply of the tissues and undue slowing of the blood stream is automatically met in part by a mild vasoconstriction, hence the pulse is usually small.

Blood Pressure.—Sphygmomanometer readings may or may not show a diminished pulse pressure.

In pure mitral stenosis it is likely to be low but as such cases form the minority the pulse pressure is likely to be equalized by the mitral regurgitation and vasoconstriction.

Thrill.—A peculiarly characteristic and indeed an almost pathognomonic purring presystolic thrill, peculiarly constant in cases of presystolic *bruit* and regular rhythm, and at times, to be felt in *diastole even in the absence of the*

typical murmur is one of the most valuable and interesting of physical signs and when present, permits an almost certain diagnosis to be made by palpation alone.

We may anticipate the best thrills in cases showing the typical murmur, but it is conceivable that palpable vibration of a less typical sort may result from the proto- or even the meso-diastolic flow in fibrillation, provided that intra-auricular pressure is high, the auricle large and the stenosis decided yet not so extreme as to limit flow to the initial period, as may actually happen.

Necessary Clinical Division.—Two divisions are necessary to a clear description of the physical signs of mitral stenosis inasmuch as these vary greatly according to whether the left auricle is definitely contracting or is in a condition of mere fibrillary flickering which indicates an entire failure of orderly effective auricular systoles.

(A) Cases Lacking Excessive Pulse Rapidity and Extreme Irregularity.—*In these the pulse is either regular or, if irregular, is relatively slow or of moderate rate.*

MURMUR.—Quality and Character.—*A vibratory, thrilling, rumbling bruit; distinctly crescendo in type and ending abruptly in a much exaggerated, slamming, shock-like, first mitral tone.*

This may also take the form of a murmur, continuous or showing a meso-systolic interval, almost filling the diastolic period, commencing as a proto-diastolic diminuendo murmur and in presystole changing to a crescendo and terminating as above described.

Area of Maximum Audibility.—The entire area of audibility in mitral stenosis is usually characteristically limited to a sharply circumscribed area at, or slightly within, the apex-beat or left ventricular border, in the fourth or fifth interspace.

The maximum intensity or area of clear definition is sometimes so contracted as to be covered by the bell of an ordinary stethoscope which may cause one to overlook it entirely on a casual or hasty examination, though the character of the first sound is usually suggestive.

One occasionally hears the murmur over the epigastrium (right ventricular hypertrophy or dilatation) and sometimes, more or less distinctly, well into the axilla for some little distance; upward or outward over the left ventricular strip; or inward, for a variable distance, over the right ventricle but such transmission is seldom or never clear or distinct.

(B) Cases Characterized by Fibrillation- or Flutter-Arrhythmia.—Auricular fibrillation is characterized by an utterly disorderly rhythm associated with irregularities in the force and spacing of individual beats and, usually, but not always, a tachycardia. Any suggestion of its presence may be strengthened by the absence of the normal *presystolic* jugular wave and the presence of direct systolic jugular pulsation.

Some individuals may carry fibrillation for months or even several years.

In such cases the auricle, though it is the seat of myriad flickering con-

tractions, is little more than a tidal estuary and the right chambers and left ventricle are carrying the entire burden of helpful activity.

Murmur.—The typical presystolic thrilling vibratory murmur disappears and the blubbering diastolic murmurs may be present or the heart be silent save for the shock-like first sound.

Owing to the varying length of diastolic period and the unequal force of the diastolic contraction characterizing the rhythm of fibrillation, such bruits, if present, may vary in quality and duration from one beat to another.

A relative pulmonary insufficiency is doubtless more common than has been supposed and the rhythmic ebb and flow of a pulmonary capillary pulse has been actually demonstrated in several cases by fluoroscopic methods. Rarely, a mid-diastolic murmur only is present, crescendo in type *but failing to extend to the first heart sound.**

In such cases, as Mackenzie has shown, there is a partial heart block or actual dissociation and the auricular systole is out of its proper sequence with the ventricle. In "dissociation" such a murmur would be only occasional in the succession of ventricular beats.

Distribution of Overload.—Left auricular overload must be the first effect of mitral stenosis, but this is practically inseparable from *pulmonary stasis* and increased right ventricular output.

Thrown back,

Strain on right heart.

The dependence of the auricle upon the right heart is peculiarly great by reason of the fact that an important left ventricular function, that of preventing passive congestion and increased vascular tension in the pulmonary field, is in abeyance because of the inability of the left auricle to supply the blood. The more powerful of the two pumping chambers is therefore wholly unaffected save that actual atrophy may result.

Left auricular dilatation, fortunately slowly produced, prompt consecutive hypertrophy of its thin walls and right ventricular work-increase and hypertrophy, also gradually induced, are the only *cardiac* changes worthy of note in pure mitral stenosis until the indifferently effective compensation characterizing this lesion begins to wane. Even then the left auricle is the chief sufferer but the right heart also feels the strain and at the same period the auricle may be acting merely as a huge reservoir.†

Paralysis of the left recurrent laryngeal nerve is observed occasionally in cases carrying extreme left auricular dilatation.

* The proto-diastolic gallop rhythm, often present at the apex, represents a third sound interpolated between the second sound and the succeeding first sound, at a point represented by a time interval about equal to that elapsing between the first and second sounds, *i.e.*, it interrupts the long pause by a sound occurring in early diastole (proto-diastolic).

As has been stated elsewhere the proto-diastolic gallop or "canter" rhythm is not limited to mitral stenosis, but may also occur in persons carrying a slow pulse, but apparently sound, when they lie upon their left side. (*Thayer.*)

In the presystolic gallop or canter rhythm, the interpolated sound falls, as the name suggests, just before systole, *i.e.*, it is presystolic or post-diastolic whichever one may chose to call it. (See "Gallop Rhythm.")

† Cases have been reported in which a left auricle was capable of containing 3000 c.c. of blood.

The right ventricle is slowly yielding to strain, the pulmonary artery and conus dilatation increases, tricuspid regurgitation may follow, and even a

FIG. 375.—Dominant mitral stenosis. Imperfectly compensated. Ambulant patient The profile shows clearly the change in the mid-convexity of the left border, due to the great enlargement of the left auricle. The enlargement of the right heart is obvious; but left ventricular change is manifest. The total transverse diameter in this case was 14.75 cm. Although no mitral systolic murmur was audible, the presence of past incompetence of the valve is strongly suggested. The lungs have for years been the seat of persisting congestion intensified by the fact that cardiac reserve had been severely taxed for years by reason of the fact that the patient kept at work even when suffering from serious pulmonary symptoms of decompensation.

pulmonary leakage may add its diastolic murmur (maximal in the second left interspace) to symptoms of stasis in the systemic circulation.

Changes in the Cardiac Outline.—*The typical heart of pure compensated mitral stenosis is therefore relatively small, but when the right ventricle begins to fail and dilatation follows, it may completely overlap the unchanged or atrophic left ventricle and carry the area of superficial cardiac dulness to, or beyond the right border of the sternum. Relative (auricular*) dulness to the right may become excessive. Such typical hearts are unusual, and left ventricular hypertrophy and dilatation usually are manifest because of coexistent mitral leakage.*

Rarely typical.

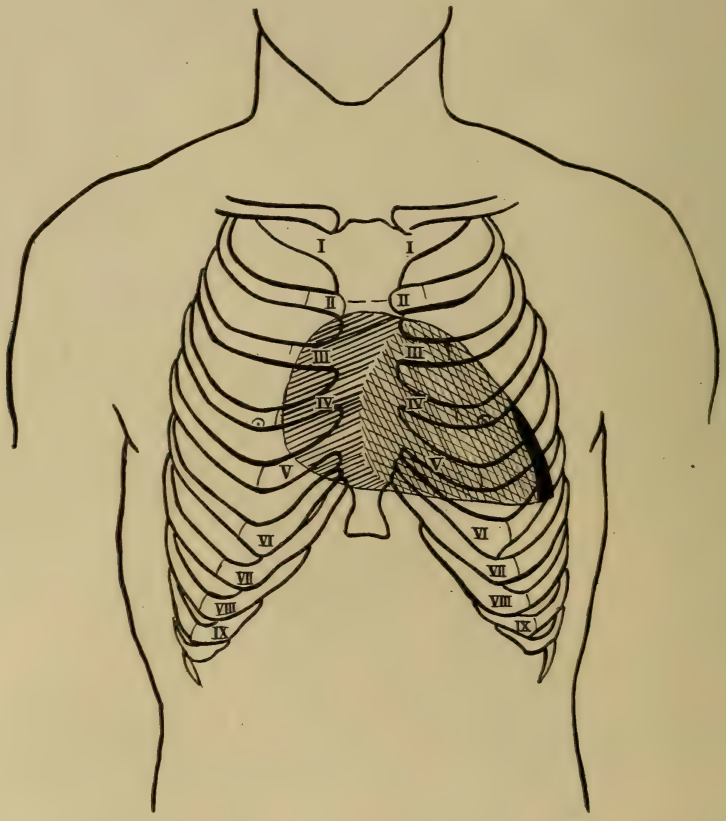


FIG. 376.—Greatly dilated right heart (auricle and ventricle). Cross-hatched area represents the right ventricle whose right border should be shown as extending obliquely downward to the right costoxiphoidal angle; black strip, the left ventricle; obliquely lined area, the right auricle. Note that practically all of the surface accessible to percussion represents the right heart chambers. Even the small strip of left ventricle here shown may be lost. (*After Mackenzie. Modified.*)

Effect upon the Heart Sounds.—The diminished load of the left ventricle tends to reduce the second aortic tone and the increased load of the right ventricle together with its added force of projection results in a more forcible closure of the pulmonary valve and accentuation of its second sound in the second left interspace, this accentuation being retained until the right

Aortic tone.

Pulmonary tone.

* In decided cases of the "drop" heart the right ventricle forms the lower right border.

heart fails, tricuspid leakage ensues or the pulmonary valve itself becomes affected.

Secondary Systemic Effects.—These are the general *subjective* and objective manifestations of cardiac insufficiency as already outlined, the first gross effects being manifested by evidences of congestion in the pulmonary circuit and chiefly shown in dyspnea, varying grades of dusky red and, later, gray cyanosis together with a tendency to passive pulmonary congestion and bronchitis. Stasis

Pain is sometimes a prominent symptom and, when the right heart weakens or the tricuspid valve yields, general stasis will occur throughout the systemic veins and involve the kidneys, liver, and gastrointestinal tract, thus leading to gravity-edema and a tendency to terminal transudations in the pleural or even in the pericardial sac.

In this lesion of maximal auricular distention and relative stasis there exists a peculiar liability to the production of clot-fragment emboli leading to infarcts involving the various organs and not infrequently producing hemiplegia or even sudden death through cerebral lodgment. Infarcts.

Not only should the lesion be recognized and some measure of control instituted before the more serious of the preterminal symptoms arise but, knowing the frequency with which mitral lesions result from acute rheumatism, the physician is not wholly guiltless who does not so follow these cases during convalescence and for several months after recovery, so as to detect not only leakage but also stenosis at its inception.

A little good advice in the beginning, and more than advice whenever the compensation weakens, would greatly prolong life.

TRICUSPID INSUFFICIENCY

(Tricuspid Regurgitation, Tricuspid Incompetence)

Etiology.—Tricuspid regurgitation almost invariably represents a *relative* insufficiency, secondary to a chronic or acute obstruction of the pulmonary circulation, whether this arises from mitral lesions, by far the commonest cause, from any of the primary myocardial degenerations, from chronic interstitial pneumonia, marked kyphotic deformities, emphysema, acute lobar pneumonia, chronic adhesive pleuritis or chronic massive pleural effusions. It is far less common as a sequence of aortic stenosis or the cardiac hypertrophy, dilatation and associated myocardial degeneration secondary to chronic interstitial nephritis, though by no means rare. Usually secondary. Diseases of the lungs. Rare.

Endocarditic Cases.—*As an immediate and direct result of acute endocarditis, affecting two or more valves, it is rare; and, occurring as an isolated primarily endocarditic lesion, is a clinical curiosity.*

As might be expected from the general character of tricuspid reflux as a lesion secondary to left heart involvement and the obvious fact that both auricles must be severely punished, the greater number of cases show the "*pulsus irregularis perpetuus*" ("delirium cordis") of auricular fibrillation. Fibrillation a common accompaniment.

Primary endocardial defects or those secondary to pulmonary disease may show no marked irregularity prior to the onset of fibrillation of the auricle, though extrasystolic intermittency *may* precede it by long periods.

CHARACTERISTIC FEATURES OF THE TYPICAL CASE.—Pulse.—As commonly encountered in cases of marked or terminal decompensation, the pulse is usually markedly irregular, with an especial tendency to *pulsus irregularis perpetuus*. Lesions undoubtedly exist that show a perfectly regular pulse and it is obvious from a consideration of the etiologic factors that one is usually dealing with the arrhythmias of the primary lesion or lesions to which tricuspid insufficiency is secondary.

Commoner type.

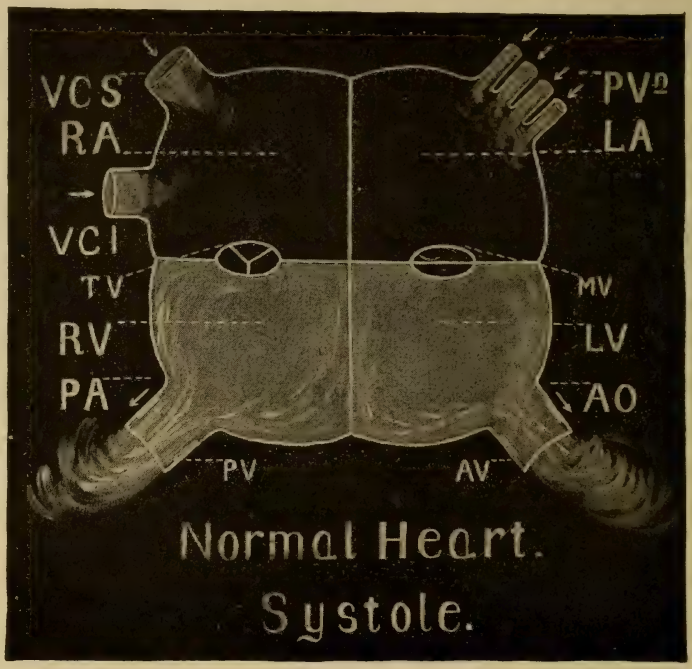


FIG. 377.—The normal heart in systole. The full ventricles are contracting, the blood flows freely from them into the pulmonary artery and aorta; the mitral and tricuspid valves are tightly closed; the auricles are refilled. MV. Mitral valve. TV. Tricuspid valve. AV. Aortic valve. PV. Pulmonary valve. LA. Left auricle. RA. right auricle. LV. Left ventricle. RV. Right ventricle. VCS. Vena cava superior. VCI. Vena cava inferier. PVn. Pulmonary veins. AO. Aorta. PA. Pulmonary artery. (*Schematic Outline.*)

MURMUR.—Quality.—Soft and usually blowing.

Time.—Definitely systolic. Coincident with the right heart first sound which it tends to modify or displace.

Area of Maximum Audibility.—It is sometimes best heard along the edge of the sternum or in the epigastrium. Slight murmurs are best heard, usually over the lower half of the sternum, but, inasmuch as the right heart presentation includes practically the whole lower substernal and left thoracic areas of the precordium, it may yield when dilated a murmur over the entire right

Lower sternum and vicinity.

Decided in advanced cases.

heart area. It is not well heard over the true heart apex, left ventricle or beyond as is the case in mitral regurgitation.

Silent leakage.

In very advanced cases with profound decompensation, extreme ventricular weakness, and a large leak, no murmur may be audible.

Associated Signs.—(*a*) *Diminution or loss of the second pulmonary tone.* (*b*) *Enlargement of the right heart area especially to the right of the sternum (auricle).** (*c*) *Cyanosis.* (*d*) *Dyspnea.* (*e*) *Direct, systolic pulsation, and*

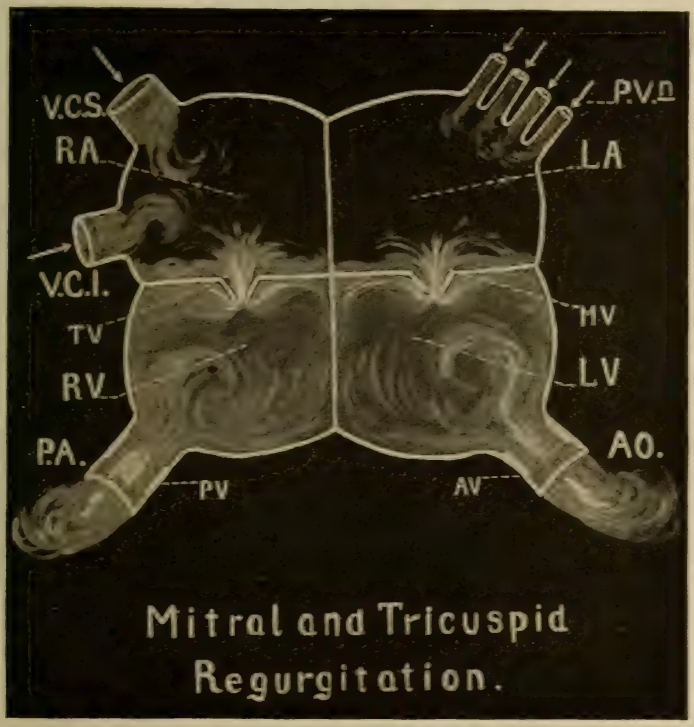


FIG. 378.—Mitral and tricuspid regurgitation. Heart in systole. Mitral and tricuspid valves both incompetent. *Result.*—Double systolic murmur, enlargement of both right and left chambers, pulsating jugulars, general venous congestion, edema, anasarca, etc. MV. Mitral valve. TV. Tricuspid valve. AV. Aortic valve. PV. Pulmonary valve. LA. Left auricle. RA. Right auricle. LV. Left ventricle. RV. Right ventricle. V.C.S. Vena cava superior. V.C.I. Vena cava inferior. P.Vn. Pulmonary veins. P.A. Pulmonary artery. AO. Aorta. Note:—Dilatation of the right heart and tricuspid leakage are much more common than was formerly supposed.

marked persistent engorgement of the external jugular vein are present in advanced cases and such persistent distention with little or no visible systolic jugular wave may be present in lesser lesions if the valves of the cervical veins are intact. Pulsation of the liver may occur, palpable or demonstrable only by instrumental means. (*f*) *Retarded circulation and a decided increase of pressure in the systemic veins leading to hepatic engorgement, edema, ascites, renal congestion.* (*g*) *Asphyxia of the medullary center.*

* In marked or extreme types of "drop" heart, the right ventricle.

RATIONALE OF TRICUSPID REGURGITATION.—The Murmur.— This is usually soft and blowing for reasons already given, but, in rare instances, may be harsh and vibrant and associated with a thrill, or, for reasons stated above, may be represented by a *mere "muffling" or impurity*

Usual murmur variations.

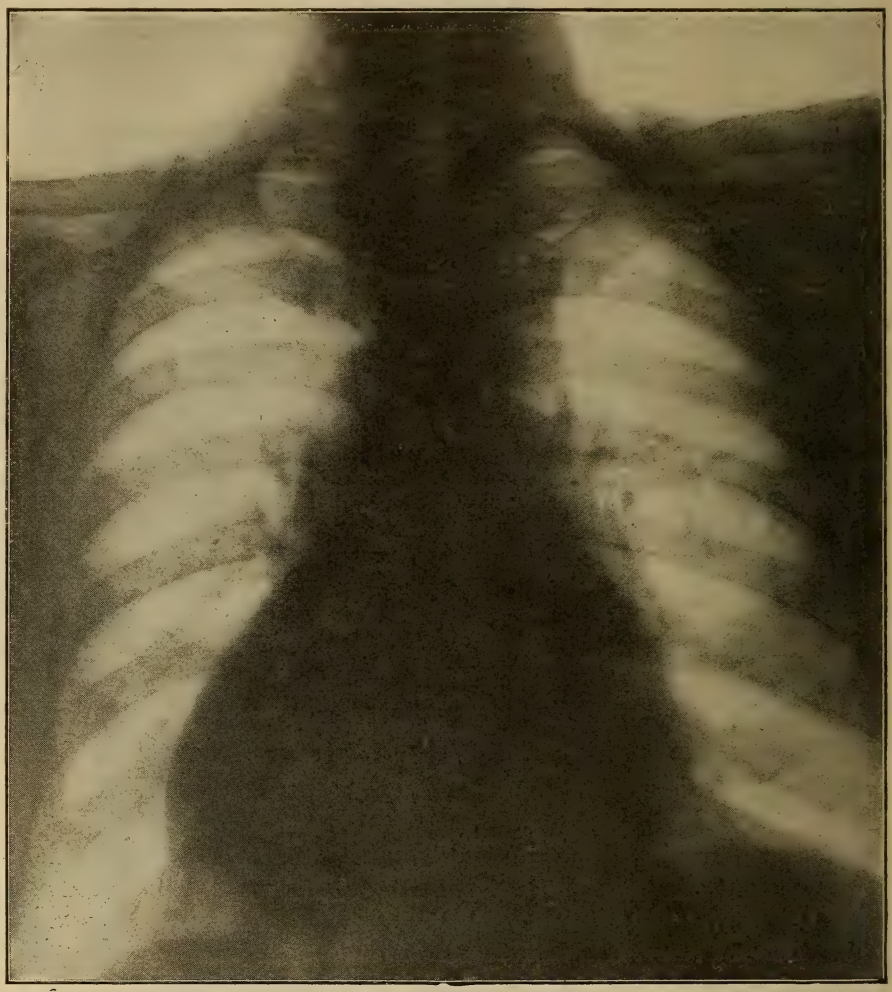


FIG. 379.—An enormous (19.8 cm.) *silent* heart of the combined mitral and tricuspid type. Disproportion between Ml. (11.3 cm.) and Mr. (8.5 cm.) is very marked. Ambulant patient. Dyspnea on exertion. Decided edema of the legs at the end of the day. Engorged firm liver. Persistent râles at lung bases. All four chambers enlarged. When the left ventricle yields in terminal decompensation, the triangular area of universal decompensation will be manifest. Heart lesion had been wholly disregarded and left untreated. Dorso-ventral aspect.

of the first tone. "Murmurish" sounds or extremely short bruits may occur in this as in any other valvular lesion.

Obviously its best conduction to the surface would be over the substernal portion of the right ventricle *to which it is conducted by the chordæ tendineæ and papillary muscles;* but logically it should be heard clearly or even maximally

Conduction.

at times to the right of the sternum over the right auricle *which receives the impact of the regurgitant current.*

The Second Pulmonary Tone.—It is evident that the pressure within the ventricle during each systole is reduced and that the load driven into the pulmonary artery will ordinarily be less in amount than is required and lacking in force of projection. As a result the pulmonary second sound (valvular closure) will be greatly diminished or even inaudible in the event of any free leakage unless this be associated with a strongly overacting right ventricle.

Effect of leakage in weakened right heart.

A markedly diminished second pulmonary tone at once suggests a failing right heart and tricuspid leakage, or a pulmonary insufficiency or stenosis,

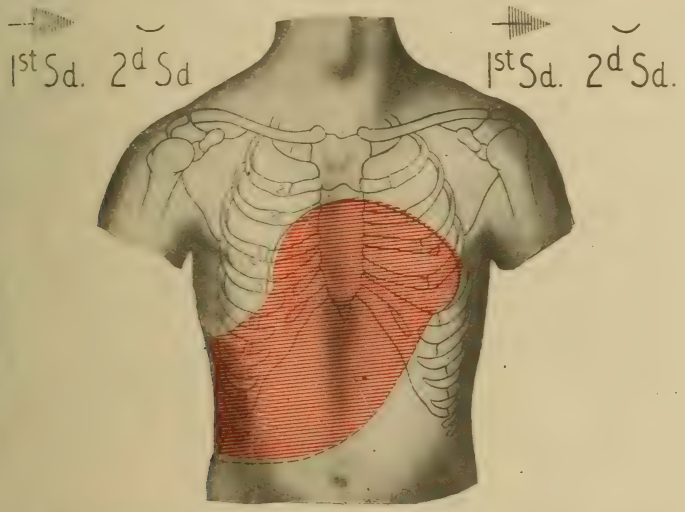


FIG. 380.—Mitral and tricuspid regurgitation. Decompensation marked. Such a heart has repeatedly been aspirated for supposed pericardial effusion, though this error is more likely to occur in a similar universal enlargement without murmurs. Bruits may or may not be very obscure in such cases as this if the valvular lesions are wholly the result of degenerative changes. (Flat-finger percussion.)

the last being excessively rare lesions, usually congenital, associated with characteristic and well-defined murmurs.

The right auricle must greatly dilate and hypertrophy in tricuspid regurgitation though to a somewhat less degree than is the case with the left auricle in mitral stenosis, in that the incoming systemic venous currents lack the persistent and primarily inexorable drive maintained by the right ventricle against the crippled left auricle in the case of mitral lesions and also because the right auricular overload, in a considerable measure and quite readily, is passed backward to the great veins (superior and inferior vena cava), thus raising and rendering strikingly intermittent the venous pressure and flow and engorging the viscera of the systemic circuit.

Throwing the load backwards.

Visceral stasis.

Venous Stasis.—*From this partial circulatory block on the venous side results the chronic passive congestion of the kidneys and the engorgement and*

Lungs, kidneys and liver.

enlargement of the liver, often associated with a feeling of excessive epigastric fullness, tension or actual pain because of the stretching of its capsule.

Stomach and intestines.

Gastro-intestinal disorders of like causation are common; edema, ascites and in terminal cases, effusions of liquid transudate into the pleural or pericardial sacs occur and the patient's general nutrition suffers greatly.

Anasarca.

Cerebral symptoms.

The brain likewise feels the stasis, and drowsiness, somnolence, stupor, or a terminal coma may be manifest.

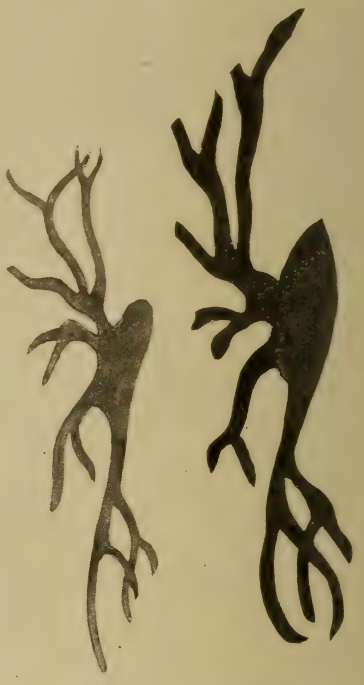
A Vicious Circle.—One of the most vicious of circles arises from asphyxia of the vasomotor center which raises pressure in the systemic arteries, adds to the burden of the left heart and greatly increases the weakness of the general circulation.

Furthermore, there is a constant shortage in the blood supply throughout the nutritional and ventilation areas represented by the systemic and pulmonary capillary deltas respectively.

Dyspnea and cyanosis are alike marked and the former tends to assume one of its most extreme forms in terminal decompensation associated with this lesion.

Epigastric pulsation due to an enlarged *right ventricle* is often evident, and at such times especially, the murmur may be clearly audible over that area. Such a rhythmic rise and fall of the epigastrium may be confused with *true expansile pulsation of the liver*, if mere anterior palpation is employed and the antero-posterior bimanual method neglected.

The epigastric pulsation is usually somewhat wavy and less determinate than the heave of a greatly enlarged *left ventricle*, as seen over the left thorax in aortic lesions.



Diastole. Systole.

FIG. 381.—Phantom-pulse in pulmonary veins. This fluoroscopic phenomenon is occasionally demonstrable, *under a special technic*, in mitral cases with tricuspid insufficiency, showing the positive (systolic) jugular pulse, of which this hilus flow and ebb is the pulmonary counterpart. (*Schematic; after Schwarz.*)

Variations in hepatic outline corresponding to the degrees of stasis are extremely marked, jaundice is common and nutmeg liver is often encountered.

Paradoxical rhythmic retraction.

Systolic retraction of the lower interspaces to the left of the sternum is usually a marked feature, attributable to the systolic retraction of the anterior wall of the enlarged right ventricle, and in mitral stenosis especially, or cases of *primary* right ventricular hypertrophy and dilatation, the true systolic *visible* apex impulse of the left ventricle may be submerged, owing to the inability of the left chamber to reach the thoracic wall.

Combined type

In such instances the wavy, diffused indeterminate right ventricular impulse is characteristic. In combined mitral tricuspid and aortic lesions a

curious combination of presystolic undulations over the right ventricle and systolic *heave* over the left may be encountered.

Slight or Silent Leakages.—*It must be remembered that slight leakages may be evident in the murmur only, all secondary signs failing, and that either slight or large leakages may be wholly silent for the reasons hitherto assigned.*

Only a small percentage of the total tricuspid insufficiencies present during life are recognized either ante- or post-mortem, though a more careful study of the venous pulse and second pulmonary tone would uncover many at present overlooked.

In cases of long standing this lesion is associated with a significant and characteristic permanent dilatation of the cervical veins.

Direct Expansile Hepatic Pulsation.—The direct systolic pulse is, of course, registered over the internal jugular and the bulb and over the enlarged liver, which may be often felt to expand following each systole when firmly compressed front and rear by the two hands. Jugular pulse.

In well-established cases, moreover, i the liver be forcibly compressed when so held, marked jugular distention and increase of cyanosis are usually demonstrable.

Significance of the Systolic Jugular Pulse.—*A direct systolic venous pulse indicates one of four conditions, viz.: ventricular extrasystoles, tricuspid regurgitation, auricular fibrillation, or, certain cases of dissociated rhythm in complete heart block, in which there is an accidental concurrence of certain auricular and ventricular contractions.* Four chief causes.

Important Deduction.—*If decided enlargement of the veins co-exists with a marked systolic pulsation, a lesion of the tricuspid valve is distinctly indicated, whatever the rhythm of the heart, and in cases presenting either auricular fibrillation or ventricular extrasystoles, a diminished second pulmonary tone or one disproportionate to any existing pathologic conditions adequate to produce marked stasis in the pulmonary circuit, indicates a leakage of the tricuspid valve.*

In marked instances, moreover, the systolic tricuspid jugular pulse is more like an arterial pulse than the gentle filling and collapse or the flowing wave of its simulators and engorgement or enlargement of the cervical veins is associated usually with general and decided venous stasis.

In advanced cases carrying this lesion a persistent distention of the veins and their prompt refilling when compressed above and stripped of blood is marked and characteristic.

Extreme Examples.—*In some cases of especially free regurgitation the systolic venous distention may be visible not only in the neck but in the arms, back of the hands and even in any existing varicosities of the veins of the thigh or leg (D. Gerhardt).* Diffused venous pulsation.

*In these cases the pulsation represents an actual systolic rhythmic recession and advance of the venous flow as may be readily demonstrated by compression.**

Over the jugular bulb and crural space when the valves are still intact, Systolic venous tone or bruit.

* In all marked cases the degree of venous pressure increase is marked. Gerhardt reports a case in which a weight of 300 gm. was upborne by the veins.

one may sometimes hear a distinct thud in systole and over the same areas, when the valves have yielded, a distinct systolic *bruit*.

In the excessively rare instances of isolated primary endocarditic tricuspid regurgitation, the left ventricle may remain normal or may undergo actual atrophy (Stadler).

AORTIC INSUFFICIENCY

("Aortic Regurgitation," "Aortic Incompetence")

Characteristic Signs in Cases, Commonly Encountered.—The following summary applies to the usual cases only (see "Rationale" for chief variants).

Hissing or blowing.

The Murmur.—Quality.—*A blowing, hissing, or rasping sound, of extremely variable intensity, loudest at its beginning and waning with the continuance of the diastole, i.e., a diminuendo murmur. It may be very clear and distinct, as soft as the faintest pulmonary vesicular murmur, or wholly absent.*

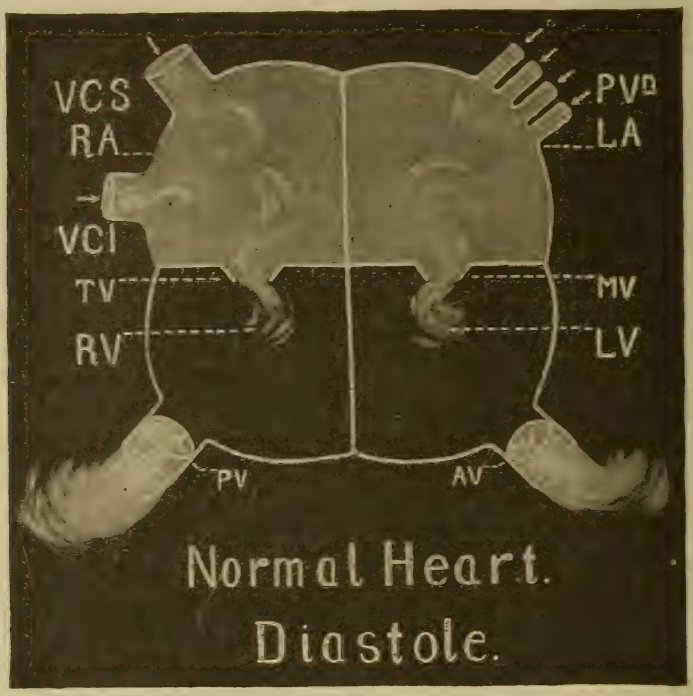


FIG. 382.—Normal heart at beginning of diastole.

Diastolic.

Time.—*Distinctly diastolic; beginning at the exact time that the second sound ends, with no detectable break, yet often tending, apparently, to obscure, modify, or displace the second sound of the heart in the aortic area, first right interspace, right half of the manubrium sterni and the carotid artery, if this second sound be shortened, diminished, or, wholly lost, by reason of the peculiarities of valvular defects.*

It is usually accompanied by a *systolic* murmur, maximal in the second

right interspace near the sternum or one interspace higher, and transmitted upward into the carotids. In the case of loud murmurs this makes the two sounds resemble more or less closely the "to-and-fro" sound of the wood saw ("see-saw" murmur).

Alternative Areas of Maximal Audibility.—*These lie along a line joining the chief points of maximal "murmur transmission," viz., (a) the second right inter-*

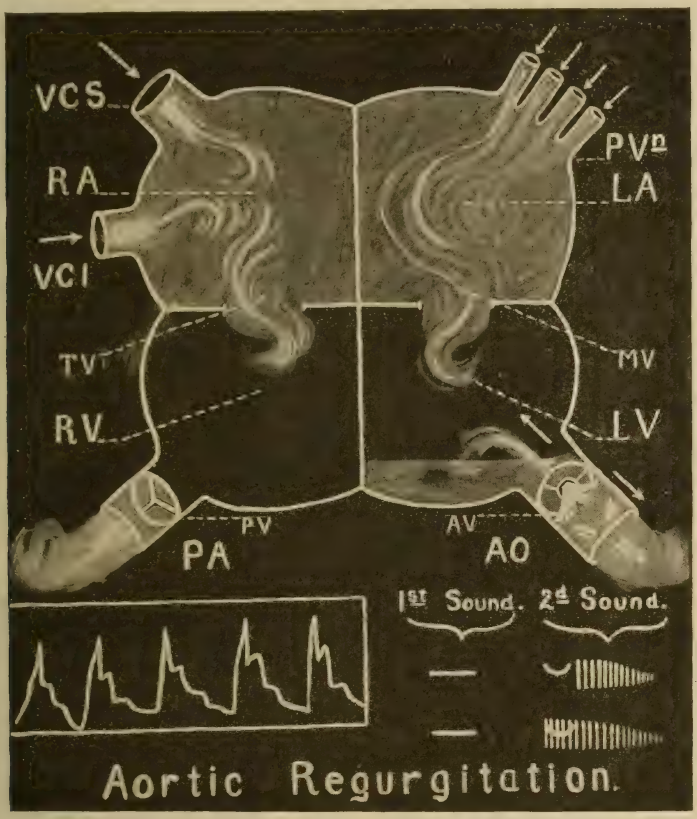


FIG. 383.—Graphic representation of murmur. Two varieties of aortic diastolic murmur. (With sphygmogram.) The heart is shown in diastole, aortic leakage being evident. The blood has just been projected into the aorta and pulmonary artery by the ventricular contraction. The pulmonary valve tightly closed maintains the blood column, but through the leaky aortic valve a regurgitant current meets the stream descending from above through the open mitral valve. *Results.*—A diastolic murmur, dilatation and hypertrophy of left ventricle, a slapping, low-tension pulse. MV. Mitral valve. TV. Tricuspid valve. AV. Aortic valve. PV. Pulmonary valve. LA. Left auricle. RA. Right auricle. LV. Left ventricle. RV. Right ventricle. VCS. Vena cava superior. VCI. Vena cava inferior. PVn. Pulmonary veins. PA Pulmonary artery. AO. Aorta.

space at the edge of the sternum; (b) the third left interspace or, (c) midway on a line joining these points; (d) at the ensiform cartilage.

The point of maximal audibility is found, therefore, usually, over or along the line of the regurgitant stream whose eddies and whorls generate the sound, a wholly logical and simple distribution.

The point of preference is usually the third left interspace at or just to the

left of the sternal margin; not over the valve above or in the second right interspace, which with the first right interspace constitute the points of election for the auscultation of the *second sound* itself.

Connects areas of maximum audibility.

Area of Maximal Transmission.—*These are the points mentioned above, the intermediate area, and along the left edge of the sternum to the ensiform. It is often especially marked along left sternal border. Oftentimes it is faintly or distinctly heard in the carotid and subclavian arteries and at the apex of the heart, though audible faintly, or not at all, over the intermediate right ventricular area, save in the case of loud and widely distributed murmurs.*

In rare instances the murmur may be audible or even *maximal* in the left axilla. (R. Cole).

Increased to left and downward.

Cardiac Outline.—*It is that of left ventricular dilatation in recent endocarditic cases, but as encountered commonly, represents combined hypertrophy and dilatation of the left ventricle. The left border is extended, therefore, outward and sharply downward.* (See "Roentgenography.")

The apex beat is lifting or heaving in all free and long established cases, the force and sustained strength of the left ventricle varying with the dominance and degree of hypertrophy as affected by the tendency to gradually induced ascendency of dilatation, and the latter is often rendered obvious by the less determinate character of the prominently visible impulse.

Slight dulness may be noted over the second right interspace if, as commonly happens in old cases, the aortic arch is more or less decidedly dilated.

Visible *pulsus celer.*

Associated Signs.—(*a*) *Visible, jerking, throbbing, pulsation of the arteries of the superficies, especially marked in the carotids, brachials, radials and temporals, but extending to the lesser branches.*

Corrigan pulse.

(*b*) *A radial pulse of unsustained initial high pressure, sharp and shock-like, the "Corrigan" or "water-hammer pulse."*

Capillary pulse.

(*c*) *A visible or demonstrable "to-and-fro," "ebbing and flowing" wave of color, "the capillary pulse," which represents the rhythmic alternation of high and low arterial pressure in the relaxed vessels. It is visible on the lips and nails or over and at the edges of a fading erythema or any flushed area, whether natural or artificially induced for the purpose of examination.*

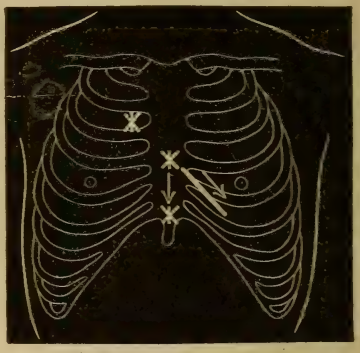


FIG. 384.—Usual maxima of audibility and lines of transmission of *bruit* of aortic regurgitation.

"Pistol-shot" sound.

(*d*) *A "pistol-shot sound" in the femorals, i.e., a sharp, shock-like sound and palpable impact, sometimes double, often associated with a systolic bruit, and sometimes with a double ("to-and-fro") murmur ("Alvarenga-Duroziez") under moderate stethoscopic pressure.*

The Heart Tones.—*The character of the aortic second sound itself varies under the influence of many factors—such as the freedom of regurgitation, the strength of the ventricle, the character of the valvular lesion, the deformity of the valves and the presence or absence of aortic stenosis* (see "Rationale").

RATIONALE.—The Mental Picture.—*The second sound of the heart represents the closure of the aortic and pulmonic valves and thus marks, clinically, the end of systole and the beginning of diastole.*

Semilunar closure.

As regards the left heart* the term diastole at once brings before the mind's eye the picture of an aorta overdistended by, and exerting compression upon the blood which, having been forced at this instant into it by the ventricular systole and shut off from the heart by the closure of the aortic valve, must seek normally the one open pathway represented by the systemic arteries.

One sees also the left ventricle relaxed in *diastole* and the now opened mitral orifice through which the blood is rushing in from the left auricle above to reload the pumping chamber for its next systolic discharge.

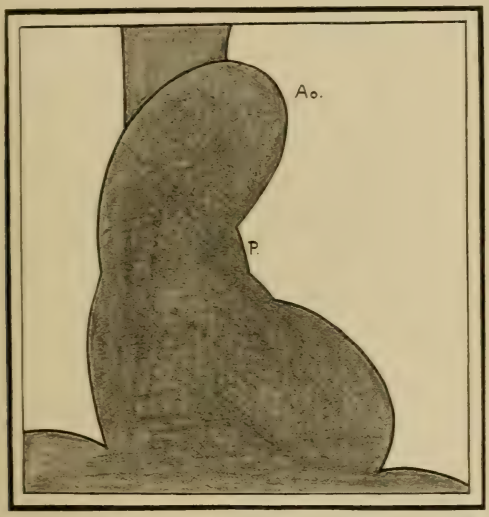


FIG. 385.—Configuration of heart and aorta in compensated aortic insufficiency. The apex is so blunt in this case as to suggest masked stenosis. Compare with normal heart outline and similar figures under mitral lesions. (*Schwarz, modified.*)

Essentials of Valvular Efficiency.—Accurate closure of the aortic valve is absolutely essential to the maintenance of normal conditions of ventricular refilling and the proper distribution of the force of the ventricular drive (pressure energy). This, in fractional part, (one per cent.) is required immediately to maintain the blood flow and, in far greater measure, to store potential energy in the distended elastic aorta, and stabilize pressure and flow alike until the next systolic discharge. This manifestly demands perfect freedom of movement and entire integrity of both the surface and outline of the three cusps of the valve itself, together with adequate strength and consistence of the muscular ring which forms their base and determines their areas of contact.

The normal ventricle.

Normal valves and ring essential.

* One need only substitute the words "tricuspid" "pulmonary" and "pulmonary artery" respectively for "mitral," "aortic" and "aorta" to get the coincident concurrent phases in the right heart and pulmonic circulation.

It is evident that calcareous plaques, vegetations, nodulations, or the deformities due to a sclerosis, no less than a yielding of the aortic ring itself, must, and inevitably will, prevent accurate approximation of the aortic

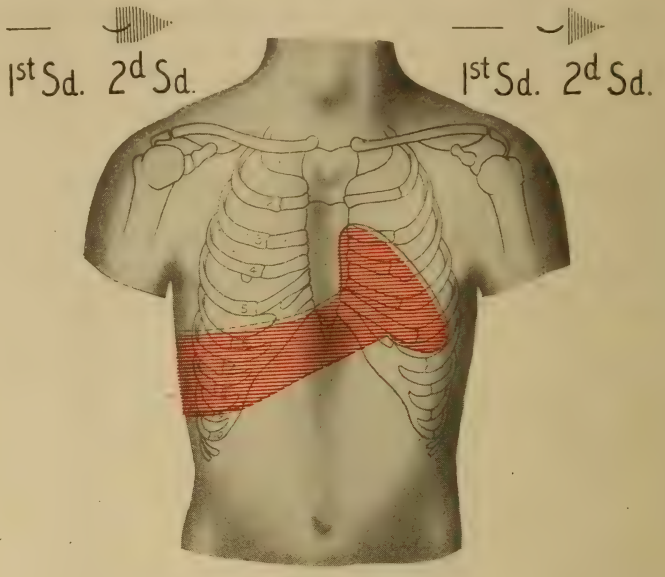


FIG. 386.—Aortic regurgitation. Flat-finger percussion area. Much more accurate delineation now possible.

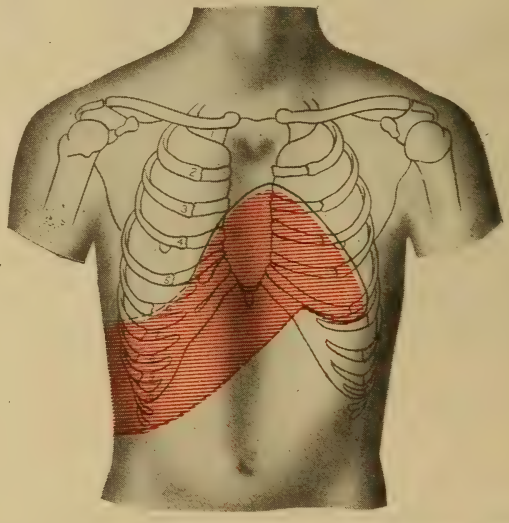


FIG. 387.—Aortic and mitral lesions. Decompensation of right chambers, past or present, suggested by hepatic outline.

cusps and establish a leak and abnormal backflow into the ventricle which thus receives blood from two sources at the same time and must perforce enlarge its cavity.

If we assume the sudden production of aortic leakage, the normal picture is at once changed and we visualize the instantaneous fall in that aortic blood pressure so necessary to a sustained and equalized circulation and the instant dilatation of the overladen ventricle which, pending hypertrophy, must at once automatically draw upon its full reserve, markedly

Sharp drop in pressure.

Ventricular dilatation.

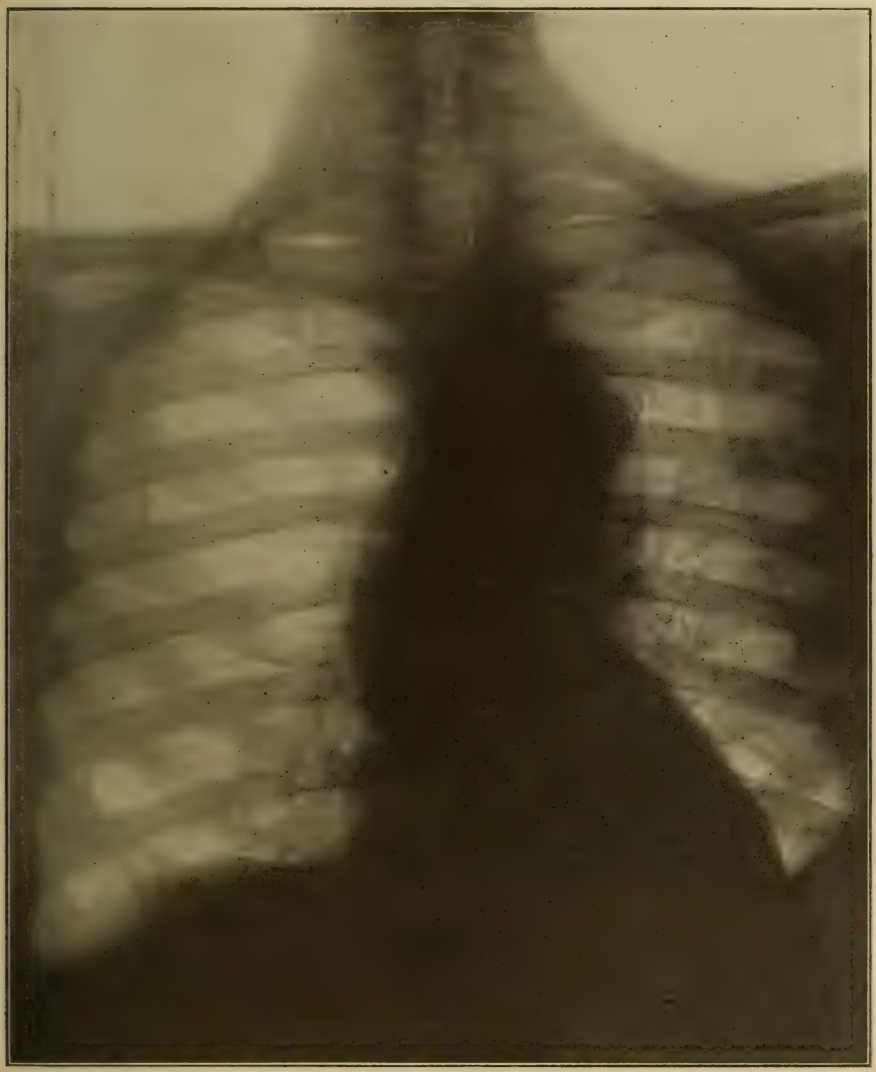


FIG. 388.—Type of aortic insufficiency with dilatation of the aorta.

increase the rate of its contractions, and so expand its cavity as to enable it to pump rhythmically into the aorta the amount of blood necessary to maintain an adequate blood supply for the body tissues and brain centers, in addition to that which is lost through the abnormal diastolic backflow from

Ventricular cavity must enlarge.

that vessel into the ventricle. By a forced increase of tonus it must prevent also a progressive stasis in, and *fatal* dilatation of, its own cavity.

In this simple picture are all of the essential clinical elements of aortic insufficiency.

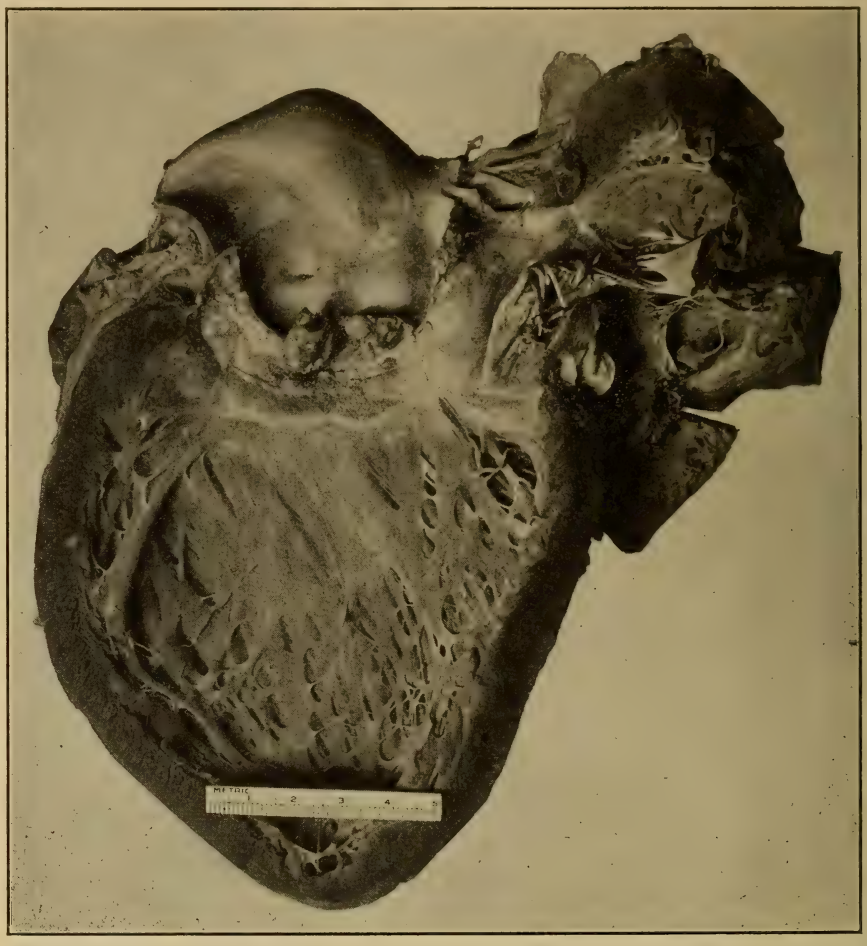


FIG. 389.—Chronic aortic endocarditis with insufficiency. There is thickening and retraction of leaflets and vegetative thrombi adherent both to the central portions of leaflets and the ventricular endocardium. The left ventricle is markedly dilated and heart is hypertrophied. Case is that of man, age 39, who was admitted to hospital because of cough, weakness and orthopnea. Clinical examination showed dilated heart and a Corrigan pulse in right radial but absent in the left. At autopsy, heart weighed 675 grams. There was also thrombosis of right auricular appendage, embolism of left radial artery at elbow, and both recent and old infarction of spleen and kidneys. (*Dept. of Pathology, University of Minnesota. Courtesy of H. E. Robertson, Director.*)

Varied pathologic cases.

The lesion may represent the effects of endocarditis, acute, chronic, simple or malignant; sclerosis of the cusps, cardiosclerosis, acute physical overstrain or myocarditis, myasthenia and myocardial degeneration of any type.

The results are the same in each instance if the integrity of the valve structure itself or of its base is affected seriously.

Obviously, Five Cardinal Symptoms Simultaneously Appear.—1. **A diastolic murmur** *due to a rhythmically recurrent backflow ("regurgitation") of a part of the blood projected into the aorta by the heart's systole.*

2. **An obligatory increase in the size of the left ventricle,** *which is inexorably determined by the abnormal increase of content due to rhythmic backflow from the aorta in each diastole.*

3. **The establishment of high "pulse pressure,"** *through rhythmic alternations of abnormally high "systolic" with abnormally low "diastolic" arterial pressure, in place of the normal alterations of level.*

4. **The "water-hammer pulse"** *reflecting these abnormal pressure variations and intensified by an automatic defensive vasodilatation adapted to a partial stabilization of rate of flow and variants of pressure in the capillary deltas.*

5. **The "capillary pulse,"** *which is merely a visible expression of the abnormalities of flow associated with this lesion and permits a systolic—diastolic advance and recession, flow and ebb, of the arteriocapillary blood stream.*

THE MURMUR OF AORTIC INSUFFICIENCY.—*The eddies and whorls of the regurgitant stream cause a murmur which in time, quality, duration, pitch and transmission follows logically the physiologic, anatomic and pathologic conditions present in this lesion.*

Time of Murmur.—Produced in diastole by the backflow of blood from an overdistended, elastic, or, relatively rigid aorta, through the damaged aortic valve at the instant of its attempted complete closure, the murmur must necessarily be diastolic in time and seem to modify, obliterate, or, more rarely, in the case of lesser defects, closely follow the second heart sound and tend to show a prolonged *diminuendo* quality, its intensity diminishing as the high initial aortic pressure falls.

Why diastolic.

Why diminuendo.

Quality of Murmur.—Under ordinary conditions it is a *blowing* or *hissing* murmur but, obviously, its pitch, quality, loudness, and duration must depend chiefly upon: (*a*) *the strength of the ventricle and the volume of the load discharged by it during the preceding systole;* (*b*) *the resulting head of pressure in the aorta and the elasticity of its walls;* (*c*) *the size and shape of the valvular deficiency; and* (*d*) *the consistence of the valve segments and the degree and character of any roughening or irregularity of the edges of the leaking orifice.*

Hissing or blowing murmur.

Factors determining quality and intensity.

The Many Variants.—*Hence, though we find the murmur ordinarily a blowing or hissing sound of moderate pitch and intensity, clearly audible over certain well-defined areas, we may find it high-pitched, whistling or even distinctly musical.*

It may be so loud as to be audible over the entire heart or thorax, may be auto-audible and a constant torment to the patient, or, so intense as to be distinctly heard by the unaided ear at short distances from the chest.

Auto-audible murmurs.

On the other hand, it may be so soft or faint as to be heard with difficulty over only a single small area, often only after exertion or with the patient in recumbency, may be absent in the erect or sitting posture, and is almost always more distinct in recumbency and increased by moderate stethoscopic pressure. In some instances a murmur disappearing or very faint in the

Faint murmurs.

Effect of posture.

sitting or erect posture may be revived or intensified by having the patient raise both arms above the head.

Silent cases.

In cases of extreme cardiac weakness or excessive size of the leak, with or without coincident aortic or mitral stenosis or insufficiency and high-grade sclerosis of the aorta, it may be entirely absent.

The split 2d tone.

An Important Sign.—*In very slight insufficiencies, usually due to physical overstrain, myasthenia or secondary dilatation of the aortic ring, but from whatever cause, leakage may be manifest primarily only in a split second sound heard only over the third left interspace, or a very short murmur, which may alternate with it or stand alone, prove transient, recurrent over long periods of time, or, become a permanent aortic regurgitant murmur of the classical type.**

Usually long, rarely short.

Duration of Murmur.—So also as to duration one finds usually the prolonged diminuendo murmur starting with the first sound, but, as in the last instance, it may be extremely short and in rare, unexplained and *always dubious*, instances, definitely post-diastolic.†

Maxima of Audibility.—The maximum of audibility is for the most part wholly in accord with anatomic relationship and the laws of sound conduction.

The usual tripod.

The murmur is loudest, normally (not over the valve itself, but over the regurgitant stream which produces it by its whorls and eddies), at the third left interspace, at the sternal margin, or over the sternum itself, midway on a line joining these points, i.e., in the immediate vicinity of the aortic valve. It may be maximal

Occasional variations.

over the aorta itself at the second right interspace, which is the point of election for the auscultation of the *second heart sound itself*, but this is a less frequent *maximal* localization than was formerly supposed. Occasionally the sound is so intensified by unusual relationships and sternal reinforcement of vibration as to be maximal at the ensiform and sometimes, probably because

Rare exception.

of the predominating involvement of the posterior cusp or an accidental diversion of the main point of impact of the stream, the bruit may be maximal at or beyond the apex by reason of the maximal, direct, left ventricular transmission thus produced.

Transmission of the Murmur.—*The transmission of the murmur is also easily explainable. The right heart occupies nearly all of the anterior cardiac area,‡ below the level of the third interspace and tends, to diminish or wholly*

* The author has reported on various occasions the apparent frequency and clinical significance of this split second sound as heard over the third left interspace in association with moderate dilatation of the left ventricle, a distinct capillary pulse, marked variations in arterial pressure, and almost invariably a history of some hitherto forgotten cardiac overstrain or exhausting illness. The sound appears often in alternation with the short diastolic murmur which may become fully established and gradually increase during succeeding months or years to a typical bruit or in rarer instances wholly disappear.

The first cases observed occurred after extreme overstrain associated with marked collapse and have now been watched for several years.

† Such cases are often due to mitral or, very rarely, to tricuspid stenosis, with fibrillating auricles.

‡ In the normal heart, the right border is wholly that of the right auricle which may give place to the right ventricle at the right edge of, or more commonly, the middle of the sternum. Only a strip of the left ventricle is normally accessible. The "drop heart" right border is that of the right ventricle alone, the auricle lying above.

suppress the conduction of any murmur of left heart origin throughout that region.

Angle of conduction.

Hence the murmur of aortic insufficiency is best conducted from the valve and the adjacent cardiac area involved in its production and in the direct impact of the regurgitant jet, along lines connecting the basal areas of maximum audibility (third left to second right interspace and down the sternum to the ensiform, the sternum serving as a sounding board), and is heard with some constancy in the carotids and first right interspace, especially in clearly defined lesions with associated diffuse dilatation of the first portion of the aorta.

Carotid conduction.

Wide audibility.

As stated previously with reference to maxima of audibility, loud murmurs are often widely generously conducted, and in rare instances may be heard faintly over the entire torso. On the other hand, certain cases show them only at a single maximal intensity area or even at the apex only, though, as already stated, the latter is far more likely to be a mitral stenosis with the

Caution.

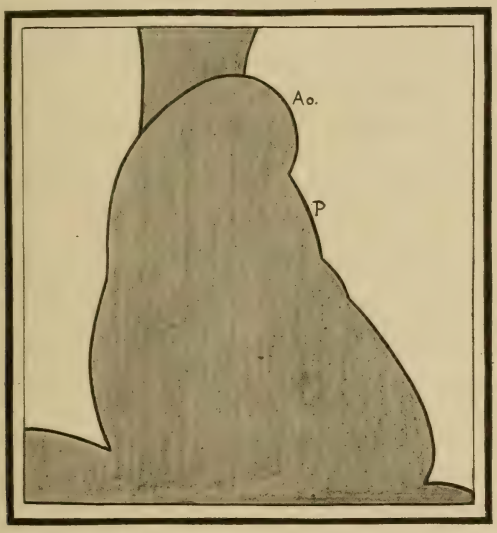


FIG. 390.—Configuration of heart in combined aortic and mitral disease. Compare two mid-curves of left border of silhouette with the normal and with those of aortic stenosis and aortic regurgitation with normal heart outline and those of aortic regurgitation and stenosis. (Roentgenoscopic.) (*Schwarz, modified.*)

auricle in fibrillation *and should be diagnosed as aortic only when the associated signs of aortic regurgitation are present.*

One must keep in mind also the possibility of interpreting the diastolic murmur of pulmonary regurgitation as an aortic reflux. Such a murmur due to over-distention of the pulmonary orifice and *conus arteriosus* may occur in association with chronic mitral disease (Graham-Steele murmur). Error is avoided readily by noting the absence of typical transmission and associated arterial phenomena of the aortic lesion.

Increase of Cardiac Area.—*As stated hitherto, ventricular overload is a marked and immediate result of this lesion and, when rapidly or suddenly produced, obviously must result in a rapid left auricular dilatation.* In acute

Dilatation and hypertrophy.

Cardiac reserve.

cases, this enforces at least maximal physical rest on the part of the patient and demands in such cases the full emergency heart muscle reserve or that amount of it which antecedent toxemia may have spared, to tide over the period, fortunately a short one in favorable cases, until hypertrophy shall dominate, limit the dilatation, and establish a larger field of response.*

Reveals enlargement.

Left Border and Apex.—The enlarged left ventricular cavity must reveal itself in widening and downward projection of the left heart area and, when combined with hypertrophy of the wall of that chamber the persistence of a forcible heaving apex-beat to the left and well below the normal site is a striking clinical symptom.

Aortic Dilatation.—If the aorta itself becomes markedly dilated, some increase of dulness may be made out in the second right interspace near the sternum and if, as often happens, co-existent or secondary mitral disease is evident, an increase in the right heart area is superadded and in advanced cases produces the enormous "ox heart" (*cor bovis*).

Cor bovis

The presence of predominant dilatation is more or less clearly shown in the substitution of an indeterminate "mixed" apex beat, already described, for the sturdy heave and lift of dominant hypertrophy.

May lack marked enlargement.

Hearts with Small Leaks.—One should remember that true small aortic leakages, secondary or from overstrain, may exist without decided, or at least, excessive, left ventricular changes and with little objective or subjective evidence of myocardial overstrain.

Key to signs and symptoms.

The Alternation of High and Low Pressures.—*Unsustained blood pressure and flow constitute the key to nearly every important objective and subjective subsidiary symptom of aortic leakage. The unsupported blood column leaps and falls back, as it were, throughout the arteries and capillaries, the "pulse pressure"† being double or even treble that of health in cases of free regurgitation.*

Pulse pressure.

Water-hammer pulse.

Corrigan Pulse.—The unsustained pulse (*pulsus celer*) is peculiarly characteristic, though not wholly pathognomonic of the disease and the jerking, throbbing, superficial arteries are no less striking to the eye than to the finger which feels the shock-like systolic impact as a tiny but definite blow or tap.

Technic.

Over the radial artery this is best felt if the wrist be actually grasped and the arm raised high up, the palmar surface of the base of the first phalanx of the examining hand being apposed to the radial artery.

Arterio-sclerosis.

As the disease progresses, or as an associated or possibly antecedent condition, arteriosclerosis may exist, and while more or less modifying the intensity and sharpness of pulse impact, renders even more obvious to the eye the arteries of the superficies or superadds to their jerky, throbbing beat the vermicular, rhythmic shortening and lengthening of the serpentine curves induced by extreme arterial rigidity.

Pistol-shot sound.

Arterial Tones.—If the stethoscope be placed over the femoral artery, a sharp, shock-like, systolic pressure tone, "the pistol-shot sound," may be

* Cases of sudden free aortic insufficiency due to valve rupture from overstrain and antecedent disease produce profound shock.

† Pulse pressure is the sum obtained by subtracting the diastolic from the systolic readings of arterial pressure.

manifest, and, if pressure be increased, a "to-and-fro" murmur (the sign of Alvarenga and Duroziez) may become audible. Such signs may be present in other arteries and the *systolic pressure tone* may be present even in the palmar arch.

Alvarenga-Duroziez sign.

The Capillary Pulse.—*Closely related to the Corrigan or water-hammer pulse (pulsus celer) is capillary pulse (of Quincke), not pathognomonic, but one of the most delicate and exquisite of clinical signs.*

Pressure ebb and flow.

This is a capillary manifestation of the abrupt ebb and flow of pressure which takes the form of an alternate flushing and paling of the beds of the nails, the mucous membrane of the lip, the margin of color in a flushed cheek or ear, or in areas of artificial erythema induced by friction.

Technic.—The simplest test for the capillary pulse, if it is not apparent on close inspection, consists in slightly blanching or deliberately reddening by friction the area under observation and watching for the rhythmic recession and advance of the border of color or the flushing and paling of the red area.

Pressure upon the tip of the finger-nail, or that of a clear tumbler or wine-glass, upon the everted lip, or brisk friction of the forehead, are simple and successful methods.

In areas of erythema thus produced, the sign is even better marked when the initial redness begins to fade and, over all, a glass slide applied with moderate pressure will intensify and render clearer the rhythmic paling and flushing, recession and advance.

The appearance in the nail often suggests rhythmic winking or blinking.

This phenomenon may be demonstrated beautifully in a dark room or even by daylight if transillumination of the fingers is secured by grasping an electric hand light or bulb, or by the use of any similar means of illumination.

"Street car diagnosis."

Sight Diagnosis.—*The jerking arteries, especially, make recognition of this ailment at sight or touch often easy though not always certain.*

Musset's sign.

The jerking foot when one knee is crossed over the other, the rhythmic nodding of the head ("Musset's sign"), the beat of the pulse felt when shaking hands, or the rhythmic waxing and waning of an outdoor flush, may make "snap diagnosis" reasonably easy even in the "market place."

Aortic 2nd tone.

Modifications of the Heart Sounds.—Additional modifications of the heart sounds may be present though inconstant. One readily understands the occasional tendency to a more or less complete absence or obscuration of the aortic second sound in cases of extreme valvular defect or cusp immobilization, despite the powerful drive of an overloaded hypertrophied ventricular and increased aortic distention and recoil. In many, if not most instances, however, it is actually increased, and when markedly diminished, one must assume as a probability that the commonly associated aortic systolic murmur is due to an actual co-existing stenosis rather than to mere sclerosis of the aorta.

Mitral 1st tone.

So also the first sound at the apex representing mitral closure is *dull*, even though it be loud, and is often impure or markedly diminished, because of the incomplete emptying of the ventricle and lack of the usual radius of excursion of the cusps, or, this combined with marked weakness of the

heart muscle, by reason of which one or both elements which enter into the formation of the mitral systolic tone may be diminished.

Pseudo-mitral stenosis.

The Flint Murmur.—A presystolic rumble or blubbering sound, arising probably from the pressure of overmuch blood, and the resulting intraventricular pressure, in late diastole, is sometimes heard in aortic insufficiency, and it is quite possible that in such cases the mitral segments are nearly approximated at the time of the presystolic auricular contraction, thus causing a false stenosis.

*According to present opinion the so-called "Flint murmur" is an extremely rare phenomenon and the presence of a presystolic rumble due to any condition other than mitral stenosis is unqualifiedly denied by many eminent European clinicians.**

Careful measurements in the autopsy room indicate that the lesser degrees of mitral stenosis are more common in connection with aortic regurgitation than was formerly believed.

Pseudo-aortic stenosis.

The addition of a direct systolic aortic murmur, having all the qualities of aortic stenosis except obscuration of the second sound and, slight delay in the bruit is very common. This is especially true if marked sclerosis and dilatation of the aorta are present or if general arteriosclerosis or the high pressure of primary, secondary, or associated, interstitial nephritis is evident.

True associated aortic.

An actual stenosis of the aortic valve with diminished or absent aortic second tone and a greatly modified Corrigan pulse is of course a relatively common finding.

When present the two murmurs often closely simulate the sound produced by sawing wood. (The "see-saw" murmur.)

General Symptoms.—As in all chronic lesions, so also in this, a patient may for years show and feel little or no effects from the disease; yet no cardiac ailment is more troublesome when decompensation gets the upper hand and periods of incompensation occur.

A vasomotor paradox.

Pallor.—Such individuals show superficial pallor though internally vasomotor relaxation is present to a marked degree and seems to represent an effort of nature to slow the capillary current for the sake of the tissue exchanges so vital to the processes of repair and waste and the integrity of cerebro-spinal function.

Mental state.

Nutrition and Temperament.—Such patients are usually thin, pale, even though anemia is absent or only moderate, high-strung and nervous, and with marked decompensation, irritability, insomnia and a more or less marked psychasthenic state or even one resembling a severe hypochondria or actual melancholia may develop.

Subjective noises such as thumping, roaring in the ears, tinnitus aurium, vertigo, throbbing headaches and even syncopal attacks are not uncommon and are subjects of constant complaint and additional sources of persistent nervous irritability in many instances.

Pain.—It is almost inevitable that the coronary arteries should suffer a

* There is no doubt in the author's mind relative to the occasional occurrence of the Flint murmur, but quite as little to its rarity. Nearly all such cases prove at autopsy to be due to an associated organic mitral stenosis.

loss of equal calibration and normal elasticity which, together with myocardial degeneration and the unstable nervous balance, leads to various types of arrhythmia and superadds the various painful expressions of angina minor or major so valuable in diagnosis.

Minor and major angina.

Dyspnea.—Dyspnea, though usually and for long periods less manifest than in mitral lesions, is often extreme or even paroxysmal if the myocardial weakness is marked or the coronary changes decided.

Dyspnea.

Sources of Danger.—The nervous system being extremely labile, fear, anger, joy and sorrow, the excessive use of tea, coffee, tobacco, or stimulants,

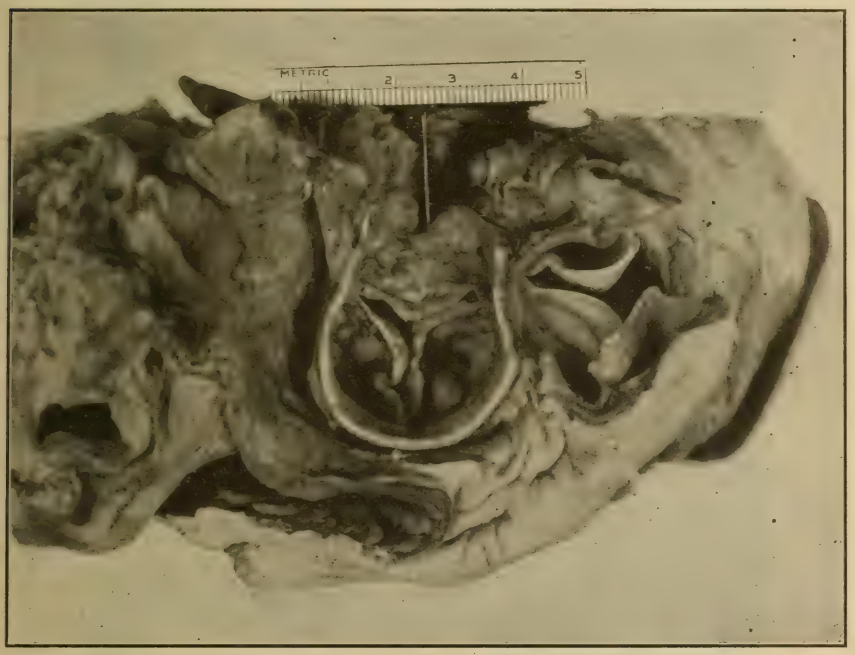


FIG. 391.—Chronic aortic endocarditis with extreme stenosis. Lumen reduced to three slit-like openings meeting in the center. Estimated reduction in lumen, 75 per cent. There was marked hypertrophy of the left ventricle. Case of man, age 80, who died of apoplexy. Kidneys showed advanced stage of chronic diffuse nephritis.

sexual excess, and mental as well as physical overstrain of any kind, may prove not only potent factors in the excitation of symptoms, but, in the case of the major factors, the cause of fatal decompensation or sudden death.

It is evident that aortic insufficiency must constitute one of the commoner causes of sudden death and in this, **as in every other case of heart disease,** *the relatives should understand the possibility of a dramatically tragic exitus.*

May kill suddenly.

AORTIC STENOSIS

Etiology.—*True aortic stenosis is a relatively uncommon lesion, affecting the adult male almost exclusively, and, as an isolated lesion, constitutes a clinical*

*rarity. In the greater number of cases it is associated with a more or less free regurgitation.**

As stated, to a large degree its causes are identical with those of the aortic regurgitations originating after the age of thirty, but it occurs also as a result of acute vegetative endocarditis. It is essentially a lesion of the adult and of advanced age, of lues and of sclerosis, rather than one of the juvenile infectious type as formerly supposed. As the result of chronic inflammation, adhesion and calcareous deposit, the aortic valves become fused into a more or less pouch-like membrane, projected into the aortic lumen and possessing a central circular or slit-like opening. The margins of the opening may be

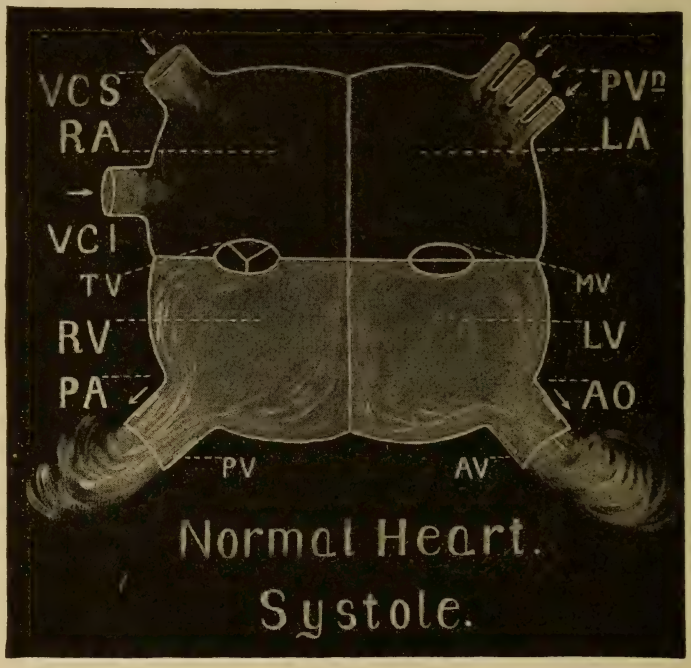


FIG. 392.—Normal heart in systole.

such as will permit a small valvular excursion or it may be merely a rigid diaphragm with a central aperture which, in extreme cases, may barely admit a knitting needle, *post-mortem*.†

Physical Signs and Symptoms.—As might be expected, the signs are extremely frank and definite in advanced cases.

* Save as it constitutes a general rule subject to exceptions, the author's personal experience has not confirmed the perfectly reasonable and logical statements of Bamberger, Romberg, D. Gerhardt and others that mild grades of stenosis are associated with regurgitation and the extreme grades are not. In several of his cases representing the maximal degrees of obstruction the regurgitation has been plainly manifest. *This in fact was the case in the most extreme aortic stenosis which he has seen at autopsy.*

† Its marked predominance in the male strongly suggests that *lues* is a more potent factor than we have been led to believe in times past.

The Murmur.—Time.—Systolic, but actually following the first sound by a fractional interval representing the presphygmic period which in this lesion is slightly prolonged.

Slightly delayed.

Quality.—A loud harsh bruit usually of a "hissing" or "sawing" character which in association with aortic regurgitation frequently results in the "see-

Usually loud and harsh.

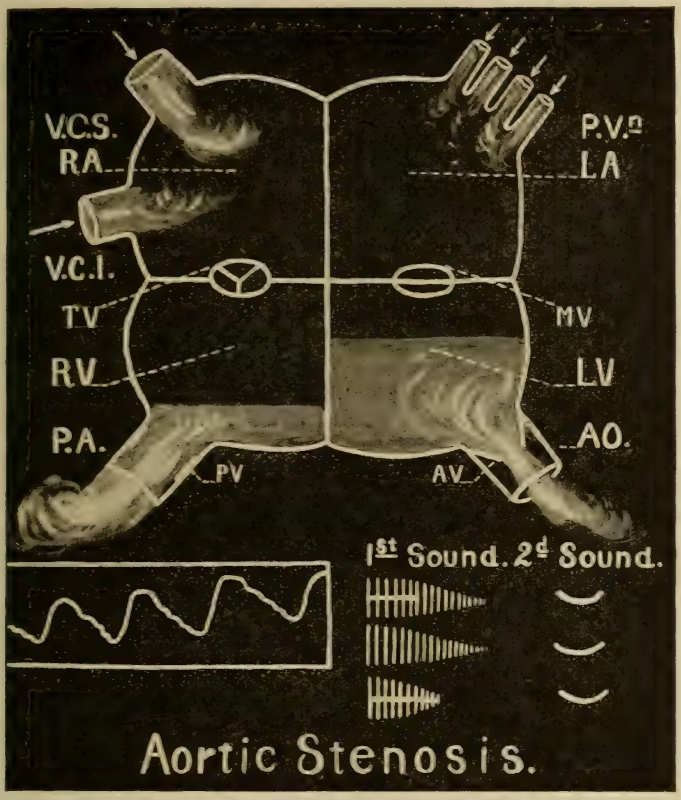


Fig. 393.—Aortic stenosis. A sphygmographic tracing is shown, and three varieties of the aortic systolic murmur are represented graphically. Diagrammatic representation of the heart in systole, stenosis of the aortic valve being present; the mitral and tricuspid valves have closed; the right ventricle is nearly empty; the left ventricle is still more than half full of blood, because of the obstruction present at the aortic orifice. *Result.*—A systolic murmur in the aortic area; enlargement of left ventricle, etc. MV. Mitral valve. TV. Tricuspid valve. AV. Aortic valve. PV. Pulmonary valve. LA. Left auricle. RA. Right auricle. LV. Left ventricle. RV. Right ventricle. VCS. Vena cava superior. V.C.I. Vena cava inferior. P. Vn. Pulmonary veins. P.A. Pulmonary artery. AO. Aorta.

saw" murmur, quite accurately reproducing the double sound of a wood saw in action.

Maximum Intensity and Transmission.—The murmur possesses few vagaries, is heard best in the second right interspace (aortic second sound area) and transmitted to the carotids and subclavian arteries.

Few vagaries.

It may also be heard down the sternum and its left edge, faintly at the apex, or, more distinctly, at the back in the region of the second, third, and

Wholly logical transmission.

fourth dorsal spines to the right and left *the latter representing the general surface area opposite the point of posterior aortic contact.*

Widely heard.

Loud Murmurs.—*The murmur may be extremely loud, harsh or musical and in such cases its conduction may cover the entire thorax and extend down the vertebral column even to the sacral region. It may, moreover, be auto-*

FIG. 394.—Configuration of heart in compensated aortic stenosis (advanced). Note blunt apex and lack of aortic enlargement. Compare with aortic regurgitation outline and that of combined aortic and mitral lesions. (*Schwarz, modified.*)

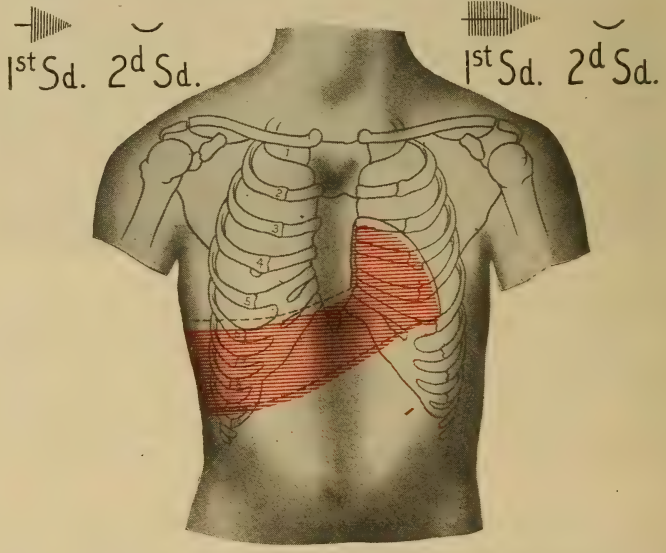


FIG. 395.—Flat-finger percussion area in aortic stenosis (strikingly misleading).

audible, and in quite numerous instances is detectable by the unaided ear at a distance of several inches, or, in rare cases, several feet.

Auto-audible bruits.

Thrill.—In the case of loud murmurs a distinct systolic thrill is almost invariably present in and about the second right interspace.

Aortic Second Sound.—*The aortic second sound is usually, but not invariably, absent or faint and is seldom audible in the first interspace or carotids if the lesion is advanced.*

This constitutes a sign of great differential value in relation to the various pseudo-stenotic systolic murmurs associated with aortic insufficiency, aneurysm and simple dilatation of the aorta, as well as the accidental murmurs, and those of certain of the congenital lesions presenting a harsh systolic murmur at the base of the heart. Occasionally, even in true stenosis, plaques and sclerotic areas maintain a second sound of normal or increased intensity and one is forced to depend upon the slightly delayed murmur, the altered pulse, or a pulse tracing for differentiation.

Valuable differential point.

A puzzling variation.

Cardiac Outline.—Of all the valvular lesions aortic stenosis furnishes us with our best example of dominating hypertrophy of a single chamber, in this case the left ventricle. The left border extends outward and the apex markedly downward.

Little changed for long periods.

For long periods astonishingly little change may be manifest to percussion and, in the presence of other signs, demonstrable enlargement of the left ventricle is not indispensable to early diagnosis.

The Apex-beat.—The apex-beat in established cases is that of dominating hypertrophy, heaving, lifting, forcible, low in position, and more or less removed to the left when the lesion is fully established.

Typical of hypertrophy.

Hypertrophy itself may at first be moderate and over periods of years *decided* dilatation of the ventricle may be lacking. Hence a surprisingly small area of increased dulness may be associated with other definite signs of true aortic stenosis.

FIG. 396.—Maximum of audibility and chief lines of transmission in aortic stenosis.

Late in the disease dilatation occurs, but even under broken compensation the beat seldom shows the indeterminate quality of dilatation to such a degree as is observed in other lesions under the like conditions.

The Pulse.—*The pulse constitutes at times a most important criterion in that it is slow and deliberate, often distinctly small, is palpably long-sustained, slow to rise and slow to fall (pulsus durus).*

Pulsus durus.

Even in cases showing a dominant associated aortic insufficiency, an associated stenosis will oftentimes strikingly modify, without wholly destroying, the characteristics of the *pulsus celer.*

Valuable aid.

RATIONALE.—Aortic stenosis represents a persistently increasing tendency to shortage of blood in the systemic circuit and is the most independent and self-sustaining of all the valvular lesions of the heart; yet there would seem to be a tendency to overrate this quality, for in some of the cases caused by syphilis the duration is surprisingly short.

Symptoms slow to appear.

A slight stenosis of the aortic outlet is readily overcome by moderate

Long enduring.

concentric hypertrophy but, like mitral stenosis, *it is at all times distinctly a progressive lesion*, though very gradually contracting the valve lumen and making its slowly increasing demands upon ventricular strength. Most of the cases *ultimately* reach a point at which ventricular stasis and dilatation become established and persistent and, in part, throw their burdens back upon the mitral valve, left auricle and right heart.

Often remains undiscovered.

It is nevertheless quite true that in many instances the lesion never becomes really troublesome or obtrusive and may not be discovered before death from intercurrent disease and then only in event of autopsy.

Arrhythmia.—Arrhythmias are very frequent in aortic stenosis, *but only when compensation begins to fail*, and take commonly the form of extrasystoles.

Heart block and fibrillation

Heart block of various degrees is not uncommon and fibrillation may occur in the late stages when, secondarily, the right heart and left auricle are markedly affected.

The interesting tables of Lewy show the effect of this lesion in increasing the work of the heart.

TABLE 1.—SLOW PULSE AND PROLONGED SYSTOLE

	Heart's work per hour	
	Rest	Moderate exercise
Ostium normal	813 mkg.	3,400 mkg.
Reduced ½	835 mkg.	4,068 mkg.
Reduced 7/10	991 mkg.	8,864 mkg.
Reduced 9/10	2,112 mkg.	43,578 mkg.
Reduced 19/20	6,386 mkg.	175,426 mkg.
Reduced 49/50	20,000 mkg.	

The strong *normal* heart can exert maximally, *in emergency*, thirteen-fold the effort required in rest, *i.e.*, 13,008 mkg.*

The importance of rest and moderate exercise and of a slow regular rhythm in cases of heart disease is emphasized by these figures, as is the reason for the marked difference in longevity observed in afflicted persons of differing temperament and occupation.

Widespread arteriosclerosis is a common complication and several of the author's patients have suffered most intense angina and repeated syncopal attacks terminating in sudden death.

TRICUSPID STENOSIS

Excessively Rare.—Tricuspid stenosis is one of the rarest of cardiac lesions and, occurring alone, is a medical curiosity. It is usually the result of an antecedent acute endocarditis affecting other valves as well. In a large proportion of the cases it is associated with tricuspid incompetence and occasionally forms a part of a complex congenital defect.

* According to Lewy's statement, if the aortic ostium is reduced by more than 7/10 its possessor cannot perform ordinary day labor, whatever may be the grade of hypertrophy,

As a matter of fact the author has seen at autopsy a heart which showed a reduction of the aortic ostium by 7/8 whose possessor had served as a Turkish bath "rubber" for days at a time up to his last brief illness. He could not perform *unusual* movements, however, without producing distressing symptoms, nor work continuously at his calling.

MURMUR.—Time.—In most instances the murmur is presystolic and of a quality almost precisely similar to that of mitral stenosis; less frequently it is distinctly diastolic (auriculâr fibrillation) and usually lacks the smooth even quality of an aortic diastolic murmur.

Thrill.—In certain instances a presystolic thrill may be felt to the right of the sternum or along its edge or even in the region of the ensiform cartilage, as in the case reported by Robert H. Babcock.

Maximum of Audibility.—The bruit is usually audible over or at some point near the right border of the lower half of the sternum. Occasionally it is maximal in mid-sternum.

Transmission.—It may be sometimes heard over the entire lower half of the sternum and the lateral area immediately adjacent.

Percussion Area.—There is no percussion area pathognomonic of tricuspid stenosis but, as would be assumed, the right border is greatly displaced to the right by reason of right auricular hypertrophy and dilatation.

Subsidiary Signs.—Distention of the external jugulars, an exaggerated *presystolic* jugular pulsation and an occasionally demonstrable presystolic venous pulsation of the liver are the most characteristic associated signs. Obviously, the pulmonary second sound will be relatively faint and the ordinary accentuation attending a mitral regurgitation or stenosis, if this lesion be present in the case, is likely to be diminished or absent.

Effect upon Preëxisting Hypertrophy of the Right and Left Ventricles.—It has been taught always that with the oncoming of this lesion any preëxisting hypertrophy of the right or left ventricle *due to mitral lesions* would undergo a recession and it is probable that this is true in some cases but not of the majority, according to most recent reports of autopsy findings.

Seldom Recognized Intra Vitam.—Only in the rarest instances have reported cases of this lesion been recognized during the life of the patient. This is explained doubtless by the extraordinary rarity of tricuspid stenosis as an *isolated* lesion, which fact tends to allow the more striking and obvious symptoms of the other lesions to obscure a condition so rare as seldom to be recalled to mind, even as a possibility. It is wholly probable that in the future more accurate diagnoses will be made during life.

RATIONALE.—The factors underlying the symptoms are extremely simple. Obviously the two chief conditions resulting from this lesion are: *First*, a failure on the part of the right auricle to convey to the ventricle the blood poured into it by the great systemic veins, the result being marked dilatation and a certain amount of early hypertrophy of the thin-walled *atrium* and, *second*, a transference of the overload and venous stasis to the systemic veins and viscera. This explains the extraordinary degrees of general edema and of engorgement of the kidneys and of the viscera tributary to the portal system encountered. It also accounts for the marked presystolic pulsation observed in the veins of the neck which is often audible together with sharp presystolic clack which would seem to be of sufficiently frequent occurrence to give it value from a diagnostic standpoint. The presystolic pulsation of the liver is attributable to the same mechanism.

The effect upon the lungs and primarily at least upon the left heart chambers is the precise opposite of that just described. A condition of relative anemia or, more properly, deficient blood supply, must inevitably be produced, according to the grade of the lesion and, however much the conditions of relative ischemia present in the lungs may contribute to tuberculous infection, it is evident that all of the pulmonary phenomena associated with the stasis of mitral lesions will be lacking in tricuspid stenosis. Furthermore, while theoretically we should find an extremely low blood pressure and radial pulse, in practice it appears that the blood pressure is maintained at a normal height in most instances and the pulse, though small, is not excessively soft save in terminal lesions. This seeming paradox, of course, is due to the compensating vasoconstriction automatically induced by the conditions present, and no doubt referable in some measure to the continuous asphyxia of the respiratory center in the medulla.

Effect of Long-continued Lesions upon the Normal Left Ventricle.—It has been stated that eventually left ventricular hypertrophy follows tricuspid stenosis of long duration. The actual reports on record would seem to indicate that this development is unusual rather than inevitable, but more information is needed upon this specific point. Babcock has shown by citation of an extremely interesting case that in this ailment a complicating interstitial nephritis may raise systolic blood pressure to 250 mm. of mercury and produce decided hypertrophy of the left heart.

Prognosis.—It has been asserted that this lesion is one permitting a relatively long life in cases where it exists alone and that it does not greatly shorten life as a complicating accident. At the present time such statements cannot be accepted as true of recognizable grades, and it is doubtful whether individuals so afflicted often live beyond the age of thirty and probable that in most instances the lesion endures but a few years. Possibly, however, it is one of excessively slow development and may for long periods fail to attain a grade which materially increases the work of the left auricle.

PULMONARY INSUFFICIENCY

(Pulmonary Regurgitation, Pulmonary Incompetence)

Etiology.—This lesion, extremely rare as a primary endocarditic affection and, as an isolated lesion, a clinical curiosity, is perhaps only relatively so in its secondary form; yet undeniably, it is rarely recognized ante-mortem. This secondary insufficiency represents a dilatation of the valvular ring such as may result from an antecedent mitral stenosis or regurgitation, chronic diseases of the lung or xiphotic spinal deformity.

It occurs in association with a congenital stenosis of the *conus arteriosus*, and, in rare instances, in connection with conditions such as chronic kyphoscoliosis, chronic pleural adhesions, emphysema, and fibroid phthisis which tend to block the pulmonary circulation.

Endocarditic cases seldom or never occur primarily save as a part of

extensive, coincident, right and left heart involvement in an acute endocarditis affecting the mitral, aortic and tricuspid valves.

The pulmonary valve is distinctly weak as compared with its aortic counterpart and the supporting ring is likewise less stable than its fellow.

A replica.

This lesion is, of course, a lesser, right heart replica of aortic regurgitation with no opportunity to duplicate the striking external vascular expressions of that lesion.

CLINICAL SIGNS AND SYMPTOMS. MURMUR.—Time.—*Diastolic, seeming to modify, obscure or even replace the second pulmonary tone.*

Quality.—*A blowing, rasping or hissing murmur, capable of any and all of the usual variations in quality, pitch, and intensity, tending to be prolonged throughout diastole, but usually faint, soft and perhaps short, these qualities being more or less characteristic of the lesser relative insufficiencies which constitute a large majority of the few cases clinically recognized.*

It is usually harsher and more superficial than the aortic diastolic murmur and may be greatly intensified if the breath is held after a forced expiration.

Maximum Audibility and Transmission.—*The murmur is best heard over the pulmonary area (second left interspace); is audible also in the third; and, if loud, is transmitted to the left; also, but less distinctly, over the right ventricle; and, somewhat more distinctly, downward over the lower sternum. If carried at all into the neck, it is heard over the left carotid more distinctly than over the right.*

The murmur is distinctly one of downward transmission and, usually, the aortic tone may be distinctly differentiated, if not lost through coincident or antecedent disease of its own tissue, by auscultation over the first right interspace or deep stethoscopic over the arch of aorta itself in the hollow of the neck above the manubrium.

The second pulmonary tone is usually decreased but, as in the case of its aortic prototype, may be either increased or diminished; the former if the lesion is slight, or associated with sclerosis,* or marked dilatation of the pulmonary artery; diminished, if marked deformity is present or actual stenosis of the valve exists.

Variable.

Alternations of Pulmonary Pressure.—As in aortic insufficiency, abrupt rhythmic changes in arterial pressure are inevitable, but in this lesion are hidden in the area of the pulmonary circulation. They may nevertheless be manifested by a systolic intensification of the breath sounds in forced deep inspiration as heard over lung areas *even though far distant from the heart.* The presence of an audible double tone over the lungs, analogous to that heard over the crural artery in aortic insufficiency is well marked sometimes in this lesion.

Hidden pulsus celer.

Rhythmic variation of breath sounds.

The marked rhythmic waves of cardiac respiratory pressure may also be registered at the nostrils by a suitable apparatus if the breath is held.

Thrill.—A thrill is not uncommon in the endocarditic lesions and is usually most intense in the second left interspace.

* There is probably no definite rule and no less an authority than D. Gerhardt declares that intensification always exists unless stenosis is also present.

Cardiac Area.—*The right heart* area is markedly increased both to right and left and *the apex-beat itself may be formed by the right ventricle. Systolic retractions over the chamber itself would be present in most instances.*

Pulmonary area.

There may be increased dulness in the pulmonary area; sometimes a *marked pulsation in the second and third left interspaces;* and evidences of a dilated conus arteriosus, not only in the form of a decided pulsation, but manifest in radiographs and to percussion in the shape of an extension of the left upper border several centimeters to the left of the sternum in the second interspace.

The Pulse and Blood Pressure.—Unless aortic regurgitation co-exists, the pulse is small and the blood pressure without special features; a state of affairs sufficient in itself to indicate the true origin of any isolated diastolic murmur, maximal at the base of the heart, which lacks the water-hammer quality and the increased "pulse pressure" of aortic regurgitation.

A weak valve.

RATIONALE.—The pulmonary valve, its ring and its artery are alike relatively weak as compared with their aortic fellows, and the valve has been shown (by the late G. W. Gibson) to show leakage under relatively slight excess of pressure. It is theoretically possible, therefore, that, as the result of persistent obstruction of high grade in the pulmonary circuit, whether this be due to disease of the lungs, mitral disease, or chronic myocardial degeneration, the pulmonary artery may dilate and the supporting ring of the valve yield and produce insufficiencies which may be slight or decided, temporary or permanent.*

One still weaker.

The effect of pulmonary insufficiency is to throw an added burden upon the right ventricle and tricuspid valve, such as would ultimately cause a consecutive insufficiency of that, the weakest of the heart's valves, or, in any event a dilatation of the right ventricle, and marked or extreme stasis throughout the systemic venous circuit.

A damaging effect.

The pressure within the lungs is lessened to a disastrous degree, and is no longer stabilized; high systolic and low diastolic pressures alternating rhythmically throughout the pulmonary circuit precisely as they occur in the systemic arteries and capillary deltas in aortic regurgitation. Left ventricular shortage of load results and the blood pressure might be expected to fall but is usually sustained through adaptive vasoconstriction.

Variants of accentuation.

It is evident that the first sound at the apex should usually become weakened and somewhat sharp from underload, though it would remain clear, and that the pulmonic second might or might not be weakened or lost according to the actual condition of the valve itself, the strength of the right ventricle and the presence or absence of tricuspid insufficiency.

Oxygen deficit.

Deficient oxygen and an increased CO_2 content must inevitably result and both *dyspnea* and *cyanosis* are marked and are usually *paroxysmally* intensified.

As with the systemic arteries and capillaries in aortic leakage, so with

*The author feels that clinical evidence is strongly opposed to such an assumption, save as it applies to rare and exceptional instances, for though the pulmonary valve is weak, the tricuspid is still weaker and should act as a safety valve in large measure.

those of the pulmonary area in this lesion great damage to the walls must gradually result.

There must also arise therefore a tendency to bronchitis and capillary oozing which is made evident by *blood-stained sputum.* Sputum.

The cyanosis may be extreme but is of the gray pallid type, by reason of the systemic, arterial anemia.

Pain.—There are no painful sensations characteristic of this rare ailment, unless it be the paroxysms of angina occasionally reported, which are quite similar to those of angina pectoris as encountered in aortic lesions, primary degenerative myocardial lesions and, less often, in mitral disease, are said to be more commonly referred to the right shoulder and arm. The same thing occurs, however, in left heart lesions, though infrequently, and bilateral referred pain is very common in major angina pectoris from any cause.

Otherwise, the discomfort or pain experienced differs in no material respect from that of other incompensated heart lesions.

Differential Diagnosis.—Theoretically, this is simple; but, practically, it seems to offer much difficulty, though many more cases will be recognized from now on, because of a better understanding of the symptomatology and a more careful scrutiny of the pulmonary area in decompensatory degenerative and valvular lesions. Difficult in practice.

Cheif Differential Points.—(*a*) *A diastolic murmur maximal in the second left interspace, with transmission downward along the sternum; or, if upward, wholly or chiefly to the left.*

(*b*) *A clear aortic second sound in the first interspace or, if aortic incompetence be present, a second diastolic murmur, never maximal in the second right interspace nor, if transmitted upward, following both carotids and subclavians.*

(*c*) *An entire absence of the water-hammer pulse unless aortic involvement is coincident.*

(*d*) *Increased intensity of the diastolic murmur when the breath is held after full expiration and during the act itself.*

(*e*) *Profound pallid cyanosis, if decompensation has begun, which differs from that which might accompany a confusing proto-diastolic mitral stenosis murmur in its relatively slight or negative response to rest and digitalis. The apex localization of such a mitral lesion is quite distinctive and confusion rarely, or never, should occur.*

(*f*) *A thrill in the second left interspace and limited to that area is most suggestive but rarely present.*

(*g*) *Sharp pulsation and marked dulness over the conus arteriosus (second left interspace) is suggestive.*

(*h*) *The decided increase of right heart dulness is a valuable sign, both corroborative and differential.*

The X-ray picture and fluoroscopic screen may greatly assist diagnosis if available.

As a factor in congenital heart disease it rarely can be more than assumed and, finally, no one need feel humiliated if he fails to make the diagnosis, for the very elect of our diagnosticians usually make it post-mortem.

PULMONARY STENOSIS AND ATRESIA

Septal defects

Etiology.—*Pulmonary stenosis or atresia is essentially a congenital lesion, is almost invariably associated with patent interventricular septum, is frequently accompanied by other developmental defects and has its inception, usually during the second month of fetal life.*

Most Frequent of Congenital Lesions.—It constitutes or forms a part of 70 per cent. of the congenital heart lesions; an even greater proportion

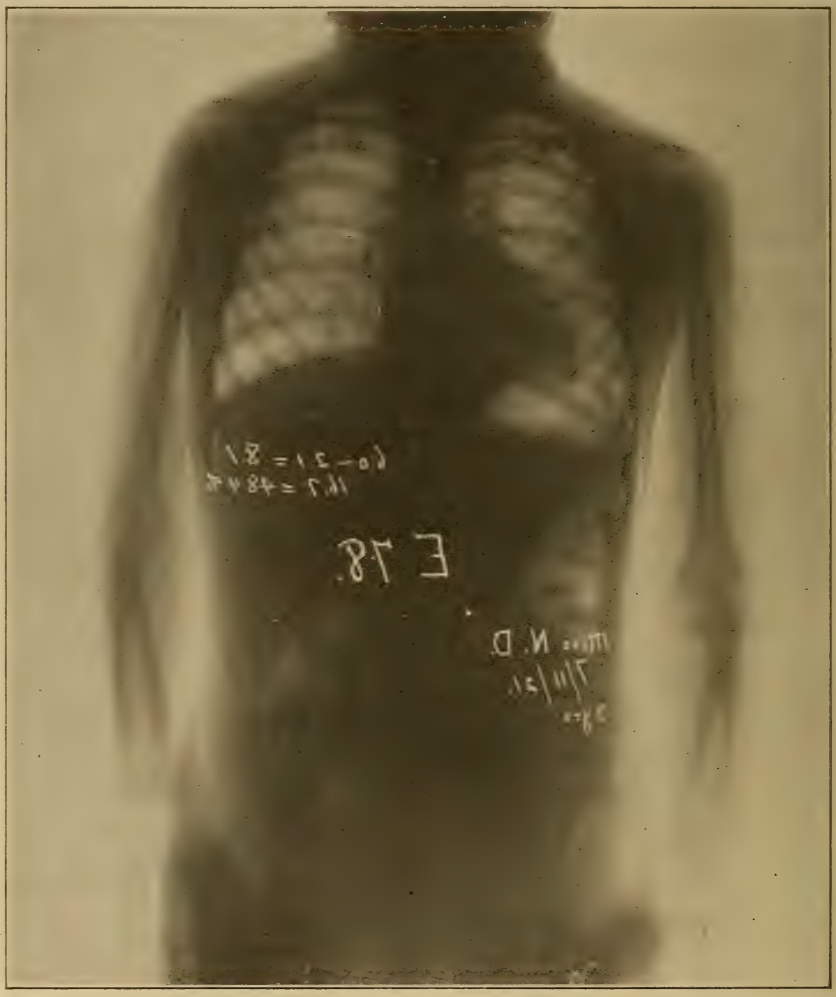


FIG. 307.—Pulmonary stenosis, probably endocarditic, in a child three years of age. Right ventricular hypertrophy beautifully shown. Left ventricle unaffected. (See Fig. 398.)

(80 to 85 per cent.) of those whose possessors have passed the age of twelve years, and is important, not only because of its *relative* frequency, but also because of its pronounced cyanosis, its associated congenital defects, and the relatively prolonged tolerance shown by its victims.

Fetal Endocarditis Rare.—A few cases seem to be caused by fetal endocarditis, but more commonly it results from arrest of development at the *conus* below the valves, or, without the valves, in the pulmonary artery itself, where the trunk is in close relation to the aorta.

Indeed, the obliterative pressure of an aneurysmal sac may produce all of the clinical evidence of pulmonary stenosis in non-congenital cases.

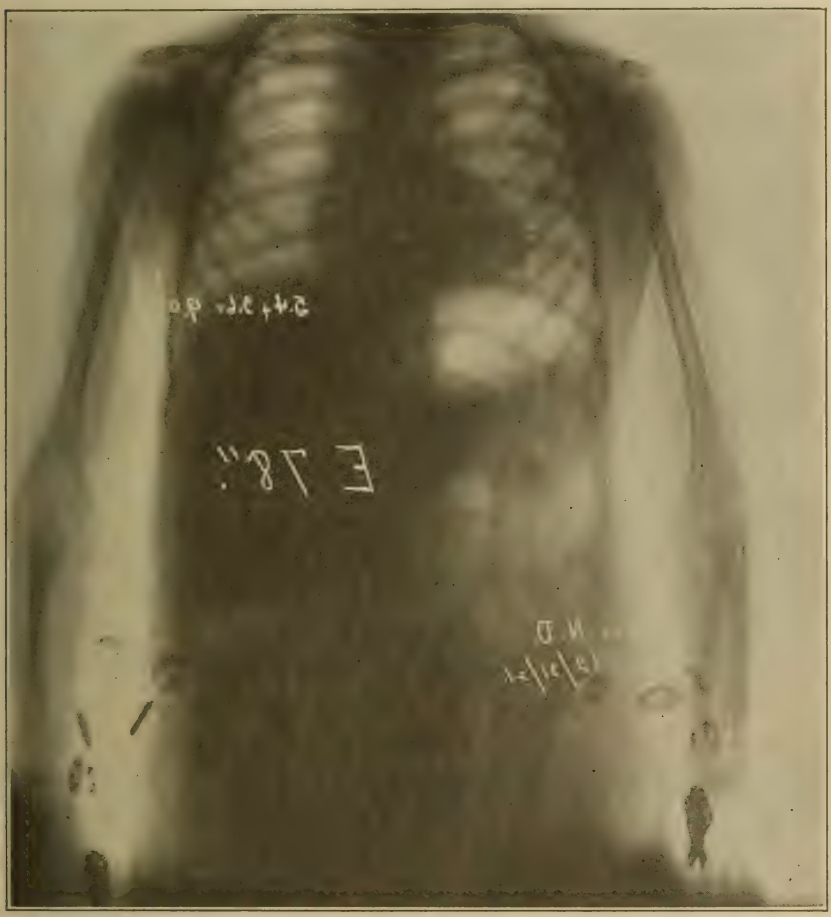


Fig. 398.—(E 78″). Pulmonary stenosis in child three years of age. Same case as Fig. 397.—(E 78), but auricular enlargement was present at the time this exposure was made. Note extension outwards of right heart border. Cyanosis was slight and clubbing of the finger-tips lacking in this case.

Extraordinary Combinations.—It is evident that, if pulmonary stenosis and a defective ventricular septum co-exist, the blood, for the most part, will pass directly from both ventricles to the aorta, and it would seem that septum defects and an open ductus arteriosus (*ductus Botalli*) both, would be necessary to life, as without them only a minimal amount, or, in high-grade stenosis or actual atresia, no blood at all could pass to the lungs. As a matter of fact, however, though present in 90 per cent. of the cases, *ven-*

Open ductus Botalli.

tricular septum defects are more often lacking in actual atresia (complete closure) than in stenosis.

In such instances the lungs are taken care of by a remarkable series of anastomoses.

Patent foramen ovale.

In nearly all of such cases the "*foramen ovale*" is broadly defective but in rare instances this also is closed.

The ductus arteriosus is nearly always open in atresia but closed in stenosis, but it may be impermeable in either, in rare instances.

FIG. 399.—Pulmonary stenosis in adults. (*Sansom.*) Shaded area represents field of maximum intensity.

FIG. 400.—Pulmonary stenosis. Maximum point and direction of transmission.

MURMUR.—Time, *systolic*, representing right ventricular systole and closure of tricuspid valve.

Quality.—*Extremely harsh, blowing and superficial.*

Thrill.—*Systolic*, and present and distinct in the pulmonary area (left 2d interspace) 20 per cent. of all cases, absent in nearly all of those presenting ventricular septum defect with a closed *foramen ovale*, and most frequent, relatively, in those with both the auricular and ventricular septum intact.*

Almost always absent.

Maximum Audibility.—*Second left intercostal space near edge of sternum.* May be widely heard by reason of its intensity, but is transmitted obliquely upward to the left clavicle, and downward, over the sternum.

It is not conducted into the carotids or heard along the right subclavian, as is the case in aortic stenosis.

Pulmonary Second Sound.—Lost or extremely faint in the region of the heart base *in most instances* though cases are reported in which it was apparently increased. The sign is not absolutely dependable.

In complete atresia with an excessively wide septal defect no murmurs may be audible.

Cardiac Area.—*The heart is markedly enlarged as to its right ventricle and ultimately its right auricle. The epigastric heave is usually striking, the chest is usually bulging and the shock of systole may shake the whole body of the child.*

Markedly increased.

* Maude Abbott. Her admirable article, in Osler's "Modern Medicine" should be read by every student.

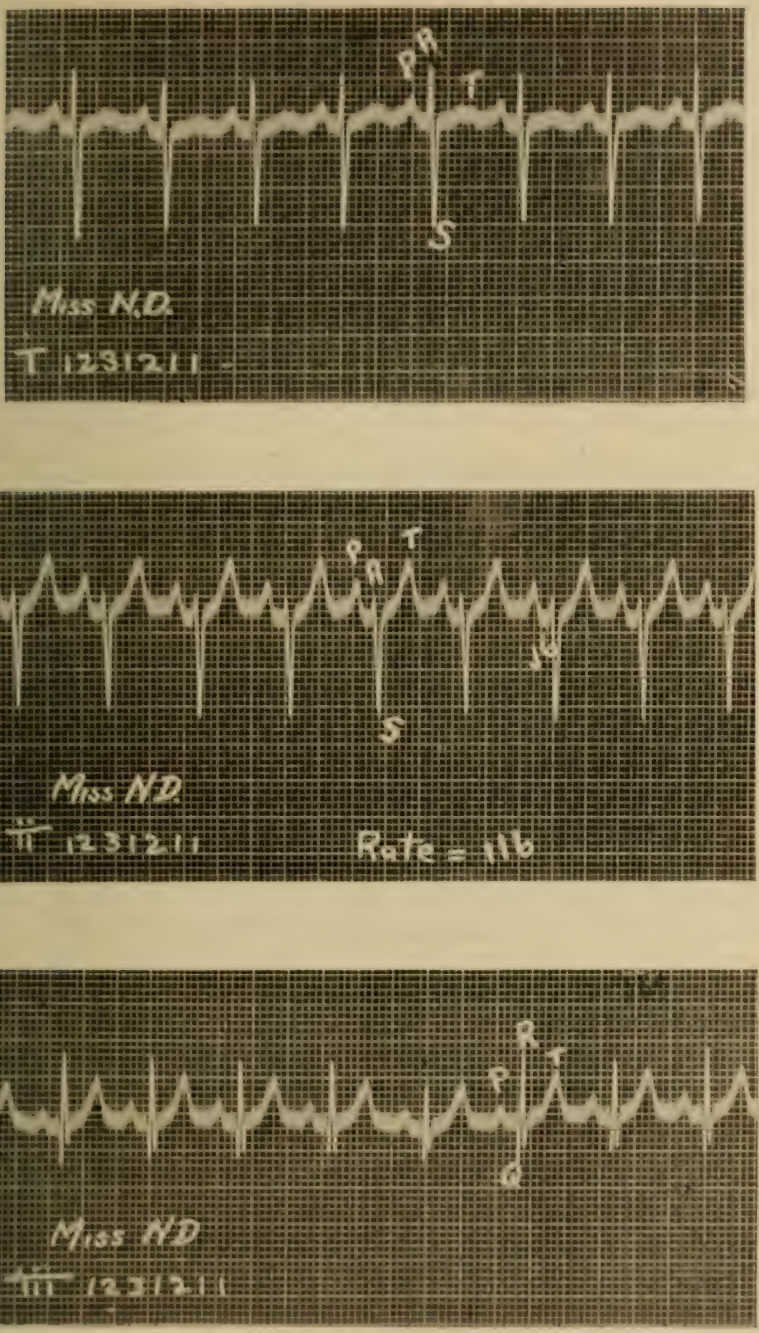


FIG. 401.—Miss N. D. Electrocardiographic record in the case of pulmonary stenosis in three-year old child. Exaggeration of P-wave shows overaction of auricles. Q-wave in Lead III shows continued action of auricles. Right ventricular preponderance indicated by deep S-wave in Lead I and R-wave in Lead III. The deep S-wave in Lead II indicates a marked asynchronism of the ventricles. (See X-ray, Figs. (307) and (308).)

Cyanosis.—*Extreme in grade* and universally distributed over the cold clammy skin of these patients, cyanosis constitutes the most striking single symptom. In the *endocarditic* cases reported by Rissema and by Rosenthal, decided cyanosis was absent.

Blue babies.

The congenital heart cases give us most of the "blue babies" and in them the deep dusky blue color is either persistent or appears and becomes a purple or even purplish black, upon exertion, crying, or in attacks of spasmodic dyspnea, which may be associated with convulsions.

Clubbing of the fingers is usually marked in degree.

Secondary Erythremia.—Cyanosis so profound must lead to erythremia and one may find an erythrocyte count exceeding 9,000,000; increased hemoglobin (120 to 160 per cent.); increased viscosity, and high specific gravity of the blood. Counts of 6,500,000 or 7,000,000 are common.

Increased viscosity.

Differential Diagnosis.—The age of the patient is most suggestive. No other cardiac lesions produce so high a grade of cyanosis as do those of the congenital type and especially this particular disease. Endocarditis is rare in infants. The localization and intensity of the *bruit* are usually fairly characteristic though curious variations occur. Finally the combination of youth, *profound cyanosis* and the physical activity so often maintained is most striking and suggestive.

On the other hand, one must remember that accidental murmurs are peculiarly common in the pulmonary area and that in profound anemia, especially if this be suddenly induced by a massive hemorrhage, both an intense murmur and palpable thrill may occur.

In one of the author's cases such a post-hemorrhagic murmur of pulmonary site was audible to the unaided ear at a distance of 2 feet from the chest.

Comment.—If, as so generally happens, there is an associated ventricular septum defect both the thrill and murmur may be more intense over the third and fourth left interspaces and lower sternum.

If the ductus arteriosus is patent, the murmur is more likely to be sharply transmitted to the back (third and fourth dorsal) and show marked inspiratory accession and expiratory recession of intensity.

An open *foramen ovale* may yield a presystolic murmur.

Compression of the pulmonary artery or its branches may accurately simulate the congenital lesion and this may occur, not only through aortic aneurysmal pressure, but from mediastinal disease (adhesions, tuberculosis, malignant disease, Hodgkin's disease, indurative mediastinitis or massive pericardial effusion).

In all these conditions, however, the pulmonary second tone is accentuated and it may be said with emphasis that diagnosis in the terms of "a congenital heart lesion" is usually simple and easy; its absolute analysis, difficult and often impossible.

Prognosis.—Cases of pulmonary atresia with closed ventricular septum die during the first six or seven months of life. With a defective ventricular septum and a closed *foramen ovale* a few may live ten or twelve years. With

open foramen ovale and defective ventricular septum practically all die within the first year.

In mere stenosis without septal defect, a majority may reach the third

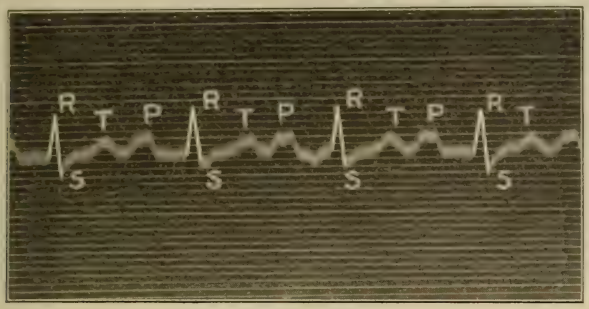


FIG. 402.—Lead i.

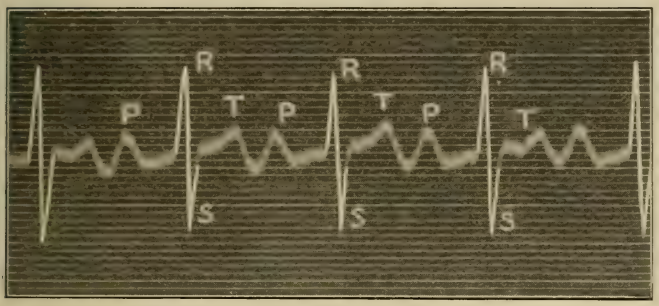


FIG. 403.—Lead ii.

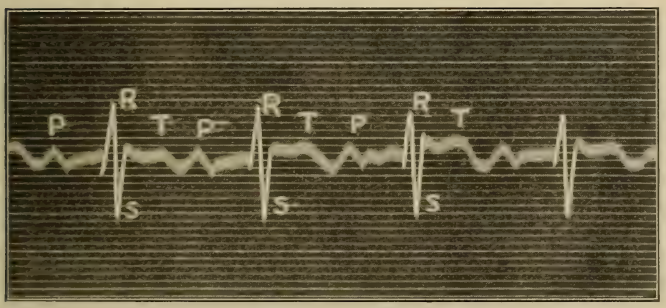


FIG. 404.—Lead iii.

FIGS. 402, 403, 404.—Congenital heart; mitral "P"s; delayed conduction from auricle to ventricle (partial heart block). Lead i shows the typical split (toothed) "P" of mitral stenosis; the less clearly indicated mitral "P" in the form of a square summit; the (relatively) small "R" in lead i and deep S in lead ii suggestive of a congenital element; a "P–R" interval of 0.3 sec. as contrasted with the normal "P–R" of 0.12–0.18 sec. (*R. Edwin Morris.*)

decade of life. With defective ventricular septum few survive the first seven years and fewer still reach the end of the second decade.

With ventricular septum and foramen ovale alike patent, all die during the first two decades.* Trivial foraminal defects are relatively common.

*Maude Abbot: loc. cit.

PATENT FORAMEN OVALE.—Ordinarily this is silent and escapes diagnosis, for even cyanosis may be absent and very slight patency is a rela-

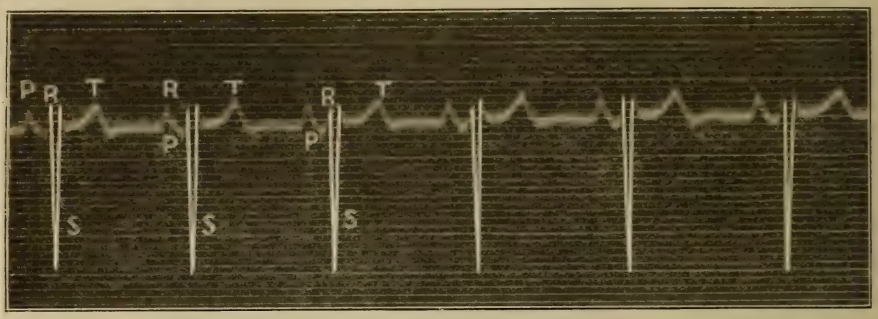


FIG. 405.—i.

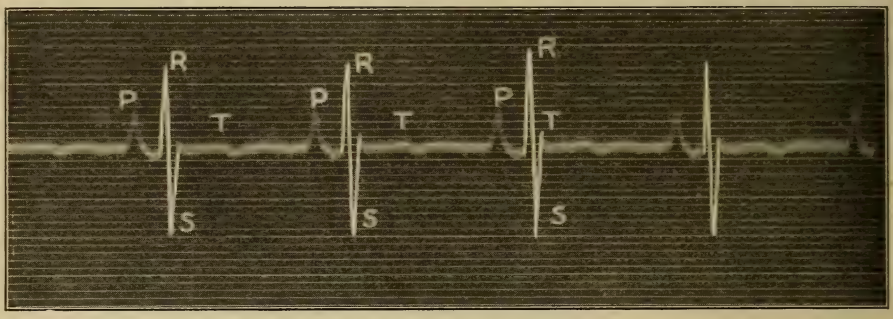


FIG. 406.—ii.

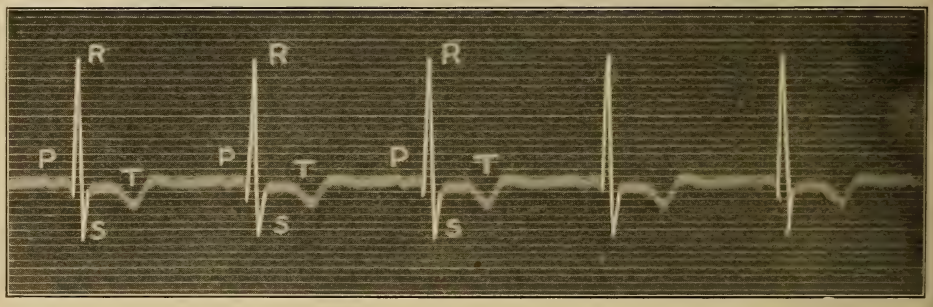


FIG. 407.—iii.

FIGS. 338, 339, 340.—Congenital heart. Fatal case coming to autopsy. The condition is indicated graphically by the (*a*) small "R" (almost zero) in lead i; (*b*) deep "S" in leads i and ii. Right ventricular hypertrophy is indicated by the inversions of the "T" summit in leads ii and iii. Small "R" and deep "S" in i. Large "R" and small "S" in iii. Inversion of "T" in leads ii and iii is thought to indicate a bad prognosis. (*R. Edwin Morris.*)

Note.—In congenital transposition of the heart ("situs inversus") *all* summits in the first lead are inverted.

tively common condition (Vierordt), but as it may be combined with mitral or pulmonary lesions or with both, a most bewildering variety of murmurs is rendered possible.

If pulmonary stenosis, extreme emphysema or fibroid phthisis be present, the increased right auricular pressure may result in a systolic, presystolic, or presystolic-systolic foraminal bruit and an exaggerated presystolic wave in the jugular. Occasionally the varying intensity of the murmur may be suggestive.

If mitral regurgitation is present, a marked systolic foraminal murmur may result from left auricular hypertension due to the regurgitant mitral current and resultant auricular overload, and this may even produce an exaggerated systolic jugular pulse without obliterating the presystolic element, together with a degree of cyanosis far exceeding that ordinarily associated with the mitral lesion alone.

In all such cases the foraminal murmur is most intense over the base though perhaps audible at the apex, and paroxysmal cyanosis may occur.

Paradoxic Embolus.—It is in defects of the auricular septum that one meets occasionally with the curious and significant formation of emboli in the systemic arteries which evidently arise from thrombi in the systemic veins. The diagnostic importance of such a decided abnormality of route with respect to septum defects is sufficiently apparent.

PATENT DUCTUS BOTALLI (*Patent Ductus arteriosus*).—It becomes evident from a review of the congenital heart lesions that without great risk any medical *poseur* might add greatly to his *kudos*, if, when facing a dusky-blue baby, which on crying assumed a yet deeper color, presented to view a heaving chest and epigastrium, and to the ear, a bewildering medley of thrills and murmurs, he were to say "offhand"—"pulmonary stenosis, patency of the ventricular septum and foramen ovale, one, or both together, *and a persistent and perhaps dilated ductus Botalli.*"

Important Structure.—Certainly this last interesting developmental defect is an extremely common and almost indispensable associate of the most frequent of the congenital heart lesions of severity and symptoms.

In fetal life the *ductus arteriosus* is a vitally important and prominent structure, being then greater than the aorta, nearly equal to the pulmonary trunk itself and a direct bond between the two great blood vessels. With the freeing of the pulmonary artery at birth, it almost invariably becomes a useless remnant.

Value in Pulmonary or Aortic Atresia.—If during fetal development the pulmonary outlet becomes actually atresic, or if on the other hand, the aortic flow is blocked (coarctation, aortic obstruction or atresia) this vascular crossing between the greater and lesser vascular systems may be the chief means of circulatory salvation.

In the first instance if the pulmonary block is complete, the left heart assumes the entire burden and pours all the blood into the aorta, the patent ductus Botalli playing the part of a pulmonary trunk.

On the other hand, if the left heart overflow be blocked, the right heart and pulmonary artery take up the entire burden and the ductus arterious may receive and deliver the systemic supply.

It is evident that an accommodating patency of the foramen ovale or ventricular septum or both must serve to switch the blood primarily from

the blocked half of the heart to the open side or wide areas of anastomosis must be opened.

The Murmur.—Obviously, the murmur will be a *slightly delayed or definitely post-systolic murmur*, "rumbling," "rushing," "humming," "churning," "harsh," "scraping," "musical" or, rarely, "flowing," and *should be*

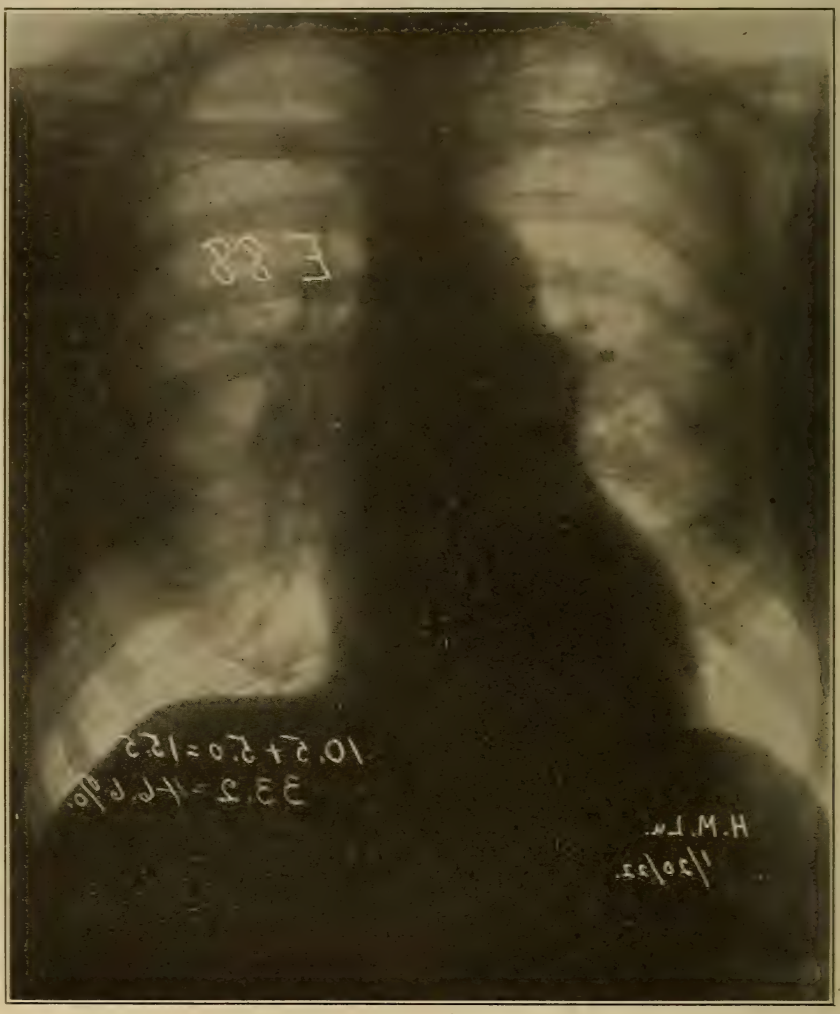


FIG. 408.—Patient ductus Botalli in a man of 55 years of age. Note the dilatation of the pulmonary artery and hypertrophy of the right ventricle.

best heard in the first or second left interspace. It may or may not run over into the diastole, or be continuous. If aortic atresia exists, it may be heard plainly at the left back (third to fourth dorsal).

It shows a distinct respiratory variation and is most intense in expiration. The pulse, on the other hand, is full in expiration and weak in inspiration (pulsus paradoxus). The condition is reversed if pulmonary atresia exists, or

if patent ductus alone is present and the flow comes from the aorta through the ductus to the lungs.

The murmur is then carried *from* the aorta and may be no longer heard at the back unless as a part of the *general dissemination* of a loud murmur by reason of the dilatation of the *ductus* itself and direct transmission from that structure. *It is loudest in inspiration when the pulmonary current comes from the aorta .*

In cases where the aortic and pulmonary valves are competent and the patent *ductus* constitutes the only anomaly the double flow into the pulmonary artery increases the pressure in its circuit and a loud second sound is audible over the second left interspace.

STENOSIS OR ATRESIA OF THE AORTA AND COARCTATION OF THE ARCH

Sites.—*Blocking of the aortic lumen may occur either within or just beyond the site of the ductus Botalli (ductus arteriosus).*

In the former case the systemic circulation will be carried on by the right heart through the aid of the ductus to whatever degree the severity of the lesion demands, for in such congenital lesions septal defects almost invariably permit the switching of the circulation within the heart.

Coarctation.—In the second type the ductus Botalli is of no aid and the circulation must be carried on by anastomoses.

In the first form a positive diagnosis is impossible. There are merely cyanosis and more or less indefinite single or double murmurs.

In coarctation (blocking of the aortic isthmus), the diagnosis may be easy, extremely difficult, or impossible. Visible arterial pulsation of trunks in unusual locations at once suggests it. There may be sharply defined differences between pulsation in the branches given off above the isthmus and those supplying the lower extremities through the arterial anastomoses. Pressure from mediastinal tumor or aneurysm must be excluded.

DEFECTIVE VENTRICULAR SEPTUM.—Though usually a part of a combined congenital lesion it may stand alone. Its manifestations must depend largely upon the question of the size of the defect and the relative strength of the right and left ventricles.

If the normal left ventricular preponderance is present or increased, the escape current will flow during systole from the left to the right ventricle, distend and dilate the latter and produce right ventricular dilatation and hypertrophy which is indeed an almost invariable finding. If pulmonary stenosis or obliteration co-exists, the flow would be in the opposite direction but the right heart would still be chiefly affected (though the left would participate) because of continuous overloading.

Murmur.—In either case a systolic murmur would result, most clearly audible over the base of the heart and, obviously, such a murmur might be less sharply related to the heart tones than would one of valvular origin.

One would naturally assume that it would commence with the maximum

ventricular filling just before the first sound, that it would become suddenly intensified in systole and somewhat overstep the second sound of aortic closure because of there being no valve to check it.

Maximal Audibility and Transmission.—The *bruit* is usually maximal at the base of the heart, at mid-sternum, or in the second or third left interspace, and diminished equally in all directions from its center of intensity.

A striking thrill is often present over the same area and the murmur remains unchanged for years.

It is obvious that with large septum defects there will be neither murmur nor thrill and that very marked changes in intensity must accompany conditions affecting the relative strength of contraction and the load of the ventricles.

No cyanosis need be present in uncomplicated cases but as before stated the lesion is usually associated with pulmonary stenosis and a part of the clinical picture of "*morbus ceruleus.*"

Transposition of the Arteries.—One should bear in mind the possibility of transposition of the arteries in situs inversus or dexiocardia.

CONGENITAL HEART LESIONS IN GENERAL.—*Congenital lesions, aside from the patent ductus Botalli and patent foramen ovale, have these things in common, viz.: (a) They are evident at birth or shortly after. (b) They kill for the most part within the first decade and to a remarkable degree in the first eighteen months of life. (c) They are characterized by marked enlargement of the heart, clubbed finger tips, bulging of the chest, and decided or profound cyanosis, either persistent or easily induced. (d) They represent multiple lesions and are associated usually with well-marked murmurs and thrills.*

They may be, but seldom are, the result of fetal endocarditis, usually due to acute rheumatism in the mother; affect male children twice as often as the female and show a tendency to multiplicity of lesions which is more or less an adaptation of arrested development to the needs of the primary disease or deficiency.

If, for example, the pulmonary artery be markedly stenotic or actually atresic, the ventricular septum may remain undeveloped, the blood be carried to the aorta in large part or *in toto* and the ductus arteriosus may remain open to insure a diversion of a part of the abnormal aortic stream to the pulmonary capillaries. In the same manner an open foramen ovale may relieve the auricular overdistention by equalizing the pressure in the two auricles.

If the changes are in kind or degree insufficient to disturb the circulatory balance, they may remain innocuous for a lifetime.

THE COMMONER COMBINED VALVULAR LESIONS

Caution.—Certain basic principles should govern one who seeks accurately to determine and place the murmurs of multiple valvular lesions.

With respect to **systolic or imperfectly developed diastolic murmurs** *he should attempt ordinarily no more than a tentative partial diagnosis, limited to absolutely provable and obvious lesions, in the presence of:*

(*a*) *Excessive dilatation and extreme cardiac asthenia.*

(*b*) *Excessive pressure upon the heart and consequent decided displacement, whether this be upward, downward or lateral.*

(*c*) *Evidences of decided hyperthyroidism.*

(*d*) *High fever and profound toxemia.*

(*e*) *Extreme anemia and debility.*

(*f*) *Excessive rapidity or extreme lability or arrhythmia of the heart.*

This caution does not mean that one may not make a provisional diagnosis but rather that any final opinion be reserved. Under such conditions, the wise clinician names the murmurs by their time (systolic, diastolic, etc.); places them as to their maxima of audibility and apparent transmission; expresses himself as to their apparent differences as to pitch and quality, associated thrills, pulse tracings, electrocardiograms, radiograms, and what not; carefully outlines whatever of the heart is percussable; searches for secondary decompensation signs and notes them and *stops there* until the conditions permit something in the way of a more complete specific diagnosis.

Misleading pressure effects.

Multiple Murmurs.—*Under the conditions outlined above systolic murmurs may appear to arise at every orifice, yet ultimately be reduced to one or even to zero.* The chief need in such instances is that one should recognize and relieve the associated or resultant myocardial weakness, set aside any existing pressure, and thus lift the burden from the heart and blood vessels.

Primary indication.

Hair-splitting diagnosis under such adverse conditions is a fascinating diversion, wholly praiseworthy, but ultimately disciplinary, chastening and beset by thorns, largely represented by a more or less cold-blooded and critical pathologist. The autopsy room is a veritable "mine-field" for the overconfident diagnostician.

A mine field.

Essential Data in Differentiation.—To repeat all of the intrinsic characteristics and associated signs of the various valvular lesions would be but a waste of time.

Pitch, quality, time, transmission, localized thrills and the like, in so far as they relate to specific individual lesions, are to be sought and made to serve the purpose of differentiation so far as possible.

One is primarily concerned with murmurs originating in the left heart or its outlet, for these constitute literally at least 80 *per cent. of the valvular murmurs.*

Any systolic murmur, therefore, is assumed to tentatively represent either: (*a*) *mitral reflux, maximal at the apex; or,* (*b*) *aortic stenosis, maximal at the base. The former, if endocarditic not "relative," or "accidental," is usually clear and distinct unless the ventricle is very weak or a mitral stenosis co-exists and dominates it or blocks the back flow of a weakened ventricle by the extreme volume pressure of a fibrillating widely dilated paralytic auricle.*

Recapitulation.

Helpful Suggestions with Respect to Systolic Murmurs.—*In the absence of evidence of concurrent mitral stenosis or excessive left auricular dilatation a relatively soft and low-pitched murmur, not typically transmitted, but maximal at the apex, is likely to be due to relative mitral insufficiency rather than to mitral endocarditic valvular disease.*

The true typical systolic murmur of aortic stenosis is transmitted above the

right base of the heart to the vessels of the neck and usually associated with a relative diminution or total loss of the second aortic sound and a more or less characteristic pulse effect.

Common Diastolic Bruits.—If the murmurs heard are diastolic in time, the assumption is that any homogeneous murmur, maximal at the third left interspace, ensiform, or the right second interspace, transmitted chiefly along the lines indicated, and, if loud, usually heard in the right carotid or in both is caused by aortic regurgitation. This assumption is confirmed if we find even a modified water-hammer pulse (*"pulsus celer," "Corrigan pulse"*), and a high "pulse pressure," *in the absence of signs of marked hyperthyroidism* (Parry's disease, Graves' disease, exophthalmic goiter) and, in the presence of the *diastolic* murmur, is a sound one to rest upon even if hyperthyroidism is present.

If an audible diastolic murmur is maximal just within the apex-beat, begins just after the second heart sound, is *blubbering or vibratory*, and especially, if it shows a thrilling purring crescendo in presystole and ceases *abruptly* as if "shut off" by or actually terminating in a loud clapping or slamming first sound, we know that it is due to **mitral stenosis** unless aortic regurgitation is present. This suggests the *possibility* of the pseudo-mitral-stenosis bruit ("Flint murmur") caused, probably, by a false rhythmic stenosis due to the regurgitant blood from the aorta plus a large residual ventricular content which causes the valves to lift prematurely in late diastole and somewhat obstruct the flow from auricle to ventricle during the atrial systole.

Flint murmur.

The Flint murmur is so rare as shown by autopsy that the wise man merely records it as a possibility if aortic regurgitation is proven and makes a tentative diagnosis of "associated mitral stenosis."*

Tricuspid stenosis and pulmonary stenosis are excessively rare save as a part of the complicated and striking clinical pictures presented by congenital heart disease which cannot, and need not, be dealt with here, although the former probably occurs more frequently in connection with tricuspid regurgitation of the endocarditic type than is generally known.

Tricuspid regurgitation is relatively rare as represented by a clean-cut sharply defined murmur of chronic endocarditis and offers little difficulty with respect to differentiation, whether from aortic stenosis or mitral regurgitation of the endocarditic type.

It must always be borne in mind, nevertheless, as a murmur which represents an important secondary (relative) insufficiency, in all cases associated with obstructed flow in the lung circuit whether from mitral disease, primary or secondary, or from excessively chronic pulmonary lesions. Unless very slight it cannot exist without producing a decided or excessive extension of the right border of the heart (right auricle) and diminution or loss of the second heart sound in the pulmonary area.

Extremely important lesion.

It is usually associated with either persistent distention of the anterior

* As previously stated its very existence as a clinical phenomenon is denied by some of the most eminent of foreign clinicians.

cervical veins or direct (systolic) jugular pulsation and is probably never maximal either in the mitral or aortic area, but over and about the lower sternum. Important associations.

Pulmonary insufficiency is dealt with fully elsewhere. It is excessively rare as a clinical finding, though, possibly, this represents the imperfection of clinical methods rather than such extreme rarity as our statistics would seem to prove.

Thrills are usually accurately related to the classic auscultation points of the valves and when present are extremely definite and helpful. Aortic stenosis very commonly shows one, mitral regurgitation only occasionally, aortic regurgitation quite frequently, and mitral stenosis quite constantly if the presystolic murmur is marked and, not infrequently, when only the diastolic bruit is present or even when no murmur is audible even to the lightest stethoscopic contact or upon changing the position of the patient.

The presystolic thrill over the limited classical area of mitral stenosis murmur audibility and the systolic shock or sharp tap imparted to the finger as the purring tremor abruptly ends is as nearly a pathognomonic sign as any in medicine.

Even in its absence the sharp, circumscribed tap usually remains palpable and often visible and justifies the experienced diagnostician in a tentative opinion even in the absence of a characteristic bruit.

The Cardiac Outline.—This is of the utmost importance and by orthopercussion, modified auscultatory percussion or, the radiogram, if the X-ray is available, one largely may confirm or correct previous inferences and oftentimes gain much additional information (see "Radiography").

It should be remembered that, in general, aortic lesions, of themselves, cause left ventricular hypertrophy and dilatation resulting in an extension of the left border and apex-beat downward and outward; that mitral lesions affect chiefly the left auricle and to a less decided degree the left ventricle primarily; the right ventricle secondarily and, but only when the latter weakens or secondary tricuspid leakage ensues, the right auricle.

All of these effects are shown upon the screen or by a radiograph and may usually be determined with considerable accuracy by modern percussion methods.

Paradoxic Right Heart Pulsations.—*It should be remembered also that the impulse (apex-beat) of a decidedly hypertrophied left ventricle is direct, deliberate and systolic both to the palpating hand and to the eye, whereas that of right ventricular hypertrophy or dilatation which may push aside the left ventricle and form the apex-beat in mitral lesions or primary right heart enlargement, may be diastolic to the sight, because of its withdrawal from the chest wall in systole and the resulting wave-like recession of the overlying tissues.* Midway pulsations.

If an hypertrophied left ventricle is only partially blocked off from the chest wall by an enlarged right heart the characteristic deliberate heaving impulse may be only shortened.

In such cases the right heart systolic recession of interspaces may blend curiously with the left ventricular systolic thrust ("mixed" apex beat) to form a widely diffused undulant impulse.

If both ventricles are hypertrophied but weak and dilated, and yet the left ventricle can reach the wall, a curiously indeterminate breaking-wave effect may result especially marked in double mitral lesions.

Any epigastric impulse or heave, systolic as timed by the carotid beat, and not transmitted from the aorta, is left ventricular in origin (*Mackenzie*).

Any epigastric impulse or heave which is due to the right ventricle is systolic in its descent, as timed by the carotid.

Actual systolic pulsation of the liver itself is expansile and involves the entire viscus. One hand in front and one at the back enclosing it, readily determines its expansion.

Any diffuse impulse heave or apparent retraction of cardiac origin, maximal between the true apex and the left parasternal line or left edge of the sternum, is due to right ventricular hypertrophy, or if it be wavy and indeterminate, to right ventricular dilatation, which is usually confirmatory evidence of primary lesions situated in the left heart, of chronic obstructive lung lesions, or of generalized extreme myocarditic weakness.

Finally, massive hypertrophy of the right ventricle extends the superficial area of dulness chiefly to the left and outward *tending* to displace the left heart from the chest wall. It also carries it inward to the median line of the sternum. All increase of dulness to the right of the sternum, if of cardiac origin, is due to enlargement of the right auricle unless a "drop" heart exists. A dilated left auricle lies chiefly posterior but when dilated causes a radiographic projection shadow or area of percussion dulness in the left heart profile below the bend of the pulmonary artery.

RATIONALE.—Multiple Lesions the Rule.—The nature of endocarditic processes and the deformity and limited radius of excursion which the valves usually undergo makes it evident that single lesions must be the exception and multiple defects the rule.

Endocarditic sequelæ.

Whether the primary change be endocarditis or sclerosis, the tendency is toward relative fixation and more or less deformity of the valve segments and thickening and retraction of the chordæ tendineæ of the mitral or tricuspid leaflets.

One, for example, can picture a rigid deformed stenotic mitral as obstructing normal flow from the auricle to the ventricle, but it is difficult to see how such a valve can ever completely shut off a reflux from that chamber by absolute closure in ventricular systole. The same statement applies to the tricuspid and semilunar valves.

Nevertheless, isolated stenosis, rarely, and pure regurgitation, commonly, do exist, as shown clinically and by autopsy.

A crumb of comfort.

The frequency with which *chief* endocarditic valvular lesions lack *signs* of other lesions coexistent cannot be stated accurately because of the wide variations in all tabulations, but doubtless such cases are common, a bit of comfort for erring students and clinicians alike.

Pure valve defects are rare at autopsy and relatively so when judged by clinical findings.

Mitral stenosis and regurgitation are peculiarly changeable and all

clinicians have noticed the tendency to dominance of one element or the other, oftentimes to a degree which eliminates the lesser murmur. Shifting dominance.

One can see with his mind's eye a residual mitral endocarditis which has thickened and deformed the valve and thus prevented its accurate closure and caused a typical mitral systolic murmur. If he follows the image further, he may see that the valve margins have become adherent and the opening narrowed, an event reflected perhaps only in a sharply increased first sound, due to relative rigidity, but if the process goes on, as mitral stenosis inexorably does, he finds that the contracting ring is actually reducing the volume of the regurgitant current and that a murmur diastolic-decrescendo and presystolic-crescendo, running into the loud snap and slam of the first sound, must wholly dominate, if the systole of the auricle is maintained. If the auricle fibrillates and its systole is lost, the clear presystolic-crescendo element of the mitral *bruit* disappears and the relatively stronger ventricle may carry into its original dominance the systolic *bruit* of the regurgitant stream. The intra-auricular distention-pressure in fibrillation may be so great, however, as to actually block the backflow sufficiently to remove any preëxisting murmur beyond the range of audibility, if the ventricle be weak and the aortic outlet unobstructed. Under rest and treatment the auricle may cease its futile flickering and again show tonus and systolic contraction and assume its dominant rôle. Genesis of stenosis bruit. The silenced murmur.

Aortic Stenosis and Regurgitation.—Somewhat similar changes occur in stenosis and incompetence of the aortic valve but are of a relatively simple sort and a tendency to progressive increase of the systolic (stenotic) murmur is likely to be noted as time progresses. Progressive lesion.

Multiple lesions are demonstrable clinically in about one-half of all cases of chronic endocarditis.

Bridging Gaps.—Even though an endocarditis may have spared the aortic valve and assailed the mitral primarily, it may bridge the short gap between the anterior mitral cusp and the aortic valve or *vice versa*. This occurs doubtless in cases in which, during the carefully observed acute disease-phases of, and in convalescence from, acute rheumatism, only one valve seems to be affected; yet when the patient is examined later, two valves are found defective with no history of intervening fresh infection to account for it. Postponed secondary involvement.

The interaction of multiple valvular defects combined with the variations in the relative strength of the heart chambers permits almost as many combinations as a pack of playing cards.

If an incompensated mitral stenosis and an aortic stenosis co-exist, for example, the ventricle receives little blood and less work is required to deliver it to the aorta. As a result of these peculiar conditions the left ventricular hypertrophy so characteristic of aortic stenosis may be wholly lacking throughout the entire (short) duration of the lesions. Neither would the pulse be the small "*pulsus durus*" of aortic obstruction (Romberg). A clinical paradox.

If mitral and aortic regurgitation co-exist or aortic stenosis and regurgitation, the "water-hammer" pulse ("Corrigan," "*pulsus celer*") is diminished in its definition and intensity decidedly with progressive obstruction at the Modified "Corrigan pulse."

aortic outlet. Indeed a combination of the "*pulsus celer*" and "*pulsus durus*" may be manifest to the trained fingers.

The effect of combined lesions in general is not conservative, however, but destructive, though this does not mean that the victim cannot endure them for decades, in some instances doing hard manual labor.*

CARDIOVASCULAR SYPHILIS

Recent Expansion of Our Knowledge.—But a short time ago our exact knowledge of cardiovascular syphilis might have been written upon a postal card. Today it boasts a tremendous literature and embraces important diseases seemingly wholly alien to their basic cause.

The discovery of the Treponema pallidum, its fortuitous and roundabout but definitive articulation with complement-fixation, the specific allergic test of Noguchi, and our ability to demonstrate Treponema pallidum in the diseased tissues, have transformed etiologic assumption and suspicion into actuality, and made the terms "para"- or "meta"-syphilis, or even "luetic sequelæ" misnomers.

Illuminating discoveries.

We know now that from the time of the initial lesion to and through that of tertiary manifestations, even decades removed, we are dealing with actual syphilis —with the activities of the living causative microörganism.

Anachronistic terms.

Enormous Cardiovascular Field.—*Cardiovascular syphilis embraces a large part of toxemic, primarily degenerative arterio- or atheroscleroses, a yet greater and more important proportion of the cases of obliterative endarteritis, a large number of myocardial impairments, and nearly the whole of chronic productive mesaortitis and mesarteritis.*

Persistent living causative organism.

Etiologic catholicity.

Many cases of chronic hypertensive renal diseases must be added, together with the specialized obliterative endarteritis of the basal cerebral vessels, so potent in precocious or fifth decade apoplexies.

Recent investigations moreover add locomotor ataxia and general paresis, almost if not quite in toto, and in each, cardiovascular lues is evidently the basic factor.

Renal disease and apoplexies

Cerebrospinal Syphilis.—*In tabes dorsalis* the primary change is a luetic leptomeningitis, with most extensive involvement of the arteries, marked infiltration, and a perivascular inflammation which follows the arterial pathways to the selective areas of attack.

"Tabes" and "paresis."

Paresis.—*In paretic dementia we find with astonishing frequency syphilitic mesaortitis of some grade; basal, luetic, obliterative endarteritis and, constantly, in the cortex, a luetic endarteritis.*

A luetic leptomeningitis.

In the case of both, the high percentage of positive Wassermann and luetin tests, and the repeated demonstration of *Treponema pallidum* in the tissues, have shown them to be the best representatives of the effects of selective

* In one case observed by the author, a double aortic lesion and a free mitral regurgitation had apparently been carried from the age of fourteen to that of thirty-two although for fourteen years the patient had worked as a locomotive fireman. His compensation failed abruptly upon his receipt of a *slight injury* to the left chest and was never fully regained. Similar cases often arise.

endarteritic cardiovascular lues and the leading examples of *cerebro-spinal syphilis.*

To the list must be added:

Myelitis	50 per cent.
Congenital mental defect	50 per cent.
Epilepsy	50 per cent.
Paroxysmal hemoglobinuria	100 per cent.
Primary optic atrophy	100 per cent.
Disseminated sclerosis	30 per cent.
Infantile cerebral palsy	50 per cent. plus.

and several other conditions.

FIG. 409.—Syphilitic mesaortitis. Aneurysm involving ascending and transverse portions of the arch, coincident aortic regurgitation. Symptoms of aortic regurgitation and "neurasthenia" only at time photograph was taken. Later those of the aneurysm present became evident.

Factors in localization.

With respect to general paresis the stress of urban life and congenital vascular inadequacy when imposed upon a syphilitic are the potent factors in localization.

In Berlin between 30 and 40 per cent. of the male *residents* seeking hospital admission are said to be paretics, *but it is rare among those coming from the country.*

Kraft-Ebing's etiologic reference of this ailment to "*civilization and syphilization*" is proven now to be more than a striking phrase.

Mesaortitis.

Importance of Luetic Aortitis.—The author will deal only with certain basic statistical facts and a brief discussion of the most anatomically characteristic and clinically complex cardiovascular expression of acquired syphilis, namely, "*chronic productive luetic mesaortitis.*"

To emphasize the importance of this condition one need but know the following facts:

First.—Ninety per cent. of all persons dying of syphilis will show microscopic or macroscopic mesaortitis and in 50 *per cent. or more it will be the actual cause of death.*

Second.—Eighty per cent. of the symptomatically recognizable cases present aortic regurgitation.

Third.—In all cases embraced in this subgroup, more or less decided impairment of the coronary flow results, although in but 10 *per cent. is angina pectoris major present.*

Fourth.—About 90 *per cent. of these cases become manifest by decided clinical signs between the ages of* 30 *and* 60. (*4th decade,* 25 *per cent.; 5th decade,* 50 *per cent.; 6th decade,* 15 *per cent.*).

Fifth.—It kills in the very prime of life, is peculiarly intractable and progressive once severe decompensation is established and constitutes, one of the most disabling, harrassing and painful of all lethal ailments and a not infrequent cause of sudden death.

Sixth.—In no chronic disease is early diagnosis more desirable or less frequently attained.

Great frequency.

Some Astounding Figures.—It is asserted that the United States now contains approximately 2,700,000 *adults* carrying some grade of syphilitic aortitis; that of these, 2,160,000 are men, 540,000 women, a large proportion of the latter and a small number of the former being wholly unaware of past infection. Ninety per cent. are found to deny infection under routine case taking and one-half in the face of positive complement-fixation tests.

Denial of infection.

The following are among the latest results reported:

Hubert, 8652 consecutive admissions to a general hospital service. Result 8.8 per cent. positive Wassermanns.

All admissions to Bellevue and Presbyterian Hospitals, New York. Result 20 per cent positives.

Same procedure at Peter Bent Brigham Hospital, Boston. Result 15 per cent. positives.

An extended investigation by the United States War Department (Vedder) showed that nearly 17 per cent. (16.88) of accepted recruits were Wassermann positive, and upon the basis of results obtained at West Point, it is asserted as probable that 5 per cent. of college students are infected.

From 3 *to* 6 *per cent. of babes under one year are victims of hereditary syphilis.*

These figures, though startling, are quite in correspondence with those of other highly civilized and Christian countries.

Concealed Identity.—*Though almost unknown on certificates as a written cause of death, it is evident that syphilis must kill annually an army of men in the very prime of life, after a sneaking, insidious course, extending usually over many years. It is the slow poisoner and concealed assassin of many who have long thought themselves cured, and its cardiovascular types are more fatal than all other syphilitic lesions combined.* Striking facts.

Reduction of Life Expectancy.—The inadequacy of our therapeutic methods is demonstrated clearly by the experience of Life Insurance Companies. These have always exacted at least the evidence of effective, long-continued treatment and prolonged freedom from clinical symptoms, yet Deneke accurately asserts *that a history of past syphilis means an increased death rate* 68 *per cent. in excess of the expected mortality.* Inadequate therapy. Insurance experience.

Recently published statistics show an astonishing selective distribution of the excessive death loss. Selective incidence.

The "Gotha," for example, finds that in insured syphilitics the following causes of death showed the tremendous increases set opposite them, namely:

	Per cent. over normal loss from that cause
Malignant growths	60
Renal disease	64
Gastro intestinal	84
Cardiovascular	116
Apoplexy	128
Mental and nervous diseases	145
Suicide	122

Without further comment we pass to our main topic:

CHRONIC PRODUCTIVE MESAORTITIS.—Mesarteritis vs. Atherosclerosis.—One must distinguish the *inflammatory or subinflammatory* luetic mesarteritis from the *degenerative* arteriosclerosis or atherosclerosis which represents the commonest expression of vascular disease. Important distinction.

Mesarteritis is not primarily degenerative, but irritative and destructive. Atherosclerosis as a type is degenerative from beginning to end.

The former represents in all probability the direct activity and actual presence of the pathogenic microörganisms; the latter, a low grade of toxemia from any one of many sources one of the chief of which is syphilis. Basic factors.

Nothing could be more striking than the degenerated rigid "*pipestem*" arteries of certain cases of congenital syphilis *and lues can either itself initiate, or blend its endarteritic changes with, any type of toxemic degenerative arterial disease.* Luetic degenerative changes.

Indeed in the earlier stages the luetic nature of its endarteritic types is usually readily demonstrable under the microscope.

Lymph Channels and Veins.—*In all of its irritative, inflammatory or subinflammatory forms* the disease is as avid of lymphatics and veins as of arteries and exhibits a tendency to selective involvement, whether of certain regions, mere sections of an affected vessel, or an attack predominantly focal.

Site of Preference.—*In syphilitic productive mesaortitis, the site of election is the first portion of the aorta* and the patchy involvement may be sharply delimited. If the arch itself is involved, only rarely does the Sharp delimitation.

process extend beyond its transverse portion though the *terminal* thoracic (supradiaphragmatic) segment is occasionally an initial site.

Luetic mesaortitis is so steadfast in its seat of election as almost to exclude syphilis as the cause of such a lesion in other regions.

Mesaortitis luetica may be said to be limited, practically, to the adventitia and media.

Primary changes.

Pathology.—The disease begins in the perivascular tissue and extends inward along the course of the nutrient vessels (vasa-vasorum) whose sheaths

Line of march.

are seats of marked small-cell infiltration, consisting chiefly of lymphocytes and plasma cells.

Within the infiltrated zones localized necrosis occurs, and in these areas groups of the *Treponema* may be demonstrable.

In the media, areas of complete destruction and solution of fiber-continuity may occur.

In active cases the zones of necrosis, reparative fibro-plastic activity and actual fibrosis may co-exist.

In the peripheral areas giant cells are found.

The early involvement of the vaso-vasorum and the direct action of *Treponema pallidum* and its toxin are the chief factors in the peculiar "line of march" of this process and the patchy distribution of its lesions.

The intima may escape wholly the *destructive* process and merely show some late fibrosis and possibly an irregularly distributed subendothelial proliferation.

Macroscopic appearance.

These microscopic changes are more or less clearly reflected in the macroscopic appearance of the inner surface of the affected portion of the aorta. Curious opaque, succulent, circular elevations, or tubercles, alternate with or are irregularly disseminated among sharply defined (punched out) depressions and pits, these often showing some translucency at their bases and a peculiar "silky crinkling" of the endothelial lining.

Many investigators have found the *Treponema pallidum* in the affected tissues.

The Francis Welch Aortitis.—*Such is the luetic mesaortitis of Francis H. Welch, sometime Professor of Pathology at Netley, who first described its important features, microscopic and macroscopic, in* 1875 *and asserted strongly its luetic origin.*

Long miscalled.

Long afterward, Döhle, Heller, Benda, Chiari, and many others, refined the investigations of Welch. Somewhat ungraciously, Continental, American and even certain British writers refer to this "*chronic productive syphilitic aortitis of Francis Welch*" as the "*Döhle-Hellersche Aortitis.*"

Importance of location.

Inevitable Sequences.—It is manifest that both the symptoms and results of this condition will vary greatly with the site of the lesion. The nearer the site of the inflammatory process to the aortic ring, the greater the damage and the more pronounced the symptoms.

Dilatation or aneurysm.

Obviously the weakened walls tend to yield, and upon the question of excessive focal activity, or a relatively extended diffusion of the destructive changes, depends the production of aneurysm on the one hand, or a mere diffuse dilatation,

or a fluoroscopically demonstrable increase of pulsatory excursion upon the other.

Aortic insufficiency.

It is evident that the commonest type of involvement, i.e., that of the first portion, must tend to weaken and enlarge the aortic ring. In 80 per cent. of the cases of luetic mesaortitis, such a secondary aortic insufficiency is established.

Possible mitral involvement.

The valves themselves are usually more or less wrinkled, thickened and shortened, extension to the anterior mitral segment is not uncommon, and even a mitral stenosis may result.

Angina pectoris.

Inclusion of the sinuses of Valsalva and the coronary apertures is almost inevitable in such cases, *and the wonder is that in only 10 per cent. of the cases does major angina pectoris occur.* The coronary orifices may be reduced to pin-point size in such instances. *It is almost equally strange that in only 10 per cent. do we find true aneurysm.*

Misleading Statements.—It is generally stated that in this disease blood pressure is normal, the heart but slightly affected, dilatation slight, hypertrophy laggard, and compensation poorly achieved in the instances of aortic valve involvement.

Such sweeping statements are most misleading. High systolic pressures and a greatly increased pulse pressure accompany all free aortic regurgitations and renal impermeability is, at least, a relatively common complication and may produce excessively high systolic and diastolic pressures, which exercise a most disastrous effect upon a weakened artery or an inadequately nourished or definitely toxic and degenerated myocardium.

The assertion, oft repeated, that the heart is but slightly affected when the coronaries are free and aortic regurgitation absent must, of course, often be true, but frequency of exceptions must be asserted by the author on the basis of personally observed cases of aneurysm of the arch, no less than our growing knowledge of the relative frequency of early and late luetic myocardial involvement, although the former and indirectly, no doubt, the latter form in great measure are checked by the early institution of antiluetic treatment.

An erroneous assertion.

The assertion of a famous German clinician that good compensation in an aortic regurgitant lesion proves it to be non-syphilitic, is certainly erroneous, for the author has seen several instances of excellently maintained cardiac reserve over long periods in men doing hard manual labor and carrying luetic aortic incompetence.

A striking fact.

Importance of Early Recognition.—*It is strikingly true that once the break in compensation is permitted to occur under our present laggard diagnosis and therapy, its repair is extremely difficult and seldom more than partial and temporary.*

Early cases responsive.

As stated previously, early diagnosis and treatment are more essential in this than in any other cardiovascular ailment, and these hearts often prove as responsive to rest, with mercury or repeated fractional doses of salvarsan, as they are resistant to digitalis in their terminal stages.

They are not especially resistant to this drug in the lesser myocardial decompensatory periods and this fact should be known and acted upon.

Laggard Hypertrophy.—With respect to retarded and sluggish hypertrophy it is evident from the nature of the process that only in cases of involvement of the aortic valve is there with constancy any necessity for decided hypertrophy and equally obvious that the *relatively gradual* produc-

Readily explained.

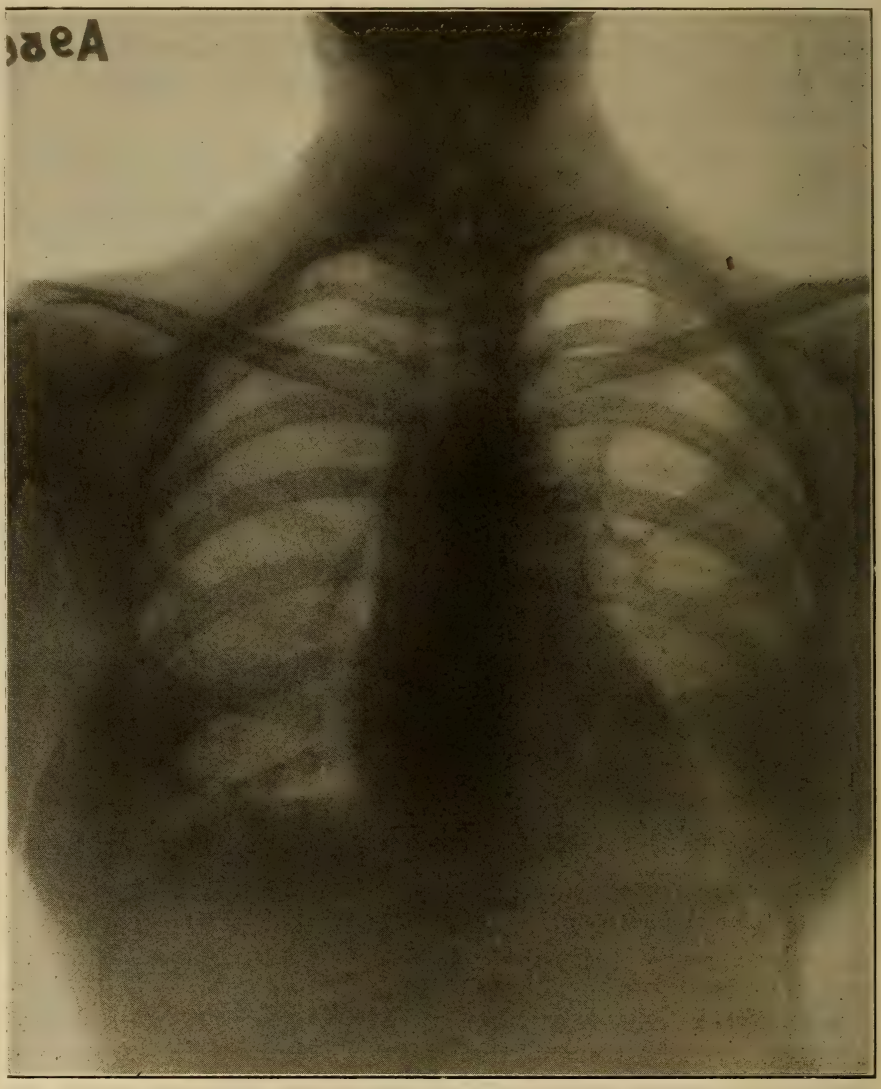


FIG. 410.—Combined mitral and aortic insufficiency (typical). Marked dilatation of arch suggests syphilitic mesaortitis with extension from aortic to mitral valve.

tion of free regurgitation would limit primary dilatation and slow the rate of hypertrophic changes.

GENERAL SYMPTOMS.—Space permits but a brief review of the symptoms of this important condition. Fortunately, they for the greater part are those of simple aortic regurgitation and to a lesser degree those of

angina pectoris, diffuse dilatation of the arch, and aortic aneurysm, which are dealt with fully elsewhere.

Luetic mesaortitis in its early phases is peculiarly a disease of subjective expression and this assertion applies, measurably, even to the cases of the aortic regurgitant type.

The first matter to be considered, therefore, is the proper evaluation of its subjective symptoms upon which so much stress has been laid elsewhere in this volume.

Pain and Discomfort.—*The character and degree of pain encountered depends upon the presence of several factors, chief of which are: (a) The presence or absence of true aneurysm. (b) The actual degree of hyperdistention or distensibility and diminished aortic reserve. (c) Disease of the coronary arteries, and (d) myocardial weakness from any cause whatsoever.*

Overstretching, spasm, and overload.

One must remember that the chief sources of pain in hollow automatic muscular viscera are: (a) Excessive overdistention. (b) Extreme spasmodic contraction. (c) Sudden interruption of the intrinsic blood supply, and (d) effort that exceeds muscular reserve, i.e., obligatory overstrain with underlying weakness and insufficiency.

An exploded fallacy.

That cardiac pain necessarily or inevitably arises from coronary sclerosis of itself cannot be maintained in the light of modern investigations showing extensive sclerosis without angina pectoris and typical "heart-pang" in the entire absence of sclerosis.

Coronary insufficiency.

That a temporary insufficiency of the intrinsic myocardial blood supply may result in weakness and pain, especially under emotional stress or effort, is certain, and is readily explained by the instantaneous reflection of blood shortage in a heightened myocardial irritability and narrowing of the field of painless muscular response.

Extracardial pain.

Pressure Pain.—One additional cause of pain, extracardial in its nature, may be encountered in aortitis with decided dilatation or actual sac formation, namely, *pressure*, and it should be remembered that luetic aneurysm, by reason of its preferred locations, is especially adapted to the production of pressure symptoms related to the diverse and important structures which traverse the mediastinum. (See "Aortic Aneurysm.")

Pain alternatives.

The commonest source of error in relation to aortitis is the failure to remember that early in any case of cardiovascular insufficiency the clinical meaning and significance of a mere sense of constriction, oppression, or crowding, may be exactly the same as the vice-like gripping or the rending tearing pain of major angina pectoris. Of no less importance is the misleading fact that, frequently, anginal pain and discomfort are maximal in the epigastrium or over an even wider upper abdominal area.

Angina pectoris miniatures.

As the author has pointed out frequently of late years, significant miniature replicas of angina pectoris may occur, and the cardiovascular pain-complexes of vital importance are often mere fragments or, at best, imperfect mosaics of the classical picture.

Pain often absent.

Many or, indeed, most of the cases of luetic, rheumatic, variolar, scarlatinal, influenzal or gonococcal aortitis, may wholly lack severe pain, and he

who waits for it to appear in its typical form will accomplish little for his patients.

Radiating referred or transposed.

One must recall also the fact that aortic pain of severity may or may not radiate from mid-sternum, or mid-sternum and epigastrium, to the left arm, shoulder, wrist or fingers, to the neck or the roots of the lower molars. It may be both left- and right-sided, or, rarely, affect the right side alone, involving in its cephalic radiation the same (brachial plexus) distribution wholly or partially in each instance.

Dyspnea.—*No ailment presents more striking and dramatic dyspneic pictures than may aortitis in its advanced stages, but in none should one seek more carefully for the earlier and far more important, lesser or masked manifestations.*

Its most extreme forms are, (*a*) cardiac or aneurysmal asthma, often absolutely indistinguishable from bronchial asthma, unless suggestive associated- or interval-pain and characteristic physical signs are present; (*b*) laryngeal spasm, a most distressing symptom usually due to pressure upon the recurrent laryngeal nerve, and; (*c*) an abruptly enforced orthopneic dyspnea which instantly and imperatively brings the recumbent patient to a sitting or even standing posture, and, in the author's experience, is associated usually with a *conviction* of impending dissolution, suggesting its relation to angina pectoris.

As stated repeatedly in this volume, instant death may result from a natural but mistaken effort on the part of the attendant to *enforce* the recumbent or semirecumbent posture in such instances.

Another interesting variant of obligatory orthopnea is that which forces the patient to lean far forward day and night.

Not peculiar to aortitis.

None of the preceding or following forms of dyspnea or pain are peculiar to luetic aortitis. They may be encountered frequently in other forms of cardiovascular disease, in mediastinal tumors of almost any sort, and especially in progressive enlargement of the mediastinal glands in Hodgkin's disease.

Of far more value in relation to *early* diagnosis are the minor dyspneic variants *when persistent or frequently recurring.*

Among these are: (a) Slight exertion dyspnea. (b) Inability to walk against a wind or in cold air without inducing breathlessness or precordial oppression. (c) Excessive sensitiveness to a close atmosphere. (d) Frequent sighing. (e) A mere subjective sense of deficient lung ventilation. (f) Inability to hold the breath on command or at will.

Such symptoms may precede by months or years the more serious manifestations of arterial or myocardial insufficiency.

Of cardinal importance.

Narrowing the Cardiovascular Field of Response.—*With respect to both physical and mental exertion, one may perceive as one of the earliest symptoms of this as of other cardiovascular lesions a narrowing of the field of effortless, fatigueless response.*

A common error.

So marked may this be and so striking the nervous symptoms as to lead physicians in many instances to diagnose and treat the condition as a "neurasthenia," that bastard euphemism for "don't know."

Physical Signs.—With respect to the physical signs little need or can be

said without entering into a useless repetition of the descriptions of aortic regurgitation, aneurysm, aneurysmal dilatation, or, angina pectoris.

Extreme value of the X-ray.

Roentgenoscopy and roentgenography afford important information. Both the screen and plate should be used, with sagittal and oblique exposures, and, in the absence of either actual aneurysm or diffuse dilatation, the quality and outline of the aortic shadow itself and any increased range of pulsatory excursion should be carefully noted.

FIG. 411.—(See Fig. 412.)

Dulness *en casque.*

Percussion.—This is of slight value relatively but one should always test the note over the manubrium, third right interspace and third rib and the second right interspace for the dulness "*en casque*" of Potain, and inspect and palpate carefully for expansile pulsation.

The split "second."

Auscultation.—The character of the second sound as heard over the third left interspace may be of the utmost value before any considerable

degree of aortic leakage is established. It may be blurred and murmurish, *or remarkably and suggestively clear and intense*, and one of the most important variants in the author's experience has been the split second sound, often heard over the third left interspace near the edge of the sternum, or

FIG. 412—.One type of luetic diffuse dilatation of the aortic arch (2d and 3d portion). Strong resemblance to actual aneurysm as in Fig. 409. Correct diagnosis shown in Fig. 412 in which the oblique method was employed. First exposures several months before showed no arterial change though the same enlargement of the heart existed together with the "fatty tail" at the left cardiodiaphragmatic angle, indicating fatty overgrowth. Patient highly "*neurasthenic*" at this time and showing some subjective signs of limitation of myocardial reserve. See Fig. 411.

over its corresponding left half at the same level, as described by him several years ago. This may be present persistently or intermittently long before the true murmur appears and may later alternate with the short imperfect diastolic bruit even during a single clinical *séance*.

Much importance is to be ascribed to a "*clanging*" second tone, for although it can hardly be regarded as in any sense pathognomonic, or as of great importance *differentially*, certainly it suggests when present aortic dilatation or abnormal dilatability, a very important link in the chain. An important sign.

The same may be said of palpation of the lengthened aorta in the suprasternal notch when this is accessible.

Obviously the fundamental suggestive factors are:

(*a*) *Aortic aneurysm involving the first portion, or the transverse portion of the arch.*

(*b*) *Diffuse dilatation in the same areas.*

(*c*) *Aortic regurgitation first becoming manifest in individuals above the age of 30 or even 25, and in some degree all aortic regurgitations not directly attributable to other known causative illnesses.*

(*d*) *Symptoms suggestively mediastinal in origin.*

(*e*) *Angina and dyspnea, major or minor, frank or masked.*

(*f*) *Subjective manifestations of impaired reserve.*

(*g*) *A positive Wassermann or luetin test or frank evidences of syphilitic infection.*

(*h*) *A favorable response to proper antiluetic medication.*

It should be borne in mind that demonstrable peripheral arteriosclerosis present or absent is of no importance in relation to diagnosis.

A man may drop dead from luetic mesaortitis when every accessible artery is as soft as a baby's.

Premature Assumptions.—A few words of caution may not be misplaced with reference to the diagnosis of a condition of most extreme interest and importance. It must be remembered that exceptions exist to every rule and that before one jumps to the conclusion that he is dealing with cardiovascular syphilis he must first of all be sure that the patient reacts positively to the Wassermann or luetin tests, or to conservative therapeutic procedure, and that the former tests have been made by men thoroughly competent to perform and to interpret them.

Many "Wassermann Tests" Valueless.—*In the case of the Wassermann test it cannot be stated too frequently and emphatically that no one save an expert serologist can be trusted to do accurate work and that a vast number of reports, now made the basis of opinion and action of the utmost importance to the individual affected, are not worth the paper they are written upon.*

A negative Wassermann test must not be permitted to override positive data of importance nor should any single negative test be accepted under such conditions. It must be remembered also that the mere fact that a Wassermann co-exists with angina pectoris, or aortic leakage does not absolutely prove that the lesion is luetic, though the chance of misinterpretation is trivial in such an instance.

It should also be stated that experience has proven that Ehrlich erred in sounding a warning against the active treatment of luetic vascular lesions. They should not only be treated but any measures should be instituted as early as possible and repeated as may be necessary. According to the A fallacy. Value of timely treatment.

opinion of the author, mercury and to a less degree the iodides are likely to prove quite as valuable and possibly more so than salvarsan, the effects of which seem to be somewhat fleeting on the one hand, or, on the other hand, excessive, if the drug is used in full doses rather than by the fractional method.

SUMMARY

Great increase of knowledge.

1. *The discovery of Treponema pallidum, perfected methods of demonstrating it in body tissues, the development of the Wassermann and luetin tests, and careful and extended studies based upon these, have resulted in an exact knowledge of the wide dissemination of syphilis and its enormous importance as a factor in the causation of diseases of extraordinary diversity.*

2. *We now know that the term "cerebro-spinal syphilis" embraces a large number of diseases in which its presence was formerly only assumed or wholly unsuspected.*

Seat of election.

3. *It now appears that to an extraordinary degree, the mortality of the disease depends upon its vascular ravages which are proven fundamental in many ailments of strikingly diverse complexion.*

4. *It may assume any one of the various forms of sclerosis, endarteritis or myocardial degeneration, but presents, in most instances, in the earlier stages of the latter, certain characteristic lines of attack and modes of histologic expression.*

5. *The most characteristic of these is the productive mesaortitis of Francis H. Welch, extraordinarily prevalent, almost constant microscopically in a more or less developed form at autopsy in every case of death from proven syphilis, and the actual cause of death in 50 per cent. or more of such cases.*

Aortic localization.

6. *This disease shows a peculiar affinity for the first portion and arch of the aorta and tends to assume at the root of that artery a characteristic line of march which in 80 per cent. of the cases results in the establishment of secondary aortic regurgitation of a peculiarly progressive and intractable type, once it reaches the stage of frank myocardial insufficiency.*

Aneurysm and "heart pang."

7. *In 20 per cent. of such cases of the advanced type, true aneurysm or frank angina pectoris major, occur in about equal proportion.*

8. *The leading symptoms, aside from those of aneurysm and actual angina pectoris, consist of pain, dyspnea, diffuse dilatation of the aorta, and progressive crippling impairment of vascular reserve.*

Silent cases.

9. *Many of the cases are wholly silent and the symptom complex may be blended, incomplete, or so misleading, as to be most readily misinterpreted.*

Reform imperative.

10. *Only through recourse to more rational methods of early diagnosis, dependent upon the proper recognition and weighing of minor symptoms, subjective and objective, may these cases be detected early enough to render specific treatment properly effective.*

11. *The utmost importance attaches to the Wassermann test, but only when it is in the hands of an expert serologist.*

Damaging errors.

12. *The improper use of this test and the false reliance placed upon it must vitiate many reports, and result in errors of omission and commission, damaging to the physician and a source of humiliation and injustice to the patient.*

13. *Finally.—A consideration of this topic emphasizes not only the importance of the individual cardiovascular luetic lesions, but also the terrible potency of syphilis as a cause of death at a period remote from the primary infection and through channels which so conceal its identity as to obscure the fact that it may yet come to be called the "Captain of the Men of Death."*

Lethal potency.

ARTERIOSCLEROSIS

ATHEROSCLEROSIS.—Definition.—A chronic, inflammatory degenerative disease of the arteries, either generalized or more or less localized, slowly but inexorably progressive, and tending to regional predominance.

It assumes one of three forms, viz.: nodose arteriosclerotic, a senile type, or a more or less distinct luetic variety.

In all cases the dominant change in the arteries is a fibrosis and the clinical results are diminished elasticity, palpable thickening, a tendency to diminution of caliber and lessened vascular reserve.

Etiology.—Sclerosis of the arteries suggests an overdone effort on the part of nature to forestall or repair the damaging effects of chronic toxemia, such as that induced by renal disease, lead, gout, infections of various sorts and *especially syphilis, and cryptogenetic local sepsis,* together with auto-intoxication, chronic alcoholism, emotional strains and excessive overwork (bodily or mental) carried on under unfavorable conditions.

Weakness in strength.

The summation of minor, more or less transient, periods of *toxemia* with or without associated arterial hypertension, or persistent hypertension whatever the cause, are doubtless the chief factors in causation.*

In aortic regurgitation, whether syphilitic or rheumatic in origin, the enormous, persisting, rhythmic variations in arterial tension must be at least a factor in the promotion of complicating aortic changes.

Heredity.—It cannot be doubted that heredity plays a large part in the occurrence and age incidence of many cases. The individual is as old as his arteries and the arteries of the ancestors determine to a large degree the longevity of their descendants.

Physical Work.—*The part played by hard physical labor alone has doubtless been overestimated in the past, though when combined with other untoward conditions it is doubtless of importance.*†

Interstitial Nephritis.—This kidney lesion is invariably associated with a more or less generalized sclerotic change, even to arterio-capillary fibrosis, and in these cases the sustained excessive vasoconstriction and, to a small degree, the sclerotic change in the vessel itself, combine to produce very high sphygmomanometric readings. In fact, nearly all cases showing a systolic

* It is a well-known fact that variations in blood pressure *normally* attend hard physical labor, just as it is certain that this involves a multitude of radical and suddenly induced changes of rate and force, but if the machine is sound, fuel and lubrication alike perfect, the machine runs on without damage even though the blood pressure varies from time to time from a systolic level of 125 mm. Hg. to 170 or even more.

† In occupations involving the daily exposure of the hands or arms, legs or feet, to violent changes of temperature, one may see decided localization of atherosclerotic or productive (obliterative) endarteritis in the portions of the body so exposed.

blood pressure persistently above 160 and a low "pulse pressure" yield other clinical evidence of morbid change in the kidney.

Conversely, a type of kidney (the arteriosclerotic kidney) is encountered as an apparent result or concomitant of primary arterio-capillary fibrosis.*

It is evident that serious arterial degeneration may exist within, without visible or palpable outward signs, yet fortunately, in most instances, some of the accessible arteries of the body reveal significant changes.

Arterio-sclerotic kidney.

An exact separation of the senile arteriosclerotic kidney from that of chronic diffuse arteriolar (interstitial) nephritis is sometimes impossible though it is probably true that the high blood pressures characterizing the more advanced stages of the latter are not reached in the former.

A deadly combination.

Vicious Complication.—*The combination of arteriosclerosis and hypertensive renal disease constitutes a most damaging combination inasmuch as it presents chronic toxemia and excessively high blood pressure coincident with rigid arteries, a toxic state of the myocardium and increased demand upon the heart.*

Degree and distribution variable.

Distribution of Lesions.—One seldom finds either a universal arteriosclerosis of uniform grade, or a regional sclerosis, without *general* changes of some degree. Both the degree and distribution of the pathologic processes are often strikingly irregular and in many instances affect chiefly those portions of the body which are most directly concerned in overwork in connection with chronic toxemia. In the brain worker the cerebral arteries may suffer most; in the day laborer predominant involvement of arteries of the upper extremities is common. Little that is positive can be known of the actual distribution *ante-mortem*.

Aging.

Physical Signs.—Inspection.—A more or less characteristic outward change in individuals with arteriosclerosis is that known as "aging" and this is peculiarly striking in certain cases of the "precocious" type in which one sees slight or marked senile changes in the skin and in the general appearance of the juvenile or relatively young patient.

Precocious types.

The most striking evidence, however, usually, is the appearance of readily palpable superficial *arteries* in the forearm, at the wrist, or in the region of the temporals. Outward evidence of this kind may be reënforced in cases of an advanced type with arterial rigidity, by a peculiar tortuosity of these vessels and in the brachial or especially at, or just above, the bend of the elbow a peculiarly *vermicular* lengthening and shortening of the arterial curve may be apparent. This may be so marked as to simulate the jerky throb of

Vermicular pulsation.

* The development of a chronic Bright's creates a condition of impaired renal permeability and more or less constant or persistently recurrent, minor toxemia such as would tend to promote or even cause an arteriosclerosis, and its co-existence leads certainly to progressively increasing tonic vaso-constriction of damaging degree.

We are still in the dark as to the actual time relationship between the onset of interstitial nephritis, or even the arteriosclerotic kidney and that of a generalized arteriosclerosis.

A growing appreciation of the importance of the cryptogenetic foci of chronic, chronically remittent, or intermittent, streptococcic infection seems likely to place under this head many chronic degenerative conditions now lacking any specific etiology and the three conditions may sometimes be proven to have a common chief cause.

aortic regurgitation with which lesion it is associated in many instances so that either simulation or actual concurrence are common events. *Mere prominence of the temporal arteries does not prove a sclerosis and is very common.*

In extreme cases the veins of the forearm or back of the hand may show a sclerosis on palpation (*phlebosclerosis*). Phlebosclerosis.

Retinal Arteries.—To the skilled observer his ophthalmoscope often reveals arteriosclerotic changes in the vessels of the retina at a comparatively early stage (see "Ophthalmoscopy"), but otherwise our eyes cannot be trusted in the diagnosis of this condition, even when external, and we must resort to palpation.*

Palpation.—An artery which, when the pulse is obliterated by proximal compression by one finger, can be rolled under the finger or distinctly outlined as either a rigid or a *soft* tube, is the seat of sclerotic changes and if plaques are formed the sensation of trachea-like inequalities imparted is extremely characteristic (windpipe arteries). "Goose-craw arteries."

One should not be content to examine the radials but should carry the investigation to other accessible arteries and if any complaint of rheumatic or "gouty" pain or lameness is made by the patient one should carefully palpate both the dorsal pedis and posterior tibial arteries for any deficiencies of pulsation such as may be associated with intermittent limping (*"intermittierende Hinken"*) or may suggest the possibility of oncoming senile gangrene. Intermittent claudication.

These circulatory evidences of what may prove to be a dangerously advanced, but otherwise concealed peripheral, *obliterative endarteritis* have been relatively frequent occurrences in the author's practice.†

Auscultation.—The aortic second sound is usually accentuated and may be markedly so if, as so often happens, the aorta itself is diseased. In such cases one often encounters in addition a systolic murmur transmitted into the vessels of the neck, precisely as is the case in aortic stenosis, and oftentimes to be differentiated positively from that lesion only by the presence of a well-defined and accentuated second sound in the aortic area. Accentuated aortic 2d.

The author has found the maximum of the murmur and of the accentuated second sounds alike, in such cases, is usually *not at the second right intercostal space but one space higher on the same side.* It must be remembered that after the fourth decade sclerotic changes in the aorta or aortic valves are progressively common and that the aorta is one of the selective sites of early luetic invasion and belated clinical expression. (See "Cardiovascular Syphilis.") Area of maximal accentuation. Lues.

Mesenteric Arteriosclerosis.—This interesting condition, said to be most

* The traditional, specific association of general arteriosclerosis with the "*arcus senilis*," so often seen as a whitish ring more or less completely encircling the cornea at the corneo scleral junction, is a medical myth.

† In a number of instances a *cardiovascular breakdown* and death have followed the usual resort to "Spas" for the relief of what was supposed to be rheumatism, or, in a few instances, chronic sciatica.

In all of these the deficiencies of the pulse in the foot or ankle were readily recognizable, and a history of long sustained *subjective symptoms* of impaired reserve was seldom lacking.

Paroxysmal splanchnic spasticity.

frequently associated with interstitial nephritis and high blood pressure, is rare and can seldom be positively diagnosed, unless we assume its presence in all such cases showing *very high* systolic sphygmomanometric readings and a low "pulse pressure," or so translate attacks of severe abdominal pain or discomfort of a paroxysmal nature, often peculiarly relieved by the sitting or erect posture and increased in recumbency. It is asserted, wrongly, that excessive systolic and diastolic readings and low pulse pressure do not occur in arteriosclerotic cases until the splanchnics are involved.

Obligatory change of posture.

In some cases associated with terminal interstitial nephritis or disease of the aortic valves this distress is so marked *either precordially or abdominally* as to lead the patient to disobey the most stringent orders as to absolute physical rest and to even leap from the bed to the floor or, at the least, abruptly assume a sitting posture in bed.

In such crises as in the ordinary major angina pectoris, the sphygmomanometer may reveal a sharp rise in blood pressure.

The author is convinced that in most of these, the crisis should be regarded as an epigastric or abdominal *maximal expression* of an angina pectoris. Cases of coincident or *alternating* paroxysmal thoracic and abdominal pain are common in major attacks and the tendency of decompensated hearts to refer their pain to the upper abdomen is well proven.

Dangerous discipline.

As stated previously, in such instances it is extremely unwise to attempt to force a patient back into the recumbent position as sudden cardiac death has resulted in many instances from such mistaken discipline.

Caution.—Crises of spastic vasoconstriction, whether of abdominal or precordial localization are characterized chiefly by increased distress in recumbency, intense pain, tympanites and an abrupt and excessive increase of any preëxisting arterial hypertension.*

Both may be initiated by indiscretions in diet and consequent flatulent overdistention by physical fatigue, anger, fear, joy, or other emotional disturbances.

In true abdominal crises, it is probable that a spastic contraction of the vessels results in ischemia and spastic intestinal colic. This ischemia is initiated, no doubt, in many instances, by mere *excessive* flatulent distention which greatly diminishes mesenteric blood-flow in *normal* vessels.

Mesenteric arteriosclerosis is rarer, however, than was formerly supposed and the frequency of true spastic crises of the abdominal type probably has been overestimated greatly.

Too ready an acceptance of paroxysmal spastic vasoconstriction of the mesenteric arteries involves the danger of serious or fatal neglect of far commoner, more important, and oftentimes lethal lesions of the abdominal viscera and of the heart and its intrinsic arteries.

Limited Auscultatory Field.—*It will be understood that auscultatory signs in arteriosclerosis are practically limited to those cases in which there is aconjunction of high pressure and sclerotic changes in the aorta or its valves.*

* The author has measured an increase to 300 mm. Hg. in a case showing just before the attack 190. The usual rise is 50 mm. plus.

In the latter case there is often an obtrusively visible and palpable pulsation in the jugular fossa most evident during the act of swallowing which may falsely suggest aneurysm and this erroneous influence may be further strengthened by the existence of a fairly well-defined pulsation in the second right intercostal space. The X-ray picture in such instances is likely to show a marked lengthening of aortic curves and a low position of the heart closely simulating the condition present in an aneurysm of the arch.

Jugular fossa pulsation.

X-ray.

It constitutes nevertheless one of the indications of diffuse dilatation of the aortic arch. (See "Cardiovascular Syphilis.")

Another misleading feature with respect to the same lesion is the occasional occurrence of decided inequality and lack of synchronism in the carotid and subclavian pulse, this indicating, usually, a decided aneurysmal involvement of the transverse portion of the aortic arch *but occurring also in its absence in many instances*. In such cases both the carotid and subclavians are likely to be palpably sclerotic and to show exaggerated length and abnormal curves.*

General Symptoms of Arteriosclerosis.—*These are extremely variable, oftentimes indefinite, but in general they are those of a snail-like progression of circulatory insufficiency together with a progressive narrowing of vascular fields and of arterial and cardiac response.*

Cardiovascular insufficiency.

We are all familiar with the senile type which results from an obliterative cerebral endarteritis with the impaired, chronologically reversed, memory and clouded intellection, diminished reasoning power with increased tenacity of opinion and intolerance of opposition, pronounced mental confusion, and even incoherence, slovenliness and degenerate acts.

Senile decay.

Early Cerebral Symptoms.—Still earlier symptoms, also of the cerebral type, are irritability, headache, vertigo, and readily induced fatigue. Day drowsiness and disturbed sleep are also prominent features in some instances and the early waking in old people is more or less characteristic of all cases in which the cerebral vessels are involved, *but by no means limited to this form of circulatory deficiency.*

These "early" symptoms as a whole are common to all types of cardiovascular decompensation though less constant in appearance and type in forms other than the arteriosclerotic.

Gastrointestinal Symptoms.—Disturbances of the gastrointestinal tract are common such as loss of appetite, capricious appetite, inordinate appetite, or indiscriminate gorging, flatulence and general dyspeptic manifestations, often of a misleading sort and very frequently to be referred to the heart rather than to the sclerosis itself or to the stomach.

Dyspeptic symptoms.

In cases of marked splanchnic involvement usually associated with renal changes one may encounter the painful seizures already described and these may be associated with a decided and dangerous tendency to meteorism and even, *possibly*, to acute dilatation of the stomach.†

Splanchnic crises.

* *Slight* inequalities are encountered very commonly in instrumental pulse records.

† As common diagnoses of the cause of sudden death "acute indigestion" and "acute dilatation" are merely the euphemisms of "don't know." In nearly every instance, undoubtedly, previously unsuspected cardiovascular disease is the actual cause of death.

Curious Cerebral Seizures.—Curious transient attacks of unconsciousness, monoplegia, pseudo-cerebral epileptic seizures and temporary disturbances of speech may occur suggesting localized vasoconstrictor spasm and ischemia, these oftentimes being associated, according to the experience of the author, with temporary but decided cardiac weakness and, not infrequently, a transient pulmonary congestion or edema.

Vertigo may be an exceedingly troublesome and intractable symptom and *deafness* is often marked and slowly progressive.

Pulmonary Block.—*Whatever tends to impair free respiration retards the pulmonary circulation and many pulmonary ailments actually reduce the capillary area or block both venous and arterial flow.*

Thoracic Ailments Commonly Associated with Arteriosclerosis.—Therefore, we expect to find a tendency to ultimate hypertrophy and dilatation of the right heart in such extreme chronic pulmonary or thoracic affections as emphysema, acute and chronic asthma, extensive chest deformity, as in kyphosis, chronic pleurisy, extensive adhesions, fibroid phthisis, or severe chronic bronchitis and a considerable number of old chronic phthisical cases of the ordinary type will show decided arteriosclerosis and cardiac changes. So also sclerosis of the pulmonary artery itself or of the coronary arteries (the right, chiefly) may be present in many such conditions as are noted above and all grades of right-heart cardiac insufficiency ultimately may appear.

Demands upon right heart.

Arterial changes.

Disturbing Factors.—*Several factors interfere with accurate diagnostic results in this connection.*

1. *The voluminous lungs of asthma and emphysema.*

2. *The fact that right heart hypertrophy and dilatation may be so great as to simulate combined left and right heart change, the apex-beat formed by the right heart being sometimes carried beyond the nipple line.* (See Heart Section.)

3. *The various changes in the position of the heart itself due to adhesions, contracted lung, mediastinal pleural or pulmonary growths, unilateral effusion and the like.* (See Lung Section.)

4. *The deformity and displacements due to kyphoscoliosis.*

Dyspnea, cyanosis, enlarged liver, renal stasis and edema of the lower extremities, are the striking symptoms in serious incompensation.

Attacks of cardiac asthma are not uncommon, pulmonary edema is frequently a terminal event and pulmonary infarct a common complication.

Kyphoscoliotic victims, who escape death from the local disease of the spine, die of right heart weakness in middle age or of acute pulmonary affections of which it constitutes a serious complication in at least 80 per cent. of the cases.

Rationale.—*It should be remembered that by no means all of the individuals carrying arteriosclerotic changes give evidence of them or suffer serious impairment of health by reason of their presence.*

In many instances the changes are so slight or their localization so fortunate as to produce no effect upon the health such as is clinically appreciable.

It is also true that arterial hypertension is often absent, seldom or perhaps never, extreme, by reason of the arteriosclerosis itself and that such rise as does persist is due chiefly to peripheral and not splanchnic sclerosis (as formerly

*believed), although these vessels may be the seat of extreme and damaging tonic or labile vasoconstriction. A decided, persistent, but seldom excessive pressure ultimately appears in about two-thirds of the cases of demonstrable arteriosclerosis, and to increase of blood pressure, rigidity of the vessels and diminution of their caliber, there may be added an increased irritability, leading to excessive tonic vasoconstriction in the persistent presence of toxins. This is best illustrated in complicating chronic nephritis, or, visibly in cases of recurrent spasm in the legs (or occasionally the arms) in cases of intermittent claudication (intermittierende Hinken).**

Lesser Hearts.—The arteries are lesser and subsidiary hearts which in their normal condition are capable of regulating and to a certain extent, of diverting, blood flow, according to the passing needs of the human organism.†

Storage Power Plant.—The aorta by its elasticity converts what otherwise would be an intermittent stream into a continuous current through the elasticity of its walls which take up the primary excess of pressure represented by the systolic ventricular drive and so store and gradually release the energy represented by the individual systoles of the ventricle as to maintain a sufficiently uniform pulse pressure and that steady slow flow through the capillary deltas, necessary to the vital chemical exchanges of body metabolism.

Exactly as in the case of the heart, we find in an artery a certain amount of reserve force, indispensable to the proper maintenance of circulation.

Arterial reserve.

Sclerosis of the arterial wall diminishes the storage capacity or, *reserve*, of the aorta, and the excessive initial amount of the power transmitted through the ventricular systolic drive unduly *accelerates* the flow. The lack of stored power permits the pressure and the stream to *slow* with undue rapidity and thus the diminished elasticity tends to establish an abnormal variation between pressure and rate of flow in systole and diastole, *i.e.*, to increase the so-called "pulse pressure," and convert a relatively stabile and sustained continuous current into an intermittent or remittent flow.

In advanced arteriosclerosis, a marked or extreme disturbance of the marvelous automatic adaptive and compensatory action of the vasomotor mechanism and an inability to correct and balance localized or general inadequacy of blood supply, lead to a narrowed and depressed functional activity, to actual atrophy, or to necrosis (obliterative endarteritis) in local areas.

How greatly this need may be felt in the arteries of the great splanchic area (the "regulatory reservoir" of the circulatory system) and how vast an increase of peripheral resistance results from sustained excessive vasoconstriction of their own is evident.

Increased resistance means increased ventricular drive. This must be steadily maintained and emergency demands upon reserve must be gradually increased by reason of the breakdown in the regulatory vasomotor mechanism

* In "Raynaud's disease" the vessels are normal through the seat of spasm.

† "The heart gives to the blood stream its motion; the blood vessels, its apportionment." (Jacob Henle).

"The arteries are true hearts under a different form," (Senac).

and usually the added peripheral resistance due to the excessive variations in an already excessive blood pressure which accompany increased physical effort.

In the meantime the nutrition of the heart itself must suffer, perhaps only through the general depression of metabolism, but oftentimes because of the participation of its intrinsic arteries in the arteriosclerotic circulatory *debacle*. Responsive obligatory hypertrophy, increased work under unfavorable conditions, increased degeneration of the heart muscle and of the larger vessels, progressive increase of the element of dilatation and diminished myocardial reserve would roughly express the factors leading to the circulatory breakdown. This is often anticipated by death from intercurrent infection, apoplexy, or uremia, if the kidneys are involved, as is usually the case when excessive arterial hypertension is evident.

PAROXYSMAL SPASTICITY OF THE CORONARY ARTERIES AND ANGINA PECTORIS

With or Without Coronary Arteriosclerosis

CLAUDICATION OF THE HEART.—Etiology.—The etiology of coronary sclerosis is of course identical with that of the arteriosclerosis of which it is a part, but merits a special description because of its frequent association with major and minor angina pectoris and the far rarer cardiac aneurysms.

Angina pectoris.

The left heart vessels are chiefly affected save in emphysema and extreme kyphoscoliosis in which involvement of the right usually predominates.

Morbid Anatomy.—A peculiarly frequent feature of this condition is the arteriosclerotic involvement of the aorta itself at the point where the coronary arteries are given off and the tendency of those vessels to show sclerotic changes at this point and at their branchings. This results in a diminished intrinsic blood supply and a *poorly maintained and illy-regulated* arterial pressure for the myocardium.

Important selective foci.

Obstructed flow.

The changes themselves are of the most varied description both as to their form and the degree to which their caliber is diminished in different areas.

Varying grades.

The associated myocardial degeneration usually takes the form of a dominant cardiosclerosis more or less diffused and there is a decided tendency to obliteration of the lesser branches and replacement of muscle fibers by scar tissue. This may result, to a greater or less degree, in the condition known as *myomalacia cordis* and lead even to *cardiac* aneurysm.

Myomalacia cordis.

Cardiac aneurysms.

In the presence of chronic recurrent infection there may also be changes due to a chronic myocarditis of the parenchymatous type. A true endarteritis is uncommon and if present is usually syphilitic.

Parenchymatous degeneration.

Symptoms.—For long periods no symptoms may arise and such as first appear are manifested usually only under excitement or such exertion as makes abrupt demands for increased coronary flow. Inasmuch as the myocardial blood supply progressively diminishes and the irregularity of its distribution slowly becomes more marked, the arteries become concurrently more irritable, and less responsive to the heart's demand, and the field of unconscious, effortless and painless cardiac response is greatly diminished.

Long latent and silent.

Even in this stage the general symptoms usually are merely those of minor myocardial insufficiency, as a rule unattended by marked dilatation or by any murmurs save those of associated disease of the aortic or mitral valve or the relatively common "pseudo-aortic-stenosis" murmur due to sclerosis of the arch and associated with a markedly accentuated second aortic tone most intense in the first or second right interspace.

The cardiac area may be much enlarged later and, in the terminal periods, show a marked dilatation both right and left and a peculiar outline somewhat resembling radiographically that of the percussion area of a pericardial effusion, evidently because of an extreme lack of general myocardial tonus (*"the heart of extreme general insufficiency"*).

The Pulse.—The pulse will vary greatly according to the stage of the disease and the nature of any associated lesions, but in pure coronary sclerosis *of marked degree* it tends rather to be weak than strong and is usually, but by no means invariably, arrhythmic.

Arrhythmia common.

As the disease advances any preëxisting arrhythmia tends to become confirmed and, with the progressive destruction of muscle fiber, a well-defined bradycardia sometimes supervenes which not infrequently corresponds to a period of immunity from any preëxisting angina or paroxysmal cardiac asthma and to the onset of typical and decided heart block.

Bradycardia and heart block. Luetic type.

If the block be complete and the pulse drop into the thirties such patients may show the complete clinical picture of the Adams-Stokes syndrome (see "Heart Block"). The lesser degrees of block (increased "a-c" interval, etc.) are naturally somewhat common in this ailment.

Inconstancy of Pain.—Many cases of well-marked sclerosis of the coronary arteries exist for years and terminate in death without any such severe manifestations of pain as cause serious complaint on the part of the patient. On the other hand, in many instances, *typical angina pectoris major occurs in connection with valvular disease, fatty degeneration and cardiosclerosis in which no material sclerosis of the coronary arteries is demonstrable at autopsy.*

Sclerosis *sine* angina.

Angina without marked sclerosis.

Nevertheless *decided* coronary sclerosis is commonly associated with attacks of precordial pain of a peculiarly atrocious and alarming character.

Angina Pectoris.— *Major Seizures.*—An attack of major angina pectoris is most dramatic and terrifying both to the patient and the onlooker.

Often without warning and, frequently, in the early morning hours between midnight and four o'clock, the patient is awakened suddenly, feels as if his heart were held in a vise and is overwhelmed by an inward *conviction* of impending death. He finds himself in an obligatory sitting posture which rigidly he must maintain. His terror-striken face is pallid, gray, and covered with cold perspiration; his breathing is shallow, but his pulse, though oftentimes feeble and sometimes momentarily lost at the radial, may be normal, markedly accelerated or distinctly slow, and arterial tension is in most instances decidedly or extremely high, shortly following the onset and throughout the attack. He may have originally clutched at the precordium and if so usually maintains that position supporting himself by the opposite hand, but

Time and abruptness o onset.

Fear of death.

Facies.

Pulse and tension variable.

Obligatory posture.

Immobility.

his whole attitude is one of intense suffering, extreme apprehension and rigid immobility.

Duration.

These attacks may at first last but a few moments and be very mild but tend

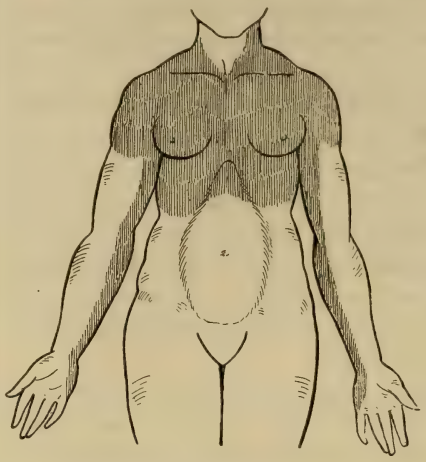


FIG. 413.

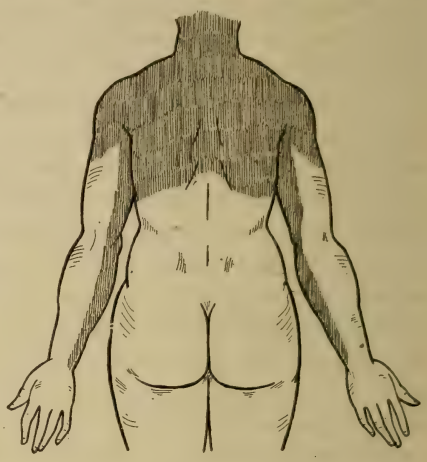


FIG. 414.

FIGS. 413 AND 414.—Relatively common area of pain and residual tenderness in extreme angina pectoris, especially in those with extremely high blood pressure. In one such case observed by the author the slightest touch applied to the skin overlying the sternum brought on a spasm of cough. (*After Henry Head.*)

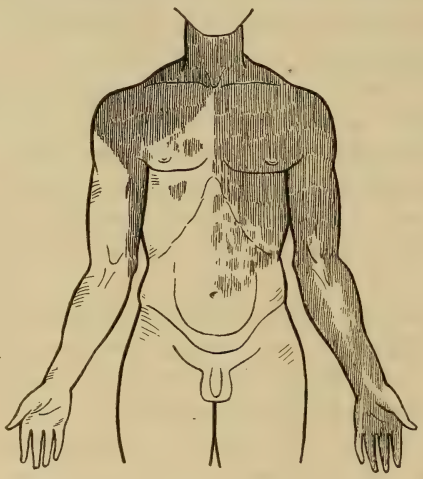


FIG. 415.

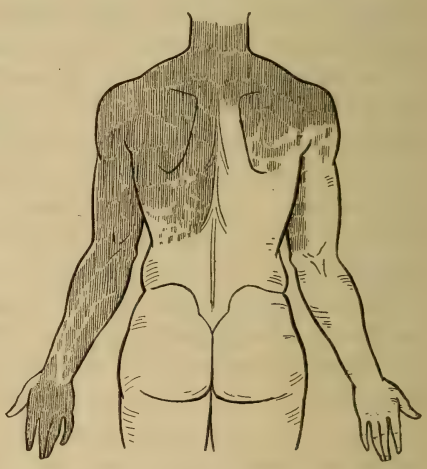


FIG. 416.

FIGS. 415 AND 416.—Another relatively common area of pain and residual hyperesthesia in angina pectoris major. (*After Henry Head.*)

*to increase in frequency and in some instances would last for hours without medical aid.**

Exciting Causes of Anginal Seizures.—*These attacks may and often do occur at irregular periods during the day, being precipitated by such causes as emotional*

* All such patients learn to have near at hand some means of ready relief or amelioration.

excitement, cold air, facing a heavy wind, physical overstrain, a distended stomach or intestine, straining at stool or sexual intercourse.

Pain.

Distribution and Degree of Pain.—Three features in connection with angina pectoris should be especially emphasized, viz.:

1. *The pain of angina pectoris major and minor may be, and often is, maximal at points remote from the heart.*

2. *Not infrequently more or less complete replicas of the major seizure have been observed by the author in cases of myocardial degeneration with or without coronary sclerosis or valvulitis and seem to be associated with the lesser degrees of dilatation and lack of muscle tonus and effective contractility.*

Potent factors.

3. *Any or all anginal pains may be accompanied by most misleading areas of coincident and residual hyperesthesia.*

Common Distribution.—In a large majority of cases of major angina the pain and hyperesthesia are referred to the left shoulder, inner aspect of the arm and over the forearm along the ulnar or radial distribution or over the entire hand. They may also be carried up the neck along the left carotid, may effect the right side exclusively* in rare instances or involve both simultaneously.

Important Variations.—*More interesting and important is maximal intensity of both pain and tenderness over the epigastrium and region of the gall-bladder, common as to the former, occasional as to the latter, sometimes misleadingly associated with antecedent or subsequent nausea or vomiting. In such cases there may be relative freedom from referred pain in the upper extremities or over the heart itself, but the latter is usually sufficiently involved to yield significant evidence upon close examination and inquiry.* (See "Pain in Disease of the Heart and Blood Vessels.")

Referred pain.

Simulates abdominal ailments.

Dangerously Misleading.—*Both its minor and major forms if attended by this distribution are dangerously misleading to the surgeon, as is attested by many futile operations upon the gall-bladder, stomach and appendix undertaken because of the dominance of pain, alone or with misleading associated tenderness, over these structures and neglect or undervaluation of the precordial manifestations.*

Misleading tenderness.

In several of the author's cases, vomiting has intensified the patient's suffering and added to the opportunities for misinterpretation. In another a violent agonizing cough attended each paroxysm. This was found to be a reflex from the exquisitely hyperesthetic chest wall, ceasing when the desperate clutch of the patient was released.

In all such cases the possibility of a lesion of the structure underlying the area of maximal pain must be considered even though the attack itself is cardiovascular in origin.

Caution in both directions is indicated.

Pal's splanchnic abdominal crises have been described under arteriosclerosis and these, so far as they have been observed by the author, for the

* This is suggestive of predominance of a predominant right coronary sclerosis or the rare cases of pulmonary insufficiency but is not *proof* of such change.

greater part have appeared to be merely cardiac anginas attended by dominant epigastric or upper abdominal pain-reference and localization.*

Exact nature unknown.

RATIONALE.—No thoroughly satisfactory explanation of the genesis of major angina pectoris has yet been firmly established.

Diminished "field of response."

Both in this form and its rarer substitute, *paroxysmal cardiac asthma*, which obeys the same laws of incidence but may be indistinguishable from spasmodic asthma, it is evident that in most instances there is a diminished and unstable myocardial blood supply, spastic vasoconstriction and an excessive diminution of the field of painless cardiac response.

Reasonable Assumptions.—*The night attacks come on usually during deep sleep and at a period when the enfeebled and rigidly limited coronary and general circulations are alike at lowest ebb, and any reflex asphyxial vasoconstriction might readily precipitate an abrupt localized ischemia. The day attacks seem rather to represent an abrupt unusual demand unsatisfied.*†

The result would apparently be the same in both cases, viz., a *relative* myocardial ischemia and a consequent loss of adequate painless contractility, intensified by reflex paroxysmal vasoconstriction.

An everyday parallel.

The author feels that in many such instances, as in any case of cardiac fatigue, the case is parallel to that of the spasmodic cramping of the calf muscles in sustained maximal overexertion, or, the spasmodic pain of deficient blood supply due to coincident arteriosclerosis and arterial spasm, such as is observed in intermittierende Hinken. The pain induced in a hollow muscle under conditions of abruptly produced ischemia may well be spasmodic and intense, as is shown by the dramatic and misleading onset of mesenteric infarction.

Hollow muscle pain.

Acute dilatation frequent.

In most of the author's cases, recently observed, the heart has been distinctly and in some cases surprisingly dilated during the attack and in those cases where an associated interstitial nephritis was present the blood-pressure reading at the height of the seizure was from 60 *to* 110 *or more mm. of mercury above the records preceding and succeeding immediately.*

Minor Anginas.—These have been discussed under the title of "subjective symptoms" of cardiovascular insufficiency.

It is illogical to assume that everything resembling angina pectoris, but not atrocious or typical, must be a pseudo-angina.

Replicas in miniature.

Some of the minor attacks are replicas in miniature of the major attacks here described. Others are incomplete, which is exactly what one would assume as probable from our present knowledge of the pain manifestations of hollow muscular viscera. A separate classification of purely nervous attacks of precordial distress under the title, "pseudo-angina pectoris," is most

Misleading tenderness.

* In three cases observed by the author throughout the attack the distress was at first distinctively abdominal, but after a few minutes became that of classical *angina pectoris*.

† The author has been fortunate enough to find several opportunities for unusually close observation of both the major and minor anginas and believes that too little attention has been given to the attacks that strike during sound quiet sleep or absolute physical rest and relaxation during the day.

In one patient he observed the onset of three such attacks under what should have been ideal conditions for their prevention if mental calm and physical rest might be so regarded.

unfortunate as tending to obscure the importance, and render less likely the recognition of minor or true anginas such as are not at all uncommon in nervous individuals who carry weak overstrained hearts. No one who has ever seen a major attack will be deceived by a mere hysterical seizure. It is one of the most pitiful, dramatic, and *convincing* of clinical pictures.

Prognosis.—If the attacks are severe, occur persistently and with increasing severity and duration, the outlook is bad, but intelligent well-to-do patients can oftentimes be taught to observe their own limits of safety, and under such conditions often may remain fairly comfortable under measures directed to the reduction of any existing dilatation and the strengthening of the general circulation.

Among the most difficult cases are those carrying the persistent high arterial tension and marked and widespread arteriosclerosis of an interstitial nephritis, yet even these under proper management often secure long periods of immunity.

ANEURYSM OF THE THORACIC AORTA

Definition.—A true aneurysm represents a local dilatation of an artery, due to the weakening effect of a chronic arteritis.

Etiology.—Aneurysm in this region is almost invariably due to the *syphilitic productive mesaortitis of Francis Welch* and any factor that *promotes* arterial degeneration on the one hand, and abnormally great vascular tension upon the other, may contribute to its progress. As is often said, the victim is usually one who has worshipped at the shrine of Venus, Bacchus or Vulcan, but the influence of the first of this triad is dominant in etiology.*

Devotees of Venus.

Intemperance in eating or drinking and physical and mental overwork or overstrain act as contributing causes, the first symptom in many cases dating from sudden, severe or prolonged muscular effort. Several cases coming under the author's observation have dated *their symptoms* from severe falls or a railway accident.

Traumatism.

It is quite possible that a congenital weakness of the vessels is a factor in the aneurysms of the young, but in all these cases it is difficult to believe that we can exclude a primary inherited degenerative process due to one of the chief factors originally mentioned.

The disease is much more common than is generally supposed and is a frequent cause of sudden deaths, which, in the absence of autopsy, are referred to other conditions, chiefly "acute indigestion," "acute gastric dilatation," and "hematemesis."

Difficulties Encountered in Diagnosis.—The use of the fluoroscope and the X-ray photograph has added greatly to our diagnostic resources and made it possible to detect aneurysm in its earlier stages. Otherwise no disease is more easily recognized in the presence of its classical symptoms, nor more frequently overlooked when these are absent. (See "Roentgenography.")

Valuable aid.

* The results of the Wassermann and luetin tests have greatly strengthened the belief that practically all such cases represent the effect of past syphilitic infection. This etiologic relationship of aneurysm on the vascular side to syphilis is practically parallel to that of locomotor ataxia on the side of the nervous system. (See "Cardiovascular Syphilis.")

Old divisions.

Clinical Divisions.—*The old clinical divisions were*: (1) *Aneurysm with signs and symptoms;* (2) *aneurysm with symptoms but no signs;* (3) *aneurysm with neither symptoms nor signs.*

Important points.

It should be remembered that (*a*) *classical symptoms develop only in certain cases and then usually when the terminal stage is reached;* (*b*) *that large aneurysms may exist without them;* (*c*) *that in cases of sudden death from this cause aneurysm*

Sudden death.

FIG. 417.—Luetic mesaortitis. An enormous aneurysm of the ascending portion of the arch. One of three such abnormal areas seen by the author. + = only point of marked pulsation. Patient died of external rupture succeeding necrosis of thoracic tissues.

is frequently unsuspected, both ante- and post-mortem, in the absence of an autopsy; (*d*) *that ordinary diagnostic resources are oftentimes unsatisfactory, inefficient and inadequate; and* (*e*) *that the fluoroscopic picture or X-ray photograph offers usually the only certain means of early diagnosis at our command.*

Statistics of Aneurysm.—*Roughly speaking,* 60 *per cent. of all recognized aneurysms are aortic, and* 85 *per cent. of these involve the thoracic aorta. Of these,* 90 *per cent. are saccular; from* 80 *to* 90 *per cent. affect the male, and* 60 *per cent. occur after the age of thirty and kill before fifty.**

The people whose habits most largely represent intemperance in food and drink and occupations which involve the maximum of such intemperance and

* No statistics of relative frequency, age incidence, or mortality can be more than approximate, by reason of the frequency of unrecognized cases of this internal and concealed group. The diagnosis of other aneurysms, even those of the abdominal aorta is simple.

of liability to syphilitic infection furnish the chief examples to the incidence and mortality of the disease.

Favorite Sites.—The root of the aorta, the junction of its ascending and transverse portions, and the descending arch represent the chief points of attack in the frequency indicated by their order.

Termination.—The peculiar situation of these tumors with a reference to adjacent and related structures readily explains their symptomatology and termination. *Death occurs suddenly in almost every instance because of rupture of the sac.* The blood may pass into the pericardium or adjacent

FIG. 418.—Same patient as shown in plate 349, showing retraction of area of percussion dulness, following prolonged rest.

pulmonary artery, into the superior vena cava, the esophagus, any one of the four heart chambers, the lung itself, the mediastinum, or, gush forth externally in certain massive aneurysms causing pressure necrosis of the overlying tissues. As regards relative frequency, the pericardium, pulmonary artery, and right auricle head the list of recipient structures in the order given.

Predominant pressure symptoms.

Symptoms.—*A pulsating and gradually but inexorably enlarging tumor, within a limited space filled with important anatomical structures means a possible predominance of pressure symptoms the relation of which to the structure primarily compressed, determines their nature. These may be epitomized as follows:*

Esophagus: dysphagia. **Trachea:** brazen cough (gander cough), dyspnea, stridor, bronchorrhea and hemoptysis (if the blood does not ooze from the sac itself). **Root of the lung, and the pleura:** symptoms suggesting phthisis, pulmonary collapse, pleurisy, etc. **Pericardium:** pericarditis.

Chest wall: localized, dull pain. **Nerve trunks:** neuralgic pains, paroxysmal and intermittent, often severe and intractable. **Pulmonary artery:** systolic pulmonary murmur, dilated right heart. **Sympathetic fibers:** dilated or contracted pupil, unilateral sweating or pallor. **Cardiac plexus:** anginal attacks. **Superior vena cava:** edema of upper extremity, cyanosis. **Thoracic duct:** marasmus. **Vagus:** dyspepsia, nausea, vomiting, dyspnea. **Phrenic:** hiccough, unilateral diaphragm spasm or paralysis. **Recurrent laryngeal:** hoarseness, aphonia, spasm or paralysis left cord, paroxysmal dyspnea. It must not be forgotten that any or all of these symptoms may be caused by mediastinal growths other than aneurysms and that the incidence of such symptoms is variable and irregular.

PHYSICAL SIGNS.—Inspection.—This must be both direct and *tangential* and one seeks primarily to discover any local bulging or pulsation.

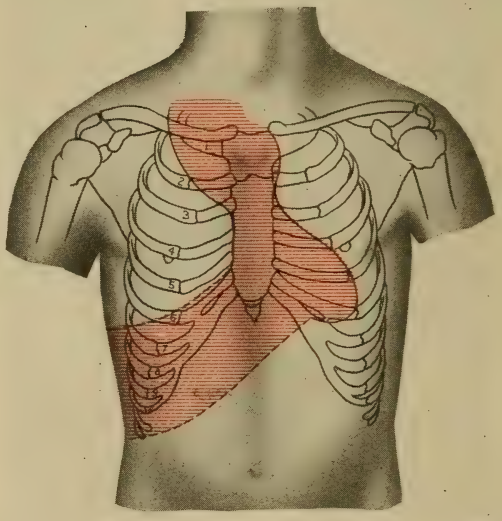


FIG. 419.—Aneurysm of the ascending portion of the arch. The aneurysm represented by the percussion outline shown ruptured into esophagus in the author's presence, as the patient suddenly sat up, because of abrupt anginal obligatory orthopnea.

The region of the manubrium, and the back between the left scapula and the spine should be examined with special care.

The Primary Signs *are:* (*a*) Abnormal pulsation usually in the locality mentioned, yet variable in position, degree and extent, and if typical, heaving and expansile. (*b*) Rarely, a visible tumor yielding expansile pulsation and covered by tissues which may be normal, tense, shiny, and congested or even necrotic. (*c*) A positive radiogram.

Secondary Signs.—(*a*) The peripheral signs of an associated aortic regurgitation may be present. (*b*) The apex-beat is low and may indicate left ventricular enlargement, a condition commonly but not invariably associated with thoracic aneurysm. (*c*) Signs of sclerosis in the peripheral arteries. (*d*) Cyanosis, localized edema and unequal pupils, vasomotor symptoms such as unilateral pallor, congestion or sweating. (*e*) Stridor, visible dyspnea.

(*f*) Paralysis of the vocal cords, associated hoarseness or aphonia. (*g*) Brazen cough.

Palpation.—Expansile pulsation and thrill and the so-called diastolic shock are chiefly to be sought. In certain aneurysms involving the transverse portion of the arch, one may find the "*tracheal tug*," first described by Oliver.

Tracheal tug valuable but overrated.

To obtain this, the cricoid cartilage is grasped by the thumb and finger of the observer as the head of the patient is tipped slightly backward; upward traction is then made and a tugging sensation may be felt with each cardiac impulse. The observer is often misled by the pulsation of vessels under the finger, particularly if aortic incompetence exists, and also by checked respiratory movements.

Furthermore, the sign is undoubtedly present in rare instances in persons free from aneurysms but carrying a strongly overacting "drop" heart, and in those who have residual pleural adhesions, and is as certainly absent in a very large proportion of the cases of true aneurysm.

Important.

Lateral rhythmic movement of the larynx sometimes may be detected (Cardarelli's sign).

The Pulse.—Aside from the signs of a coincident regurgitation the pulse yields information of real value in many cases. The observer should carefully compare the beat in the two radials and carotids and note any *decided* delay or inequality. Resulting, as these do chiefly, either from the direct or indirect pressure of the sac or from deformed arterial outlets in the portions involved, such differences, *if gross*, furnish excellent *corroborative* evidence and sometimes assist in localizing aneurysmal tumors.

Often useless.

Valuable suggestive sign.

Percussion.—In advanced and well-defined cases percussion may furnish direct evidence in the form of marked dulness over aneurysmal areas, but in the earlier cases it is deceptive and misleading. Auscultatory percussion is usually more valuable than simple percussion. Student and practitioner alike should be thoroughly familiar with the normal variations in the percussion note yielded by the *manubrium sterni*.

May be long absent.

Auscultation.—A systolic or more rarely a diastolic murmur, or both, may be heard, but the latter is due in most instances to associated valvular disease in aneurysms involving the first portion of the arch. A systolic bruit, often harsh, vibrant and associated with palpable thrill not uncommonly is produced by the sac itself. Pressure upon the pulmonary artery may also cause a systolic murmur. Such systolic bruits as are produced at the aortic orifice or by the sac often are widely transmitted along the vessels, and a localized systolic bruit heard at the left of the spinal column is an important sign of aneurysm of the descending aorta.

Thrills and murmurs.

The most important single sign of aneurysm of the first or second segments the author believes to be a distinct *ringing*, *metallic*, second sound, when heard maximally, not over the aortic valve itself, but over the sac. The maximum intensity of the true sac murmurs may occupy the same suggestive site.

In mere dilatation of the aorta this sound is maximal usually over the valve area, but this distinction is not constant.

Subjective Symptoms.—Dyspnea, precordial oppression and pain are the chief subjective signs, and, of these, pain is by far the most important.

Pain.

Theoretically, every large aneurysm should be accompanied by severe pain; actually, a large number go to a fatal termination without it. When present it may be localized, diffuse or referred, shooting, cutting, or more commonly dull or gnawing. Usually it is long misinterpreted, regarded as neuralgic, and so treated.

SPECIAL SYMPTOMS.—Aneurysm of the First Portion of the Arch.—Any statements made under this heading are liable to exception, but in general it may be said that if the first portion of the arch is involved, a

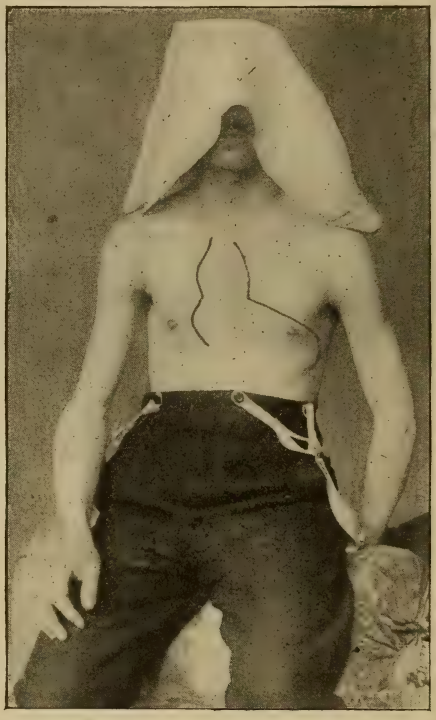


FIG. 420.—Luetic mesaortitis. Aneurysm of the ascending portion of the aortic arch. Note that application of old flat-finger percussion method yields an impossible Ml. dimension.

pulsation or rarely an expansile tumor is likely to appear at or about the second right interspace, which tends to extend its area of dulness outward and upward.

Symptoms often marked.

Attacks of angina pectoris and severe palpitation are common in this type of lesion. The aortic valves are likely to be involved and symptoms of aortic regurgitation and a double murmur are often present at the base, associated with *systolic thrill and marked diastolic shock*. Extension of the sac tends to cause localized edema, cyanosis, vasomotor symptoms and, possibly, pleural or even pericardial inflammation.

Aneurysm of the Transverse Portion of the Arch.—The common symptoms are dulness and pulsation over and about the manubrium and in the episternal notch, together with tracheal tug in some cases, paralysis of the vocal cord with a whispering voice and brazen cough and marked pressure symptoms; pain (variable and usually less severe than in aneurysm of the first portion), systolic bruit, diastolic shock (often lacking), pulse variations (often marked), dyspnea, and precordial pressure. *Pseudo-asthmatic seizures are not uncommon and lead oftentimes to mistaken diagnosis and treatment.*

Obtrusive signs.

Pseudo-asthma.

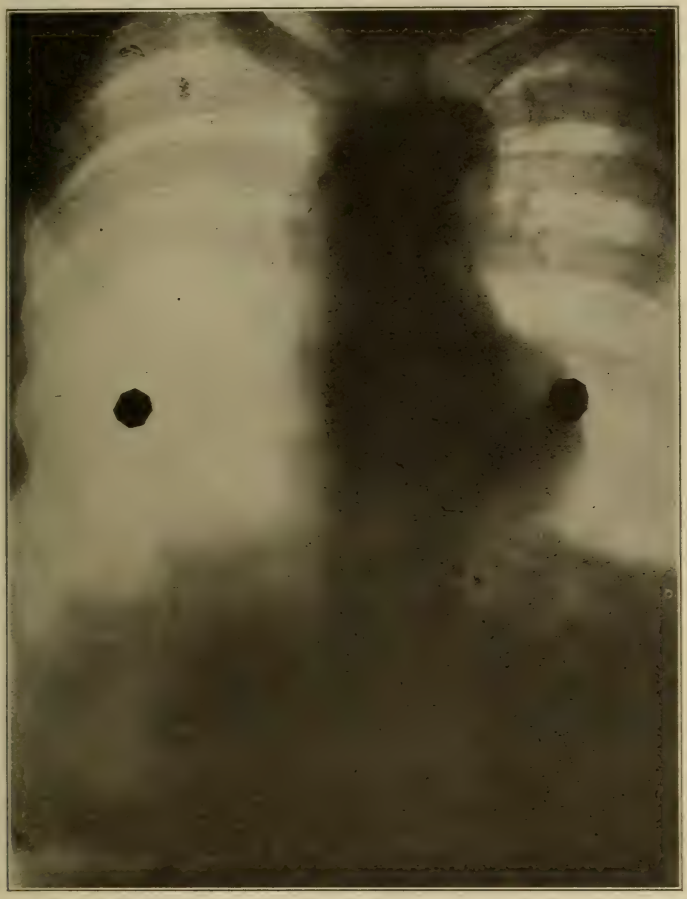


FIG. 421.—Malignant growth and aneurysmal dilatation. "Drop" heart greatly enlarged.

Aneurysm of the Descending Portion of the Arch.—The symptoms may be slight or absent, are usually posterior in localization, may include *bruit*, percussion dulness, superficial posterior pulsat on or actual tumor with ultimate external rupture, and are commonly accompanied by a considerable degree of posterior percussion dulness and gnawing pain.*

Obscure.

General Considerations and Differential Diagnosis.—Of a series of fifty

* In the author's experience, however, this pain has been of a remittent type, and relieved by absolute rest, prior at least to the terminal stages of the disease.

Early diagnosis rare.

Value of X-ray.

private cases of thoracic aneurysm observed during the past few years, many would have gone unrecognized had the X-ray not been used. Even certain advanced, rapidly progressive cases presented vague signs. Nearly all had been treated under erroneous diagnosis for neuralgia, asthma, dyspepsia, "neurasthenia," disease of the spine or pulmonary tuberculosis.

FIG. 422.—Syphilitic mesaortitis and malignant growth. Malignant growth producing stenosis at the cardia and extending upward in the mediastinum. Heart and dilated aorta are represented by the deeper shadow. Confirmed by repeated photography and finally by autopsy.

*The accepted symptomatology of thoracic aneurysm needs revision and our diagnostic methods should be reinforced oftener by the roentgenographic findings.** (See discussion of "Syphilitic Mesaortitis.")

Differential Diagnosis.—*Mediastinal Abscess.*—Its relatively rapid development with symptoms of suppuration and the absence of aneurysmal symptoms other than pressure symptoms should be sufficient to exclude this lesion.

Wassermann test valuable throughout.

Pulmonary Fibrosis.—Cases of fibroid phthisis or any case involving the retraction of lung may give rise to suggestive pulsation in aneurysmal areas,

* Of these cases nearly all have already died suddenly, in several instances without developing the more marked symptoms.

but ordinary methods should be sufficient to establish the actual conditions present and should be reënforced by a fluoroscopic inspection of the arch.

Pulmonary tuberculosis with a large cavity adjacent to the aneurysmal areas may produce pulsation and certain other aneurysmal signs, but the history, sputa, breath sounds, etc., should make error impossible.

Anemic pulsation should be readily differentiated by rational methods.

Aortic regurgitation is frequently associated with aneurysmal dilatation both dynamic and true; if unassociated, it should offer little difficulty.*

Malignant Growths in the Mediastinum.—When these are associated with evidences of malignant disease elsewhere in the body they offer little difficulty, but when not so associated they occasionally become most difficult to differentiate in the absence of the fluoroscope. They may yield all of the pressure symptoms and even *pulsation* in aneurysmal areas. The following points assist in differentiating them: (*a*) Knowledge of a primary focus of malignant growth. (*b*) Absence of the auscultatory signs of aneurysm. (*c*) Rapid emaciation. (*d*) Absence of *expansile* pulsation, whether upon ordinary inspection, or more definitely, as determined by fluoroscopic methods. (*e*) *The absence of marked improvement and relief of pain when placed absolutely at rest.* In two cases seen recently by the author a decided aneurysmal dilatation *coexisted* with a large mediastinal growth, in the one case a sarcoma, in the other, carcinoma. *Certain vascular growths with definite expansile pulsation lead occasionally to unavoidable error.*

Offer difficulties.

Important points.

Aneurysm may coexist.

Confusing conditions.

FINAL CONSIDERATIONS AND CONCLUSIONS.—Certain cases of aneurysm yield no symptoms save those determined by X-ray examination. Here, as elsewhere, a knowledge of the normal heart sounds is of the first importance. All ringing and *distinctly metallic* second sounds at the base of the heart should be regarded as suspicious *when their points of maximum intensity are found at some distance from the proper valvular area and pain or pressure symptoms are present.* Tangential inspection should never be omitted *and in doubtful cases yielding percussion dulness the effect of absolute rest should be determined.* In all walking cases of aneurysm any existing area of dulness is perceptibly reduced by a few days of rest, and the daily variability of pressure symptoms as related to the occupation or the pursuits of the individual should be considered. The temperature record, strength, and nutrition should be watched; unilateral loss of knee-jerks is suggestive and the proof of past syphilis or external signs of a denied lesion are most important. It should be remembered that active pressure ulceration of the pulmonary structures may produce the *physical signs* of a tuberculosis, that senile pericarditis is often aneurysmal, and, finally, that with the fluoroscope one usually may see and accurately measure the sac, determine true expansile pulsation and accurately record the changes induced by rest or treatment.

All important data.

PERICARDITIS

Definition.—*An inflammation of the pericardial sac almost invariably secondary to, coincident with, or superimposed upon, other diseases; occurring*

Usually secondary.

* See Roentgenography.

as an acute, subacute or chronic ailment; dry and fibrinous in type, or associated with a serous, hemorrhagic, purulent or sanious, effusion.

Etiology.—*Between 40 and 50 per cent. of the cases of acute and subacute pericarditis are caused by acute rheumatism and the etiologic factors are in the main identical with those of endocarditis, already described.*

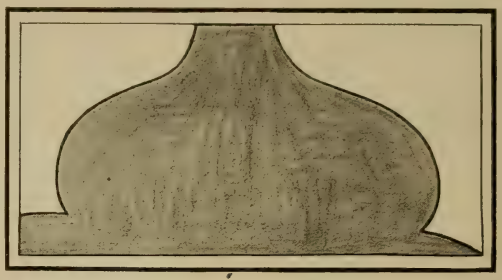


FIG. 423.—Large Pericardial Effusion (fluoroscopic outline). In the early stages the angle formed by the right border of the heart and the diaphragm (junction of right heart dulness and hepatic dulness) at the right sternal border is acute in the case of the normal heart, a right angle in the case of the extreme type of "drop" heart, with the accumulation of fluid the angle is rendered obtuse by decided dulness or flatness, on light percussion, and so demonstrable in many instances. The position of the angle is shifted to the right, but as the effusion distends the sac an acute angle is formed decided dulness or flatness extends far beyond the sternum to the right. The secondary resumption of the acute angle may not be demonstrable by percussion, though readily revealed by the fluoroscope.

Cryptogenetic focal infections of the most varied actual origin are adequate to produce the disease and a considerable number of cases result through direct extension and circulatory and structural continuity in cases of pneumonia, pleurisy and peritonitis.

Whether the disease may be said to be *caused* by Bright's disease, scorbutus, purpura and other ailments of like nature may be doubted. The depress-

FIG. 424.—General dilatation and insufficiency. This may resemble pericardial effusion yet more closely in certain universal dilatations of the "drop" heart occasionally encountered in acute prostrating infections. (*Schwartz, modified.*) (See Fig. 358.)

ing constitutional effect of such toxemias doubtless invites infection from the many obscure but potent sources now known to exist in a considerable proportion of individuals. The same statement applies to pericarditis associated with pulmonary tuberculosis in some instances, many of these being non-

tubercular and probably due to the same conjunction of predisposition and potent pathogenic organism.

FIG. 425.—Pericardial effusion, typical and decided but not extreme. (Dorso-ventral aspect.) The limitation of the ascent of the liquid by the attachment of the pericardial layers to the great vessels and the resulting stubby neck of the "flask" or "decanter" profile is indicated quite clearly. Dulness decided to right as well as left of sternum, true apex-beat absent, heart sounds lost or extremely faint at apex. (Repetition of figure shown under "Roentgenography.") (*Dr. Frank S. Bissell.*)

Pancarditis.

In all cases of endocardial and myocardial disease the possibility of a pericardial complication must be held in mind and, in children especially, involvement of the entire structure of the heart (pancarditis) is extremely common.

Age, Sex.—By far the greater number of cases occur in children and young adults and according to most statistics there is decided male predominance.

Dry vs. Wet Cases.—Cases of fibrinous pericarditis and those of the exudative form occur in about equal proportion according to available statistics. It is wholly probable that the true relative incidence, if known, would show a great predominance of the former type.

FIG. 426.—Greatly and universally dilated "drop" heart. Residual acute dilatation. Heart sounds sharp and distinct. Soft systolic bruit at apex. Apex beat diffuse, systolic retraction marked over right ventricular area. True apex beat diffuse, sharp and unsustained. Superficial and deep dulness well defined to left of sternum, former showing extension to right sternal border. Area of relative dulness to right of this point not accurately definable. In a second picture taken a few weeks later a better plate and exposure brought out the areas of residual infiltration and extremely dense hilus shadows reminiscent of the causative influenzal broncho-pneumonia. The former are suggested faintly in the shadow of the right ventricle (to the left of the sternum) and also over the same chamber to the right of the sternum. Both this and the final radiogram, showing the resumption of the narrow "drop heart" outline normal for its congenitally asthenic possessor are shown under "Drop heart." Acute dilatation may involve the right, left or both chambers of the heart. (See also Figs. 424, 425 and 427.)

FIG. 427.—A case of chronic universal enlargement of a heart primarily of the normal type. (Dorso-ventral aspect.) Hypertrophy and dilatation are present, the patient, an unusually powerful broad-chested man, attends to his business as a building contractor, but carries hepatic engorgement, diurnal edema of the ankles and persistent râles at the lung bases. Is exhausted easily and has decided exertion dyspnea. A "fatty tail" indicating some degree of "fatty overgrowth" ("fatty infiltration," "fatty heart"). It will be noted that this condition obliterates both right and left inferior angles. Total transverse measurement 19 cm. If this myocardium were to become profoundly toxic, during a pneumonia, influenza, septic cholecystitis, acute rheumatism or other such prostrating ailment such a heart might assume the form shown in Fig. 424, a silhouette usually exaggerated, as to the loss of the lower left ventricular curve, by the high position of the diaphragm so often present, according to the author's experience. Usually, at the stage where such an outline is obtainable, the patient has difficulty in breathing and quite often ascites of some degree or excessive flatus is present.

Furthermore, the author has found in two instances, at autopsy, that a complete loss of both right and left cardio diaphragmatic angles in such a heart was due apparently to a considerable amount of pericardial exudate filling them when the patient was in a sitting or erect posture, with a large amount of fluid the angles would have been indicated sharply, as in Figs. 423 and 425. This heart presented no murmurs of any kind and, as is so frequently the case, the patient was complaining only of "dyspepsia" and "nervousness" and had been treated for these conditions alone. A "fatty tail" is clearly shown at the apex.

Often obscured.

Silent and Unrecognized Cases.—*To a remarkable extent this important disease is overlooked, as must be the case with any ailment so largely secondary and so likely to be overshadowed and dominated by the primary disease.*

Autopsy reports prove its presence in 12 per cent. of the fatal cases of pneumonia and it is recognized clinically in only 1 per cent. It is found in 5 per cent. of the cases of chronic tuberculosis coming to post-mortem section and in these is almost never recognized.

To a remarkable degree its presence is coincident with that of endocardial lesions, preëxistent or otherwise, and the striking symptoms of endocarditis may dominate the clinical picture, even of a pancarditis.

Their number decreasing.

The "Idiopathic" Cases.—In the past many such have been reported but at present and in the future the number will be greatly lessened because of our recently acquired knowledge of the etiologic potency of chronic cryptogenetic septic foci notably those represented by infected *tonsils*, accessory sinuses and peridental tissues, together with our better appreciation of the peculiarly silent character of many cases of obscure *acute rheumatism* occurring in young children, among whom many of these so-called "idiopathic" cases have been noted in the absence of active joint symptoms, such as we now know are oftentimes wholly lacking in many entering into that age group.

Obscure focal infections.

Obscured rheumatic cases.

The painlessness of a considerable proportion of the acute and subacute pleurisies has served also to mislead the physician.

SYMPTOMS COMMON TO BOTH DRY AND WET CASES.—*One must sharply distinguish between these two groups of cases, viz: those with and those without a liquid effusion; and also two groups of symptoms, viz: those common to both conditions and those chiefly characteristic of the one or the other.*

Afebrile cases.

Fever.—*The number of afebrile cases of pericarditis is probably considerable and this statement is especially true of those instances in which this disease complicates a preëxisting ailment markedly affecting the nutrition and resisting power of the patient.*

In many other instances the fever of a primary lesion dominates and obscures that due to secondary, complicating pericarditis. Fortunately, in acute rheumatism and certain other ailments the temperature chart often shows a suggestive sudden ascent coincident with the pericardial onset.

Fever, when present, may endure but for a few days or continue for two or three weeks, in some instances. In septic cases the characteristic curve is present, but this form of fever is seldom long continued because of the short duration of life in such cases of this type as fail to secure immediate or very early radical surgical intervention.

Sources of pain.

Pain.—*Pain or decided discomfort is present in about three-fourths of the cases of pericarditis as a primary symptom, but one must distinguish clearly between that which is apparently due to actual pericardial friction and its resulting irritation, that which is the result of such greatly increased intrapericardial pressure* as accompanies a massive effusion, and that which is largely due to the cardiac inadequacy of associated myocardial toxemia or actual myocarditis.*

Any primary pericardial pain may subside either from (a) a marked increase

* Sufficient actually to *rupture* a myocardium so distended.

of the fibrinous exudate of the dry pericarditis, (b) the attainment by adhesion of local immobilization of inflamed serous surfaces, or (c) the outpouring of a liquid exudate which separates the pericardial layers.

The last event, as previously suggested, may relieve a *primary pain*, only to cause the reappearance of one of a different character, usually best described as discomfort or distress, yet agonizing in rare instances.

Localization of Pain.—Pericardial pain is seldom sharply localized save in those cases in which it is referred to the left shoulder and arm or to the neck as happens especially in cases of greatly increased intrapericardial pressure or associated or antecedent myocardial or coronary disease. In either wet or dry pericarditis it may be referred to the epigastrium, which is sometimes the initial seat of any pain present in the case and is frequently a point of maximal discomfort and distress in cases of effusion. In some instances attacks indistinguishable from classic "angina pectoris" occur and these are due doubtless to myocardial involvement and to interference with the intrinsic blood supply of the heart through actual extreme pressure or, later, the involvement of the coronary arteries or the heart chambers in compressing or constricting bands of adhesion.

Shoulder pain.

Epigastric pain.

Angina pectoris.

Even slight precordial pain is suggestive when occurring in diseases of which pericarditis is known to be a frequent complication.

The Pulse.—Acceleration of the pulse is the common finding, but various forms of arrhythmia are extremely common and heart block occurs frequently in its minor grades, recognizable by instrumental means, and, occasionally, in its classic form, the pulse dropping to 40 or lower. Slow pulse not rarely occurs apart from actual heart block, apparently because of involvement of the vagus nerve and resulting excessive inhibition.

Rapid usually

Slow pulse.

Dyspnea and Cyanosis.—Dyspnea is common to both types of pericarditis, though inconstant and seldom of marked degree in the dry form unless there be coincident involvement of the pleura and pericardium. In cases with effusion it may and often does attain a high grade, forcing an obligatory sitting posture on the part of the patient (orthopnea) and producing sometimes a truly pitiable condition.

Present in both forms.

Decided in "wet" cases.

If in any case *paroxysmal* dyspnea or Cheyne-Stokes breathing occurs, renal disease is suggested strongly (Cowan). The author has also encountered it in cases showing at autopsy a mere passive congestion of the kidneys, but marked myocardial involvement, and, in one instance, coronary sclerosis chiefly affecting the right heart.

If the myocardium is greatly affected, the effect of exertion, even of the slightest grade, may be reflected promptly and significantly in marked increase of dyspnea and cyanosis or take the ominous form of sudden "ashen" pallor.

Oddly enough cyanosis is common to both forms of pericarditis though it must be assumed that the slighter grade seen in the dry type of the disease is chiefly related to the coincident myocardial toxemia or to increase in the preëxisting weakness of a diseased heart, such as is so often present in this predominatingly secondary ailment.

In pericarditis with effusion, cyanosis may be of an extreme grade and its

variations together with the possible concurrence of decided pallor or its abrupt onset or increase constitute valuable evidence as to the grade of the lesion and prognosis in the individual case.

"Friction redux."

The Friction Rub.—This is the dominant symptom of true dry pericarditis, though, in a modified form and a lesser degree, it may persist in cases of effusion even of high grade in some instances, or in exceptional cases, return after the effusion has become absorbed.

The friction rub, usually double, may be single or triple; is usually audible primarily and chiefly at the base of the heart in the neighborhood of the great vessels or, not infrequently, is heard primarily or coincidently at the apex or over the right ventricle.

In rare instances, in the presence of decided dilatation of the left auricle such as might occur as a result of preëxisting mitral stenosis, pericardial friction may first be audible in the region of the inferior angle of the left scapula.

Quality of Pericardial Murmurs.—*In an overwhelming majority of cases of dry pericarditis the quality of the friction murmur is so characteristic as to be pathognomonic and makes the diagnosis simple and direct.*

In rare instances, however, the rub may be softened by exudate until quality alone does not suffice for its differentiation from an endocarditic or accidental heart murmur. In the latter instances other peculiarities of pericardial friction are evoked and usually suffice for diagnostic purposes.

Friction fremitus.

In its typical form and common variants the murmur is accurately described by some one of the terms, "shuffling," "rubbing," "squeaking," "creaking," "grating" and these are often so loud and harsh as to readily explain the frequency with which one obtains by palpation the so-called "friction fremitus."

Neither systolic nor diastolic.

Time of Pericardial Murmurs.—*One of the most important characteristics of the pericardial friction rub as affecting differential diagnosis is its failure to occur in exact synchronism with the first or second sound of the heart.*

If only the single murmur is present it is commonly distinctly postsystolic, a point of the greatest value in differentiation inasmuch as it is this isolated rub which is most likely to create difficulties.* Commonly the rubs occur in pairs ("to and fro"), less often in triplets, the last being peculiarly characteristic when present and yielding a sort of frictional gallop rhythm.

Pairs and triplets.

Superficial Quality of Pericardial Friction.—Pericardial friction conveys to the ear a sense of its nearness or superficiality, *i.e.*, proximity of source, most important in differentiation and, fortunately, rarely lacking in distinct and recognizable degree.

Stethoscopic Pressure Effect.—Save in very rigid chests or in the case of elderly persons carrying a pronounced emphysema, increased pressure with the stethoscope bell usually results in an intensification of any existing pericardial friction sounds and this phenomenon is accompanied oftentimes by the elicitation of a most suggestive localized tenderness.

Increases intensity

On the other hand, excessive pressure, in rare instances, may obliterate it.

* If the murmurs occur as triplets the third element is presystolic but utterly unlike a presystolic mitral murmur.

Inconstancy of Pericardial Friction.—The tendency of friction murmurs to show marked changes in quality and even of the numbers of elements in each cardiac cycle from day to day, hour to hour, and even from minute to minute, in some instances, is sometimes a most helpful characteristic. They also may show at times decided variation with the phases of respiration and with changes of posture. Both of these latter phenomena lacking constancy in occurrence in pericarditis but being valuable when present. With coincident pleurisy and pericarditis the "pleuro-pericardial friction sounds" fully described elsewhere may be readily elicitable.

Crescendo and Decrescendo Quality Lacking in Friction Murmurs.—Practically all murmurs due to valvular deficiency whether they be systolic, diastolic or presystolic in type show a definite and recognizable tendency to either "wax or wane" during the period of their continuance.

The "thrilling" classical presystolic murmur of mitral stenosis is typically and exquisitely crescendo; that of a mitral regurgitation is a systolic bruit and, like the diastolic murmur of aortic regurgitation, distinctly decrescendo beginning strongly and ending by gradual attenuation. With rare exceptions these qualities are wholly lacking in pericardial friction.

The post-systolic "cardio-pulmonary murmur" or those which may represent a very slight leakage late in systole due, perhaps, to slight *muscular* insufficiency can only be confounded with the rarest types of pericardial friction.

Failure of Definite Transmission.—Those definite lines of transmission so generally observed in true endocarditic lesions are wholly lacking in those of pericarditis, the area of audibility of any pericardial friction rub being dependent wholly upon its loudness and the presence or absence of conditions limiting its transmission to the surface of the chest. This point is often of cardinal importance in the rare cases of unusual obscurity.

It must be remembered, however, that the common soft flowing murmurs of *relative* secondary or degenerative *insufficiency* of the mitral valve may be quite sharply limited to the apex.

The point of chief importance is the fact that even a *loud* pericardial rub conforms to no law of transmission save the general one.

SYMPTOMS CHARACTERISTIC OF EFFUSION.—The cardinal sign of pericardial effusion is an increased area of unusually decided dulness or actual flatness representing the profile of the distended pericardial sac and therefore assuming the form of a somewhat unsymmetrical *big-bellied* stubby-necked decanter or ancient vase, extending not only far to the left and right of the antecedent percussion profile of the heart, but often carrying median dulness upward to the level of the second rib. **True outline.**

In massive effusions the left border may project so far as apparently to reach nearly to the left thoracic wall and the right border may extend to the mid-clavicular line or even beyond it.

The area of superficial dulness to light percussion is greatly extended and actually flat, and the entire profile percussion is roughly triangular and more definitely dull and higher pitched than that of cardiac dilatation.

Early evidence.

The patient being recumbent, a small effusion may, rarely,* first become manifest over a small triangular area with its base *upward*, at the level of the second interspace. In any event evidences of effusion are likely to be indicated very early by flatness just to the right of the sternum in the fifth interspace, the region of the "cardiohepatic angle," which it obliterates ("Rotch's Sign"). *This should be percussed primarily while the patient leans well forward and to the right.*

Rotch's sign.

The liquid carries the dulness outward and to the right as well as upward, as the effusion increases. In some cases it is manifested early at the left border of the heart, extending beyond the normal profile and any visible apex-beat, the latter being often visible above and internal to the apparent boundary of the left heart.

In liquid effusions of all decided grades any extension of the area of decided percussion dulness beyond the apparent apex-beat and flatness over the normally resonant area represented by the normal cardiohepatic angle at the right sternal border are symptoms of prime importance.

Enormous distention possible.

Under certain conditions especially, it is said, in scorbutic cases, and certainly, according to the author's experience, in the chronic effusions of wet polyserositis, the pericardial sac may be so distended by reason of chronic inflammation and persistent accumulation of fluid as to contain 3 or even 4 quarts of serum (Alonzo Clark).

Misleading cases.

In many such cases a coincident pleural exudate may occur, not infrequently encapsulated by adhesions and mislead the physician into the belief that the entire flat area is due to pericardial exudate.

Often overlooked.

On the other hand, such pleuritic effusions may cause the examiner to overlook a coincident pericardial accumulation. The possible concurrence of the two conditions should be borne in mind and in all cases of apparent gigantic pericardial effusions, the outer area should be first tapped, this procedure sometimes revealing only a moderate residual exudate within the pericardium, the free border of which has been pushed aside in such a way as to exaggerate the actual condition.

Changes in the Level of Dulness.—In moderate effusions, changes in the level of dulness may occur in response to alterations of posture but the sign is anything but constant. Very frequently postural changes increase any existing precordial oppression.

Pressure Symptoms.—One has only to recall to mind the narrow limits of the mediastinal space and the vital nature of its many contained structures to realize that any tumor within it, whether it be aneurysmal, malignant or only that of a distended pericardial sac, may produce striking pressure symptoms, such as are dealt with under the head of "aortic aneurysm," but it may be said that, in general, pericardial exudates produce fewer of these in any extreme form than would be assumed.

Precordial and Epigastric Bulging.—The flexible chests of children may bulge from a pericardial exudate, and, in young adults of delicate structure, flush or even bulging interspaces may be observed. The epigastrium may be

* In the author's experience.

prominent by reason of a dominant displacement of the left lobe of the liver due to the thrust of the distended pericardial sac and a striking and decided epigastric respiratory immobility, or relative passivity, is manifest in some instances.

Hydropericardium.—The pericardium not infrequently contains a liquid *transudate* in cases of general dropsy such as accompany chronic Bright's disease or profound cardiac decompensation affecting the right heart. Occasionally such a transudate occurs as an isolated phenomenon under these and similar conditions and may prove puzzling or be wholly overlooked.

Such an effusion may be wholly symptomless, for no fever is present unless it be that of some complicating febrile ailment. In other cases it may cause decided pain or precordial and epigastric oppression, seriously embarrass a crippled heart and prove a terminal event unless promptly relieved.

Pyopericardium.—Purulent pericardial exudates may be primary but are usually secondary to a similar process in the pleural cavity or, more rarely, the peritoneal space and, as previously stated, will in most instances prove rapidly fatal unless drained promptly.

Such cases may present a typical septic syndrome, be obscured by similar symptoms due to the primary ailment, or run a wholly afebrile course.

Pneumopericardium.—Almost invariably this condition is associated with a purulent rather than a serous or hemorrhagic effusion and represents in the rarest instances the activity of gas-forming bacilli though it usually arises from fistulous communication, accidental wounds or the maneuvers of aspiration. In the former case it demands immediate surgical interference. In the latter it may prove temporary and of trivial importance. Usually pyo-pneumo-pericardium.

The condition is *excessively rare, but* readily recognized, usually, by the peculiar melange of pericardial friction sounds and churning, splashing, tinkling and purling liquid sounds obviously related to cardiac activity which makes a *bizarre* picture peculiar to this condition.*

RATIONALE.—Pericardial Relationships.—A thorough understanding of the basic factors of pericardial inflammation, and of the nature and relationship of the pericardium itself, at once makes evident the genesis and logical sequence of the clinical signs and symptoms of pericarditis.

The pericardium in health forms a potential, closed, lymph space, the outer wall of which is formed by its parietal layer, the inner by the visceral pericardium which is reflected upon the heart, of which it forms its outermost protective coat and with which *it thus becomes structurally continuous.* A lymph space.

The Normal Surface.—The apposed surfaces normally are perfectly smooth, sufficiently elastic to be marvelously distensible in the presence of effusion, and so perfectly lubricated as to permit that soundless and symptomless play of the visceral and parietal layers necessary to their adaptation to heart movement and, to a lesser degree, the respiratory movement of the lungs and thorax. Perfect lubrication.

* The extremely interesting case reported by Cowan lacked fluid accumulation at the time of the X-ray examination and hence the signs just described were absent. Prior to this the case had been regarded as an obscure sepsis.

Diaphragm and Cervical Fascia.—The subserous connective tissue of the parietal layer is adherent to and structurally continuous with that of the diaphragm below, the pleural sinuses anteriorly and below, the sternum and mediastinal tissue through two fibrous bands, and the cervical fascia above.

Further structural continuity.

An Important Area.—Moreover, the great vessels at the base are ensheathed by it for 1½ inches or more before it blends with their own coverings and a small, relatively free space and a series of pouches are thus formed, in which, as in the cardiohepatic angle next the sternum or at the apex, any pericardial effusion first may be demonstrable.

A consideration of the anatomic structure and relationship makes it obvious:

1. *That only when the serous surfaces are normal can the two layers of the pericardium glide upon each other with every heart cycle without exciting either sound or sensation.*

Secondary pleuritis, pancarditis or peritonitis.

2. *That inflammation may extend readily from the parietal pericardial layer to the pleura, the diaphragm or even the peritoneum, from the parietal wall to the mediastinal tissues or, from the visceral layer to the entire heart, its valves included. A pleuritis, peritonitis or a pancarditis is, therefore, wholly possible.*

Secondary pericarditis by extension.

3. *That this process may be directly reversed and pericarditis result from direct extension, in pleurisy, pleuropneumonia, or peritonitis.*

4. *That all these structures may be simultaneously involved (polyserositis) in either an acute or chronic process.*

Complete adhesion.

5. *That a chronic pericardial inflammation and the wholesale involvement of adjacent structures in this process, may produce all grades of residual adhesion, a complete obliteration of the potential pericardial cavity through the formation of adhesions which would bind firmly to the surface of the heart (visceral pericardial layer) the parietal layer of the pericardium and even render rigid and immovable the entire mediastinum ("mediastino-pericarditis," "indurative mediastinitis").*

Mediastino-pericarditis.

Secondary myocarditis.

6. *That even though a pancarditis is absent, the myocardium is always to some degree affected and often undergoes actual infiltration and degeneration.*

7. *That many of its symptoms are chargeable in part, at least, to this factor.*

Dry pleurisy.

Pathology.—At the beginning of an attack the pericardial serous membrane normally smooth, soft, glistening, well-lubricated and velvety shows patchy areas of opacity and becomes dry and harsh from the hyperemia and arrest of secretion.

Distribution of exudate.

Beginning usually at the base of the heart in the neighborhood of the pericardial reflexions which form sheaths for the great vessels it tends to become practically universal and affect both the parietal and the visceral layers of the sac.

Exudate.

In dry, fibrinous pleurisy the adhesive exudate appears first as flakes in the region near the arterial trunks or over the ventricles, if this region be primarily involved, and chiefly accumulates later at the point originally mentioned and about the coronary vessels.

Varying distribution.

It varies greatly in amount, may be abundant and universal or circumscribed in distribution and very scant, and may be equally profuse on the

visceral and parietal layers or predominatingly affect one only. A primarily dominant inflammation of the parietal surface results from a primary *pleuritis* and represents direct extension by continuity of tissue and a common vascular supply. The surface of the pleura may be merely rippled or actually shaggy or villous (*cor-villosum*).

Adhesions.—Complete recovery from a fibrinous pleurisy may result with entire restoration of the normal appearance of the pericardium, but this outcome is undoubtedly rare. More or less adhesion commonly results, though fortunately it is seldom universal or very extensive, because of the continued movement of the heart. Adhesions may be filmy or tenuous, dense and massive, or divide the pericardial sac into several compartments. In certain instances the pericardium, the tissues of the mediastinal space, the pleura and even the peritoneum become jointly involved in chronic inflammation and permanent massive adhesions. This is the so-called "*mediastino-pericarditis*" described elsewhere. In a similar manner the great arteries, veins, auricles or even the ventricles may be enclosed, embarrassed, and even cripplingly compressed.

Adhesions.

Genesis of the Friction Sounds.—We see that, as in the case of the pleura and of serous membranes in general, the response of the pericardium to infection takes the form of inflammation of the serous lining, at first patchy in distribution, but usually becoming general. This at once checks secretion over the portion involved and converts its velvety lubricated surface into a harsh dry membrane. Obviously this must transform the silent gliding play of these membranes in response to heart action into a forced rhythmic *affriction*, usually painful, audible and, not infrequently, palpable.

Secretion checked.

"Scuffing."

The parietal layer is usually first involved and that portion of the heart subjected to the maximum of movement usually determines the primary location.

Determining factor in location.

It is evident, therefore, that the most frequent primary area of audibility of the friction sounds of pericarditis will be over the left base or, less commonly, at the apex. They may spread over the whole heart or be more or less strictly limited to the original area and its immediate vicinity.

Auscultatory points of election.

In somewhat rare instances, they may first be heard at the epigastrium, a not infrequent site for maximal distress if pericarditis attacks a heart already dilated as to its right ventricle, and, less frequently, near or over the spinal column at the inferior scapular level, being transmitted from the left auricle and base of the left ventricle.*

Epigastric and interscapular "rubs."

Modifying Influences.—The variations in the qualities of pericardial "rubs" must obviously depend upon the varying dryness and harshness of the apposed surfaces as the fibrinous exudate is freely exuded or withheld, the variations in the rate and strength of the succeeding series of heart cycles, the formation of adhesions, the rigidity and thickness of the affected pericardial layers, and the external and internal pressure to which they are subjected.

Basic factors.

* One would assume that this would be most likely to occur, or occur only, in cases of preëxisting mitral stenosis or regurgitation, attended by decided left auricular enlargement.

The quantity of reactive inflammatory exudate and its quality alike, therefore, greatly affect the sounds.

The "rub," the "blow" and the "crackle."

If both the apposed surfaces are dry and harsh, the sounds will be correspondingly rough and intense. *If not equally affected*, the murmur will be somewhat modified, and *if a profuse fibrinous exudate or serous effusion is present* they may be so modified as to resemble an endocardial murmur or, as the exudate stiffens, a superficial crackle, or, be wholly lost.

Soft murmurs.

Obviously they are well described as "shuffling," "rubbing," "creaking," "squeaking" or "grating" murmurs; yet it is evident that they may be of all grades of harshness on the one hand or as soft as the pulmonary vesicular murmur on the other, because of the modifying influence of the fibrinous exudate. It is fortunate that the latter are rare, for their differentiation is sometimes less easy than their description.

Postural Variation in Murmurs.—It is obvious that murmurs persisting after the appearance of an exudate may vary greatly as regards areas of audibility with changes in position. The same statement is true of all friction murmurs and especially such as arise in the excessively mobile "drop heart." In dry cases the greatest postural variation occurs in such as arise from the right ventricle or at the heart borders.

Variations in quality or the total disappearance of *posturally induced marginal murmurs* and the advent of wholly new "rubs" upon changing the position may be determined in some instances.

The triple element of the friction sounds, if present, is usually presystolic and presumably due to auricular systole. At times the sounds may be almost continuous, but usually they are double ("to and fro") distinctly separate, and the post-systolic murmur is the louder.

Absorption of Exudate and Return and Recession of Friction Sounds.—*As the exudate is absorbed friction sounds may reappear over wide or limited areas. In fibrinous pleurisy, as stated, they may disappear through (a) the outpouring of abundant exudate, (b) the recession of the inflammation, or (c) through the formation of adhesions.*

Relative persistence

In the recession of murmurs of this type that of the systolic phase usually persists for a longer period than its fellow or fellows though in one or two instances the author has seen a persistence of the diastolic element after the systolic had vanished.

Pleuro-pericardial Friction.—It is evident that if pleurisy co-exists, *as is frequently the case*, another element is added which is more markedly affected by respiration than the pericardial "rubs" and is usually differentiated by the fact that, in general, the pleuro-pericardial friction sounds show inspiratory accentuation, expiratory diminution or elision, and are diminished if the breath be held in expiration.

Effect of respiratory phases.

Important factors.

Other important points in differentiation are: (a) The fact that pleuro-pericardial friction occurs chiefly along the lung margins and especially at the borders of the notch bounding normally the superficial cardiac dulness, and (b) that when pleuro-pericardial friction is present one seldom fails to find indubitable pleural rubs distant from the heart.

Resolution and Repair.—If the inflammation stops short with only a moderate fibrinous exudate as happens in about 50 per cent. of the cases, the affected membrane may undergo a more or less complete regeneration with few or no adhesions.

Adhesions.—Agglutination of layers alone may result in a complete obliteration of the pericardial sac, the parietal layer of which becomes universally adherent to its fellow and in consequence to the heart itself, a condition unrecognizable, *intra vitam*, and, of itself, only occasionally interfering with the intrinsic circulation *of an otherwise normal heart* or tending, in the absence of preëxisting disease, to produce serious myocardial weakness and degeneration. If, as so often happens, the heart is the seat of endocardial lesions or a myocardial degeneration, whether primary or secondary, the lesion becomes a serious complication.

Partial or universal.

Progressive heart failure without endocardial lesions, when occurring in young people, should always suggest adhesions. As a rule, the heart will free itself in large degree from those of the ordinary type located upon its more active regions but under certain conditions most extreme manifestations occur.

Polyserositis, Mediastino-pericarditis and Indurative Mediastinitis.—It occasionally happens, and with especial frequency in the more chronic processes such as tuberculosis, that, starting most frequently in the pericardium, but nearly as often in the pleura, and more rarely in the peritoneum, a polyserositis occurs, involving the mediastinal structures, the pleura, peritoneum, diaphragm and even the cervical fascia. All these are structurally continuous to such a degree and in such manner as tends to bring about, not only an obliteration of the pericardial cavity, but an "*indurative mediastinitis.*" This may be manifested by visible systolic recession of the seventh and eighth left interspaces, diminished, absent or reversed movement of the ensiform in respiration, or actual lower sternal, systolic indrawing, with epigastric retraction or impassivity in respiration, and decided systolic tugging of the lower interspaces or of the ribs themselves anteriorly and posteriorly (the latter retraction being wholly unaffected by respiration) (*Broadbent's sign*) with each contraction of the heart.

In these curious cases the heart, pericardium and mediastinal tissues become fused by the chronic inflammatory process; the heart and its envelope alike being rigidly anchored to the sternum, the spinal column, the diaphragm (itself often wholly or partly immobilized) and the pleuræ.

Thus with each systole the contracting heart pulls in the more yielding structures both anteriorly and posteriorly and produces the important events just described in whole or part.

The fixation of the heart abolishes any shifting of the border or apex-beat such as ordinarily occurs if one assumes the lateral decubitus and a most interesting diastolic vibration or shock of almost pathognomonic significance may be felt over the *right ventricle.*

If one considers the effect of binding together by adhesions the cervical fascia with its venous and arterial sheaths, the diaphragm, the pleura, the

sternum, the pericardium and the heart itself, he can readily understand that a multitude of subsidiary signs may be associated with this curious and slowly fatal ailment. Thus, in addition to diastolic jugular collapse,* paradoxic *inspiratory* filling of the cervical veins, the true paradoxic pulse, we may find any of the symptoms due to pressure involvement or inclusion of the vagus, phrenic and other *important* structures transversing the mediastinum.

In such cases the veins of the neck may be turgid and the abnormally marked inspiratory weakening of the radial pulse (*pulsus paradoxus*) may be manifest and, indeed, finds its best and most typical expression.

Polyserositis Proper.—Universal polyserositis or what is known as "*Concato's disease*" may begin in the pericardium, pleura, or peritoneum, as a wet or dry pericarditis, pleuritis, or peritonitis, usually chronic or sluggish from the beginning.

Concato's disease is apparently inseparable from Pick's disease though the latter laid special stress upon a variant in which an amazingly enduring and persisting recurrent ascites constitutes the most prominent feature.

All varieties should be placed in the one melting pot.

The pleura may dominate in one case, the mediastinum and heart in another, the peritoneum, liver and spleen in a third, and a wet serositis keeps the physician busy draining at short intervals the pericardium, pleural sac and abdominal cavity.

Mediastino-pericarditis and the peritoneal-hepatic cirrhosis type (Pick's disease) with recurrent ascites are the most interesting clinically.

The heart may assume enormous proportions in mediastino-pericarditis and any and all evidences of progressive myocardial failure may present themselves together with various murmurs due to underlying endocardial lesions or relative, secondary or consecutive insufficiency.

Myocardial Involvement.—To a greater or less extent, and almost invariably, the myocardium *must* be affected in the course of a pericarditis and it would seem probable that in the severer and fatal cases such participation of this vital tissue constitutes the most serious element in the case.

Pancarditic tripod.

As previously stated, pericarditis forms one of the legs of the *pancarditic tripod* pericarditis-myocarditis-endocarditis, especially common in young children, in whom signs of acute endocarditis always strongly suggest the presence or probable subsequent appearance of pericarditis and, conversely, in the case of *little* children the primary appearance of pericarditis renders an ultimate pancarditis wholly probable.

In all pericardial inflammations a small amount of liquid exudate is present and even in the most massive of the exudative forms a complete disappearance may take place. On the other hand, chronic effusion particu-

* Now regarded merely as an unsustained *systolic* venous pulse, the rapid emptying of which is greatly facilitated by the events present in this condition. Paradoxic swelling of the cervical veins may occur in inspiration owing to adhesion bands which check the venous flow.

larly of the tuberculous type may be carried unrecognized and is astonishingly well borne, over long periods.

Calcification.—One of the extremely rare results of pericarditis is that of calcification which is seldom recognized except by radiographic means.

Serous Exudates.—Obviously an inflammation of the pericardium, as in the case of other serous membranes, may result in the outpouring of a liquid effusion which according to degree and character of the original inflammation will be serous, purulent, hemorrhagic or sanious.

This constitutes the more serious form of pericarditis, even though the effusion be of a simple nature, and the most fatal form if it be hemorrhagic or purulent. In general, one must distinguish between the effusion which is simply blood stained or blood perfused, such as is commonly encountered in *tuberculous and cancerous cases*, and those *primarily hemorrhagic*, such as occurs especially in scorbutus, more rarely in purpura and leukemia, which are almost invariably fatal. Bloody effusion

In rheumatic cases the effusion is prompt in appearing, the dulness being demonstrable usually within three to four days or even earlier. In other forms it may be greatly delayed.

All pericarditis cases should be watched with great care throughout their entire course, for at any time the exudate may appear.

The simple effusions resulting from acute rheumatic pericarditis offer the best prognosis and are most likely to disappear with reasonable promptitude either spontaneously or in response to appropriate treatment. Persistent effusions are usually tuberculous or a part of a "polyserositis."

In excessively rare instances, as stated, either through the accidental introduction of air through fistulæ, in the course of exploratory or drainage aspiration, or because of the presence within the sac of one of the gas-forming bacteria, a condition of pneumopyopericardium or pneumohydropericardium may result in the bizarre combination of splashes, churning and other murmurs of cardiac rhythm of pathognomonic significance already mentioned.

Either chronic adhesive or serous pericarditis may result from a chronic serositis in which all of the chief large serous cavities may be coincidently or consecutively involved.

Cardiac Outline in Effusion.—With respect to the accumulation of pericardial exudate and its effect upon the cardiac outline, there would appear to be some loose conceptions and it should be said primarily that the pericardium cannot be held to a rigid and unvarying performance. If the heart was diseased and greatly enlarged primarily, the area of dulness may be large even if the effusion is moderate.

An exudate usually accumulates somewhat gradually, *but may flood the pericardium suddenly* and produce serious symptoms of cardiac embarrassment and possibly sudden death. On the other hand, it may be extremely slow and chronic. Flooding exudate.

In general, as stated, it seeks first the open space and pouches about the great vessels at the base of the heart and that represented by the cardiohepatic angle at the right sternal margin which it promptly submerges (Rotch's sign). Beginning effusions.

Complementary spaces.

In very large effusions it actually may maintain and intensify the angle, but carries it far to the right of the sternal border* and usually yields a right angle to percussion.

Large vs. Small Effusions.—In lesser effusions, therefore, the most important signs are those of a widening of the left border, obliteration of the normal cardiohepatic angle, oftentimes a fairly well-defined area of dulness over and about the sternum above the level of the third interspaces, and a tendency to *progressive extension of the areas of dulness*, the last being of special significance and value.

Pressure Effects.—The pressure of such large effusions affects the heart in varying degree and chiefly by reason of a direct limitation of the coronary blood flow, interference with auricular diastole, and with emptying of the *superior cava*. It may exercise compression upon the various important mediastinal structures in close relation to it precisely as would be the case in aneurysm or a mediastinal tumor, but much less constantly, and to a lesser degree.

Epigastric fullness.

Compression of the Lung and Displacement of the Liver.—The liver, and its left lobe especially, is thrust downward by large effusions, the movements of the diaphragm are impaired, and the lungs undergo compression both anteriorly and posteriorly. The enlarged pericardial sac thrusts aside and compresses the anterior margins and exercises compression upon the base of

Lung compression.

the left lung, which, according to its degree, may produce posteriorly a hyperresonance of relaxation, or, a dulness due to condensation which may yield a misleading tubular breathing, save when the right lateral or knee-chest position is assumed, or, be silent from complete atelectasis with blocked bronchi.†

The lateral lung compression often produces a marginal zone of hyperresonance of the same nature. Egophany is often marked and tubular breathing is sometimes heard.

Paradoxic Variations.—In three respects we find conditions which do not seem to fit the conditions present in pericardial effusion:

Apex beat.

1. *The apex-beat, though usually lost in massive effusions, is often present above its normal site, and in most instances, considerably within the left border of dulness, especially in effusions of moderate grade.*

Heart sounds.

2. *The heart sounds themselves may be wholly lost, or, be audible over the region of the right ventricle and base, with a lessened intensity suggestive of interposed liquid, yet oftentimes little more than the laboring weakened heart would explain.*

3. *It seems obvious that if the heart be completely surrounded by liquid and the pericardial sac completely filled all antecedent friction sounds should disappear, yet even in massive effusions, though friction sounds are always markedly lessened in the area of audibility, they may not be wholly lost.*

* This is readily demonstrated by a radiogram or with the fluoroscopic screen and the difference between a small early exudate and a fully distended sac should be borne in mind.

†The essential thing for the student to remember is the fact that various degrees of lung compression *must* exist in large effusions. Various additional so-called signs are given in our text-books but are for the most part inconstant and unreliable as to exact sites.

The Paradoxes Explained.—The reason for these variations, with respect to friction, heart sounds and apex-beat alike, is found in the fact that, in the case of deep-chested individuals, especially, even a massive effusion need not separate the greater portion of the anterior surface of the heart from the chest wall.

Importance of chest diameters.

This is well demonstrated by autopsies performed upon such individuals, which show a large pericardial exudate with a free heart surface *in apposition to the anterior chest wall*, or, one covered only by a thin layer of fluid, even in the absence of such limiting adhesions as sometimes of themselves limit the spread of the fluid.

Limiting adhesions.

If the apex-beat remains visible and if by percussion it is found to lie well within a sharply defined left border of dulness, another valuable sign is added.

Furthermore, it may show under such conditions a most prompt and excessive mobility in lateral changes of position.

Diagnostic Value of the Quality of the Percussion Note.—The character of the light-stroke percussion dulness in marked effusions is usually pathognomonic.

Use light percussion.

It represents a marginal area of unusually decided relative dulness together with an internal area of extraordinarily large extent which shows superficial dulness or actual flatness.

Marginal and inner zones.

This sign is often much intensified if the patient is able to sit up and lean forward; so also the *intensity* of left border dulness or of that of the right may be distinctly increased when the patient turns upon the one or the other side respectively. The former procedure is hazardous in bad cases.

Postural intensification.

FACTORS FUNDAMENTAL IN DIAGNOSIS.—The diagnosis of pericarditis is greatly furthered by a full knowledge on the part of the physician of the etiologic factors which make clear the circumstances under which it may be anticipated.

Especially should he remember its extraordinary frequency as a complication of the acute cardiac diseases of children and of acute rheumatism at any age. Its relative frequency in severe pneumonias, in terminal tuberculosis and in the later stages of chronic Bright's disease and of scorbutus should never be forgotten.

Dry Pericarditis.—The direct recognition of a fibrinous pericarditis depends wholly upon our ability to detect the presence and recognize the nature of pericardial friction rubs. There is no other one of the symptoms save friction fremitus, which constitutes its direct expression.

Obscure Pericarditis with Effusion.—In a majority of instances of pericarditis with effusion and in a large proportion of such of the cases as occur in private practice, especially, the physician would be able to recognize the primary lesion by the presence of a friction sound. In many cases, nevertheless, and especially those occurring in public clinics amongst the poor, the patient presents himself with a fully developed and often extreme effusion and the case must be diagnosed on the basis of symptoms heretofore described as characteristic of pericardial exudation irrespective of the friction rub. A

misleading factor peculiarly frequent in public services is the relative lack of severe subjective symptoms of any kind in many of these cases.*

Chief diagnostic points.

In most instances of large effusion the decided character of the dulness, its outline, the extent of its upward projection, and the fact that it passes beyond any visible apparent apex-beat, or, the entire absence of the apex impulse and marked faintness and remoteness of the heart sounds, suffice for diagnosis. Nevertheless, that which seems so simple in written description may prove difficult and impossible now and then in practice. Recourse to the X-ray usually reveals a typical shadow of the outline indicated in our illustration and there remains always recourse to aspiration, though this must not be lightly undertaken.

Conservatism indicated.

Technic of Aspiration.—*In event that this is necessary the needle may be introduced most safely into the angle or crevice formed by the ensiform cartilage and the rib margin on the right side, the needle being directed toward the point of the patient's right shoulder. Very few accidents are reported in relation to this procedure, but now and then some highly skilled clinician introduces the needle into an acutely dilated heart.*

Many prefer the 5th or 6th left interspace at the sternal margin, and some go far to the left.

The matters of chief importance are:

First.—That the operation actually be necessitated by the urgency of compression symptoms. The procedure is not to be entered upon hastily or for slight reasons.

Second.—That the heart sounds are muffled or inaudible at the site chosen.

Third.—That neither pulsation nor friction be detectable at the point selected.

Fourth.—That a very sharp needle be used, local anesthesia secured, and a steady, unhasty, yet unhesitant puncture made.

PROGNOSIS IN ACUTE AND SUBACUTE PERICARDITIS.—Crucial Factors in Prognosis.—The nature and extent of antecedent or complicating ailments, the direct etiologic factors and the character and amount of any secondary exudate, constitute the elements of chief importance in the prognosis of this disease. The mortality rate varies greatly in different clinics or as between these and private practice amongst well-to-do classes, but is relatively high, if the cases are taken as a whole, and may reach 40 per cent. amongst the poor. The high death rate is not due to pericarditis itself but to the antecedent underlying or complicating conditions together with the defective physical constitution and resisting power of the patient, a factor

High mortality.

* As an example, the author would quote the case of a railroad brakeman who sought relief from a slight dry but persisting and irritating cough. He was distinctly cyanotic, decided dyspnea was evident on exertion and the cervical veins were prominent. He had been on duty, but had experienced some precordial oppression which "slowed him down." The cough was almost constant and of that peculiar brazen quality which always suggests to the trained ear pressure within the mediastinum and usually aneurysm of the aortic arch. In this case, however, a massive effusion was present, its left boundary extending apparently nearly to the thoracic wall, its right reaching 8½ cm. beyond the right edge of the sternum. Three weeks previously this patient had suffered from a follicular tonsillitis and ten days before his visit had had a trifling swelling of one ankle-joint.

varying enormously in a great number of determining conditions. Of the many symptoms which convey a warning to the attending physician one may enumerate the following:

(*a*) *A tendency to drowsiness, delirium or persistent insomnia.*

(*b*) *An unusually high and resistant fever or a total absence of febrile reaction in cases in which feeble resisting power is combined with serious subjective symptoms and extreme physical manifestations of the disease.*

(*c*) *The preëxistence of a badly damaged heart or vascular system and antecedent or concurrent signs of failing compensation.*

(*d*) *Evidence of pancarditis.*

(*e*) *High-grade cyanosis; rapid onset of either cyanosis or pallor, and a decided increase of precordial pain or distress, with a stationary exudate, or, one persisting after drainage of the pericardium.*

(*f*) *Excessive rapidity of the heart, especially if paroxysmal; the occurrence of the pulsus irregularis perpetuus indicative of fibrillation of the auricle or a running beat of great feebleness.*

(*g*) *Inability to maintain the obligatory sitting posture even though orthopnea be present.*

(*h*) *Sighing, Biot, or Cheyne-Stokes, respiration which may indicate renal involvement or myocardial insufficiency.*

(*i*) *Decided or rapidly progressive diminution in the urinary excretion, after the pericardial exudate is established.*

(*j*) *An excessively rapid and extreme filling of the pericardium. This rare event may terminate life in a few hours if unrelieved.*

(*k*) *The presence of a purulent or sanious effusion. As previously stated, these cases demand immediate radical surgical relief and only in rare instances can recovery take place without it.*

(*l*) *Persistence or sudden onset of extreme pain in a case of established effusion; great precordial or epigastric distress or oppression, sudden extreme physical weakness. These usually indicate decided myocardial involvement and inasmuch as the diastole of the heart and its intrinsic circulation are already embarrassed by the intrapericardial pressure of massive exudates it constitutes a serious and usually fatal complication though under prompt aspiration recovery may take place.*

(*m*) *Syncopal attacks. The gravity of such a complication needs no discussion.*

(*n*) *Manifest hepatic engorgement, excessive turgidity or direct systolic pulsation of the jugulars and progressive edema are one and all symptoms of decompensation resulting from a weakened toxic insufficient myocardium.*

(*o*) *A listless rolling of the head from side to side when the patient is sitting propped up on his pillow is sometimes observed in pericardial effusion as it is in terminal cases of mitral disease and decompensation and conveys the same prognostic meaning, viz., the probability that death is impending or not far distant.*

(*p*) *Renal complications chronic or acute.*

(*q*) *The development of valvular murmurs whether these result from myocardial weakness or an actual complicating acute endocarditis.*

The one is a direct indication of threatening myocardial insufficiency, the other strongly suggests a pancarditis.

In general, the murmurs of an established acute endocarditis are louder and more definitely transmitted than those of myocardial weakness alone.

(*r*) *Excessive pulmonary engorgement or the onset of the liquid râles of edema.*

(*s*) *Persistent and exhausting hiccough.*

Finally, if should be remembered that in every case of dry pericarditis, of whatever duration, an effusion should be sought for most diligently at each visit.

ANEURYSM OF THE HEART.—This rare condition may be saccular and associated with coronary sclerosis, acute or chronic mural (viridans) endocarditis or acute myocarditis, or gummata. As a clinical curiosity dissecting aneurysm may be encountered. Death occurs usually from rupture and the disease cannot be diagnosticated antemortem. In almost every instance, it represents an antecedent coronary infarct and consequent *myomalacia cordis.*

RUPTURE OF THE HEART.—*This extremely rare condition occurs only in persons having a degenerated heart muscle* and, in such, may result from a fall, strain or other traumatism, such as is apparently insufficient to produce such an accident. Usually it occurs in the anterior wall of the left ventricle, rarely in the right, and causes death instantly or within a few moments, preceded by severe pain, oppression and symptoms of collapse. This condition is medico-legally important.

FOREIGN BODIES IN THE HEART.—The organ is much more tolerant than is generally supposed; exploring needles, sewing needles, hat pins and even a knife or bullet may not cause death, indeed a small needle may be introduced with a minimum risk of a fatal result and it has even been suggested or actually employed as a therapeutic *dernier ressort* for cardiac stimulation. According to W. G. Thompson, war records show an astonishing *exemption* of the heart from bullet wounds, though the statement is based upon the Civil war records which antedated the use of high-penetration projectiles.

NEW GROWTHS OF THE HEART.—New growths, tuberculosis, syphilis, various forms of sarcoma and carcinoma, and more rarely the myomata, lymphomata or fibromata may involve the heart. It may be the seat of abscess in pyemia, malignant endocarditis and acute myocarditis, or the seat of degenerative or cysticercus cysts.

SITUS VISCERUM INVERSUS.—As a part of the general transposition of organs occasionally observed as a congenital phenomenon we may find the heart upon the right side, the left ventricle being anterior and the apex in the usual relation to the nipple, as in its normal position. Such transposition is sometimes complete as regards the other organs, sometimes partial. Apparent transposition of the heart is common, but usually proves to be due to a combination of unilateral pressure and lack of support.* As a temporary condition extreme degrees of displacement may be associated with pleural effusions.

* In a case seen with Dr. B. J. Merrill, apparent complete displacement was due to almost complete tuberculous excavation of the right lung, with enormous hypertrophy of the left.

DISEASES OF THE ABDOMINAL ORGANS

THE ABDOMEN. (*Abdo, I conceal*).—*Excepting the brain, this ever has been the most obscure region of the human body because of the shifting, air-containing, obscurant intestinal coils, its inaccessibility to direct examination, and the comparatively slight value of certain of the physical methods so valuable in thoracic diseases.*

Obscure region.

On the other hand, the recent astonishing development of clinical and laboratory methods and especially of the roentgen-ray technic, together with a better understanding of the meaning, scope and therapeutic needs of the so-called functional disorders of the abdominal viscera, have greatly advanced the diagnosis and treatment of the more common and important intra-abdominal lesions and as a result the legitimate field of the exploratory incision has been greatly contracted and careful and painstaking medical methods, both diagnostic and therapeutic, are replacing the former all too precipitate tendency to resort to operation.

Modern advance.

Lessening surgical precipitancy.

TECHNIC OF ABDOMINAL EXAMINATIONS.—The customary preliminary cathartic is necessary in advance if the X-ray is to be used or tympanites is present.* It removes misleading fecal masses, but may produce a flatulent distention and troublesome peristaltic activity if abundant time is not allowed for its immediate effects to subside.

Catharsis.

A thorough flushing by enemata is usually sufficient and preferable save in preparation for special forms of roentgenographic examination.

Enemata.

Measures Preliminary to Palpation.—*The head and shoulders both, but particularly the former, must be high,* the feet firmly placed and the patient in an easy and unconstrained dorsal posture.

Indispensable preliminary.

To relax the walls, it is usually stated that the knees must be drawn up, but oftentimes, as good or better relaxation may be had with the legs extended or but slightly flexed and furthermore the flexed thigh and knee are much in the way.

The breathing of the patient should be deep but easy and natural, with the mouth open and his attention should be diverted by conversation and inquiry.

The hands should be warm, the light should be adequate, its source behind the patient, and no clothing or other covering should be allowed to obstruct or embarrass the examiner.

In some cases nothing less than a full hot bath or even general anesthesia suffices to establish full relaxation.

Proper Use of the Hands.—*The flat of the palm and fingers,* not the tips, should be used with a sliding motion. Tender areas should not be immediately approached, *nor does the discovery of one justify the painful repeated and purposeless punching and poking* so often inflicted upon the long-suffering patient.

A common imposition.

* Many conditions are so frankly shown as to make unnecessary any such preliminary procedure and in the usual first examination of the patient it is customary to omit such troublesome preliminaries. For the formal and critical examinations proper preparation is indispensable.

The deep or superficial character of any pain or tenderness developed by examination and its precise localization or extent of diffusion should be accurately determined, but useless and unnecessary maneuvers should be avoided.

Respiratory mobility.

In palpating movable organs or growths respiratory movement should be met directly by the tips of the fingers at right angles to the free margin, the hand being firmly but gradually depressed in deep expiration and allowed to rise laggingly and shift slightly upward to meet the descending border in inspiration.

Vital points

The fingers should always be relaxed to the utmost, otherwise the tactile sense is distinctly blunted. Once the hand is laid upon the abdomen, it should remain there and all movements made to intercept a viscus should carry the surface tissues upward with the hand. Allowing the hand to glide over or to leave the surface is a violation of sound technic.

If the organ or growth under examination is both movable and capable of being grasped bimanually, the opposite palpating hand should be almost motionless as the other brings the organ or tumor to it.

After tolerance to palpation is established, the palmar surface of the terminal phalanges may be used to determine consistence and outline.

Involuntary Abdominal Rigidity.—This common and serious obstacle to palpation may be transient and easily controlled by diverting the attention of the patient and having him breathe slowly and count aloud the respirations or it may require the use of Galambro's bimanual technic. This is nothing more than an attempt to forcibly overcome the rigidity by upward pressure at distance, while the other hand carries out the palpation.

Galambro's method.

The Fat Belly.—A very fat abdominal wall, even though in a state of maximum relaxation, may interpose most serious or even insuperable difficulties. These may usually be overcome in considerable measure by the double-hand-palpation method of Hausmann. This consists merely in laying the otherwise unemployed hand directly upon the palpating hand and relieving the latter of all necessity for exerting pressure, to the end that it may remain relaxed and maximally sensitive.*

Useful device.

Dipping.—This special procedure is of use in detecting the presence of tumors or for determining the outline and consistency of the spleen or liver under conditions associated with the presence of fluid in the peritoneal cavity.

A valuable maneuver.

It consists of abrupt shallow or deep downward thrusts with the finger tips according to the relation of the organ sought to the surface.

Malingering or Hysterical Patients.—Such may often be misled or diverted by conversation and especially by rapid, emphatic interrogation. Abruptly directing their attention to some indifferent region which is being explored by one hand while the other palpates the supposed tender point, and the making of leading or even misleading, positive false statements as

* These methods are thus *named* in order that they may be recognized by the student when so described elsewhere.

Both are old procedures which have been employed probably ever since man became interested in the physical examination of his sick brethren. At all events the author can certify to their common use since the late eighties of the nineteenth century.

to the necessary or anticipated effect of pressure, forced respiration, and the like often exposes spurious symptoms.

The dorsal posture is the best for general purposes, but the lateral is of value for palpation of the spleen, the detection of movable fluids and floating kidney, or for the purpose of "spilling" the intestines of a large-bellied person and rendering more accessible the lateral region under examination. Lateral posture.

The patient sometimes with advantage may be placed upon the hands and knees or in the knee-chest position to bring a growth forward against a thin and relaxed abdominal wall or to demonstrate the otherwise obscure effusion. Knee-chest.

All portions of the abdomen should be thoroughly examined, not forgetting the hernial openings, the stretching or patency of which no less than actual hernias may be the cause of much obscure pain, usually absent when the patient is recumbent and increased by standing and by cough. Hernial openings important.

POINTS TO BE DETERMINED.—(*a*) *The conditions of the organ under observation* (size, outline, condition of surface, mobility, tenderness).

(*b*) *The presence of swelling or tumors* (intra- or extra-abdominal, size, outline, surface, mobility, tenderness, associated symptoms).

(*c*) *Presence of pain or tenderness* (true or false, intensity and character, exact location, superficial or deep, and the effect of pressure). Pain increased on pressure ordinarily suggests inflammation, but the utmost care and caution are necessary to avoid being misled by spurious or referred pain, tenderness and even an apparent defensive rigidity, the true source of which may be an inflamed pleura or a diseased heart.

General Inspection.—*The nutrition, musculature and abdominal outline are first noted.*

General distention may be due to tympanites, ascites, chronic tuberculous peritonitis, great dilatation of the stomach or colon, advanced pregnancy or large ovarian tumor.

Localized distention or swelling suggests hysteria (phantom tumor), ventral hernia, abscess, intestinal obstruction, new growths, dilatation or displacement of the stomach.

Abdominal Flaccidity.—One of the most important of the stigmata of congenital asthenia and its associated visceroptosis is the decided loss of tonus in the abdominal musculature. The degree of flaccidity depends upon the grade of the constitutional defect primarily, and secondarily upon the general state of nutrition at the time of examination and the occupation of the individual.* This is a condition apart from that noted below. An important stigma.

The flaccid pendulous abdominal wall of middle-aged women who have borne children may reach an extreme degree of relaxation and if nutrition has become impaired the tissues may literally hang in a fold. In such cases diastasis (separation) of the recti is frequent. "Hanging belly."

In all cases the tonus and structural continuity of the abdominal muscles should be tested by asking the patient to raise the head and shoulders alone, while lying in the full dorsal recumbent posture. Useful test.

* One should note particularly the absence of a good musculature in hard working men and women who should show, normally, decidedly firm muscles.

Surface Appearances.—The *skin* may show an acute exanthem, syphilis or skin diseases, areas of pigmentation, atrophy and the various hues and discolorations already discussed under the "outward signs of disease."

Lineæ albicantes.

The white or bluish striæ may indicate respectively past or present distention by tumor, pregnancy, ascites or excessive fat, or the *linea nigra*, a dark median vertical line, may indicate a pregnancy existent.

Linea nigra.

Enlarged Superficial Veins.—*As stated previously, the superficial veins may be enlarged in the case of portal or caval obstruction*, the former indicating hepatic cirrhosis, tumors or prolonged ascites; the latter, thrombosis or the pressure of abdominal growths.

If the superior cava be obstructed, the direction of the blood flow in the enlarged lateral superficial veins (readily determined by emptying one and watching its refilling) will be downward; in other conditions it is upward. In hepatic cirrhosis and portal thrombosis, large veins may radiate from the umbilicus (*Caput Medusæ*) and the blood flows from that as a center.

Caput medusæ.

In obstruction of the inferior vena cava the lateral superficial veins of the abdomen are chiefly affected.

If the visible venous distention is most marked in the pubic surface-segment, obstruction below the liver is suggested.

Congenital Occlusion of the Aorta below the Ductus Botalli.—This condition, indicated by enlargement, prominence and pulsation of the epigastric, scapular and internal mammary arteries, is a clinical curiosity, but usually should offer no great diagnostic difficulties. The collateral circulation may be easily and well maintained because the block lies beyond the area of origin of the great aortic branches. Over the arteries named one may hear murmurs and a palpable thrill may be present.*

The contrast between the strong full pulse in the upper extremities and the faintness or entire impalpability of that of the lower extremities is usually very striking.

Visible Peristalsis.—In the presence of emaciation and a thin and relaxed abdominal wall, peristaltic movements may be noticeable and are readily made more pronounced by snapping the surface with the finger-nail or wet towel, splashing upon it a little ether or applying the faradic current.

How to increase it.

Normally the movements are deliberate and wave-like and occur usually without borborygmi; *but in acute obstruction they may seem to be fiercely attacking the stenosed region.*

Normal stomach peristalsis runs from left to right; that of the transverse colon from right to left, a point slightly diminished in value because of occasional reversed gastric peristalsis in marked pyloric obstruction. In relation to obstruction of the bowel, acute or chronic, the sign is of distinct value; that of its commonest forms, the "ladder pattern" peristalsis, running from one region of the ileocecal valve toward mid-abdomen.† *In sigmoid obstruc-*

Obstructive.

"Ladder pattern."

* See "Congenital Heart Lesions."

† In two such instances observed by the author marked obstruction due to appendicitis existed for a period of thirty years, the diameter of the tube having been reduced to that of a crow quill. In one case the recurrent attacks had been so violent as to have led to an operation for gall-stones, the pain being to some extent referred to the hepatic region.

tion there is increased violent peristalsis along the course of the colon and a *cooing or loud rumbling* associated with any disappearance of distention which at times sharply localizes the point of constriction.

One of the commonest causes of increased visible peristalsis is stenotic dilation of the stomach.

TOPOGRAPHY AND REGIONAL DIVISIONS.—Boundaries.—The spine, inferior costal margin, iliac crest, Poupart's ligament and the pubic symphysis bound the abdomen and the diaphragm roofs its cavity and imparts *respiratory* movement to the liver, spleen, and, less directly, to the kidneys and stomach. A transverse line at the level of the base of the ensiform roughly bounds the abdominal cavity above; the brim of the true pelvis, below.

The Abdominal Quadrants.—*A median vertical* intersecting a *horizontal* line at the navel divides the abdomen into *four quadrants.*

Arbitrary boundaries impossible.

In describing the contents of the abdomen with relation to these arbitrary divisions, one can take account only of normal conditions.

These may be and commonly are greatly modified in the cases of visceroptosis so frequently present in sick and well alike.

The upper right quadrant contains the liver, gall-bladder, head of pancreas, kidney, the hepatic colonic flexure, a portion of the transverse colon and the pylorus.

The upper left quadrant includes the left extremity of the liver, the spleen, splenic flexure, a part of the transverse colon, left kidney, the fundus of the stomach and most of the pancreas.

The lower right quadrant contains the ascending colon, cecum and the vermiform appendix.

The lower left quadrant covers the descending colon and the sigmoid flexure. The uterus and ovaries are anatomically *pelvic* and not abdominal.

The Bony Landmarks.—*The anterior-superior spine of the ilium, iliac crest, pubic spine, lower ribs, costal margin, and the ensiform, are the chief bony landmarks.*

External Landmarks.—*The linea alba and the navel are always in evidence and the rectus muscles*, which are divided into segments by the *lineæ transversæ* and bounded externally by the *lineæ semilunares*, are easily distinguishable in thin muscular abdomens.

Lineæ transversæ and semilunares.

The umbilicus usually lies from 1½ to 2 inches above a line connecting the anterior-superior iliac spines, being opposite the tip of the third lumbar spinous process.

Opposite 3rd lumbar.

The tenth and twelfth ribs and the projection representing the ninth cartilage are also important landmarks.

The abdominal aorta bifurcates opposite the fourth lumbar vertebra ¾ of an inch to the left of and below, the navel.

Bifurcation of aorta.

The ileum and jejunum occupy chiefly the center and lower portion of the cavity, the ileum largely on the left and the jejunum on the right and below.

The colon, somewhat less movable, passes from the cecum upward and

Usual course.

to the right, then turning (hepatic flexure), runs transversely above the umbilicus (transverse colon) under the left costal margin; then passing (*splenic flexure*) downward and inward (*descending colon and sigmoid flexure*) to terminate in the rectum.

The *hepatic flexure* lies under the arch of the liver, the splenic even higher under the left ribs.

Vermiform appendix.

The transverse colon, sigmoid flexure and cecum are relatively superficial and the vermiform appendix lies with its base *at a point representing the intersection of the outer edge of the right rectus with a* line drawn from the anterior-superior iliac spine to the umbilicus (McBurney's point). It lies behind and to the inner side of the cecum with its apex downward, outward, inward, or rarely upward.

McBurney's point.

The position of the transverse colon especially is very variable and it may dip as low as the true pelvis in cases of marked visceroptosis.

The transverse colon, cecum, and sigmoid flexure are accessible to palpation by the method referred to under palpation of the stomach.

For the *transverse colon* the fingers should be directed upward at various levels for it may lie as low as the hypogastrium in visceroptotic patients.

It does not *move* with respiration, but is quite freely *movable* and feels like a cord under the fingers.

The cecum is palpated by passing the fingers deeply downward along the inner aspect of the ilium at the anterior-superior spine and pushing toward the navel and backward with gliding movements.

It is somewhat easily palpated and is quite often movable (*"cecum mobile"*). A characteristic gurgle usually attends its manipulation and severe pain elicited by moderate but firm deep pressure upon the gut, toward the navel, raises at once the question of chronic appendicitis.

The sigmoid flexure may often be palpated as a thin somewhat tense cord just above the fold of the left groin. Its tumors ordinarily lie too low to be reached by the fingers.

The Liver and Gall-bladder.—The normal percussion outline of the liver is shown by the following table.*

	Middle line	Mammillary line	Midaxillary line	Scapular line
Upper limit	Deep dulness	Fourth space	Seventh space	Ninth space
	Superficial dulness — Blends with heart dulness	Sixth rib	Eighth rib	Tenth rib
Lower limit	Hand's breadth below base of xiphoid	Costal margin or somewhat above or below it	Tenth space	Blends with kidney dulness

Left lobe and notch.

It should be noted that the left lobe extends nearly to the left nipple line, the interlobar notch lying in the median line. In children the liver lies lower, in old persons either higher or lower.

* Hutchinson and Rainy: "Clinical Methods."

The Gall-bladder.—This lies just without the right rectus under the projection of the ninth costal cartilage. Border of rectus and 9th cartilage.

The Spleen.—This important organ lies just outside the spine in close relation to the diaphragm above the left kidney, posteriorly, and the colon, stomach and small intestines below.

It is oval, flattened, from 3 to 5 inches in length and lies parallel to and within the area of the ninth, tenth and eleventh ribs, its anterior margin reaching normally a line dropped from the *sterno-clavicular joint to the point of the eleventh rib*, two-thirds of its outer surface being parietal and lying between the posterior and mid-axillary lines. Anterior relations.

The Kidneys.—*These lie behind the peritoneum upon either side of the spinal column on the muscular bed formed by the lumbar muscles. They are in virtual contact above with the liver on the right side and the spleen on the left and move laggardly and to a slight degree with respiration.*

The right kidney is slightly lower than the left because of the overlying and impinging liver and each lies two-thirds within the mid-clavicular line. The lower border of the right is 1 inch and that of the left 1½ inches above the level of the navel.

Posteriorly, they lie within a parallelogram represented by two verticals drawn 1 inch and 2¾ inches respectively, from the spinous process, and connected by two horizontals representing the eleventh dorsal and third lumbar spines. Posterior relations.

Therefore, about one-third of their surface is covered by the eleventh and twelfth ribs. Their lower extremities lie from 1 to 1½ inches respectively above the crest of the ilium.

THE LIVER.—Inspection sometimes shows a lower-border shadow descending with deep inspiration, a very definite bulging if the enlargement be great, and not infrequently, in malignant disease, the respiratory descent and ascent of nodules upon its surface. *The normal percussion* outlines have already been given. Simple *auscultation* is of little value save in the detection of *peritoneal friction* in inflammatory lesions or abscess, and the most useful physical method is *palpation.*

Palpation.—*The normal liver can often be palpated easily if the abdominal wall is relaxed and the liver edge is met by the fingers as the firmly depressed hand rises laggardly with the wall in deep inspiration.* Technic.

Directness and immediateness of imparted vertical movement in response to forced inspiration are characteristic of the liver as compared with the slightly delayed descent of the movable kidney or pyloric growth, or the diagonal descent of the spleen. It is absent only when adhesions exist or diaphragmatic descent itself is limited or absent. Type of mobility. Fixation.

A firm resistant liver edge suggests cirrhosis, chronic congestion, amyloid disease or tumors, and is found in many of the diseases associated with chronic enlargement and induration of the spleen. Rarely in adults and more often in children, *the liver may pulsate directly and expansively* from tricuspid regurgitation, and yet more often there appears a *transmitted pulsation* due to a dilated overacting right ventricle. Consistence. Expansile pulsation.

Reduced areas.

Percussion.—*A reduced area of percussion dulness is especially significant, if it involves the region of the left lobe, as this is earliest affected in atrophic cirrhosis.*

Diminution of the upper (thoracically sheltered) liver area may indicate hepatic contraction but is more often due to changes outside of the liver itself, such as emphysema of the lung, pneumothorax or, very rarely, diaphragmatic hernia.

By reason both of the respiratory mobility of the liver and the descent of the lung border this area varies with deep breathing.

Valuable method.

Auscultatory percussion is especially valuable in outlining the left lobe or in deciding the origin or attachment of a tumor. The stethoscope is placed over the thoracic area of *superficial* hepatic dulness while the finger taps the surface in radiating lines until the decided change of note indicates that hepatic tissue has been passed.*

Attached tumors will usually yield the hepatic note.

Under certain conditions great enlargement of either the spleen or liver may be overlooked if palpation is not commenced well below the normal boundary.

Modified percussion areas.

Conclusions.—Upward extension *of dulness* in the hepatic area suggests not only enlargement of the liver, but also pleural effusion or adhesions, pulmonary consolidation, hepatic or subphrenic abscess or displacement due to upward pressure and thrust of gas and liquid in the abdominal cavity.

Fictitious changes.

Diminution of upper boundaries suggests not only hepatic atrophy but asthma, emphysema, pneumothorax, gaseous distention of a subphrenic abscess, hepatic ptosis and rarely, massive pericardial effusion or extreme cardiac dilatation.†

General enlargement may be due to passive congestion of cardiac origin, the commonest cause, to hypertrophic cirrhosis, leukemia, erythremia, Banti's disease, fatty liver, amyloid disease, syphilis, cancer, abscess, or pyemia.

General shrinkage suggests atrophic cirrhosis, the commonest cause, with acute yellow atrophy as a rare possibility.

Ascites or tympanites.

It is obvious that either ascites or tympanites may force the liver upward and prevent any true and exact delimitation of its actual boundaries or proper examination of its surface.

Irregularities in Surface Outline.—Such may be due to congenital defects, various enlargements and displacements, atrophic cirrhosis and indeed practically all of the conditions noted in a preceding paragraph.

Nodules.

Of special significance are the nodular irregularities (sometimes showing

* The reversed method, *i.e.*, percussing toward the organ is preferred by many and one should use the one to confirm the results of the other.

† Often, a downward displacement of the liver itself is associated with the two latter conditions, although this may be detectible only through the position of the lower border.

The author has seen three cases of enormous *aneurysm of the ascending portion of the aortic arch which blended their anterior dulness with that of the liver.*

crateriform depressions) associated with secondary carcinoma or a tertiary syphilis.

Glissonian cirrhosis may produce marked shrinkage and deformity, whereas the tiny granular elevations of *atrophic cirrhosis* can be palpated only under exceptional conditions, as when the wall is thin and lax, *though the sharp resistant liver edge of an advanced lesion is striking.*

Fluctuating or elastic swellings or tumors modifying the outline are due usually to a distended gall-bladder, associated perhaps with a Riedel's lobe. Abscess or hydatid cyst are among the unusual findings, the last yielding sometimes a peculiarly exquisite thrill (hydatid thrill), echoing the percussion stroke.

Riedel's lobe.

Hydatid thrill.

It should be remembered that a greatly enlarged liver, easily palpable, with a smooth hard surface, suggests leukemia and many other splenomegalic conditions, amyloid disease, passive congestion of long duration, hypertrophic cirrhosis, fatty liver, certain tropical diseases and interstitial forms of hepatic syphilis.

Excessive enlargement.

Roughening suggests cirrhosis or tuberculosis and decided nodulation represents usually carcinoma or syphilitic gummata.

Roughened surface.

Finally it will be noted that mere increase or diminution in the hepatic area yields no information as to the actual size of the organ unless checked carefully with the conditions present in contiguous structures.

Caution.

Friction Sounds.—*If any condition causing perihepatitis exists friction sounds may be audible upon auscultation.*

THE GALL-BLADDER.—*Palpable only when distended, it may then form a pear-shaped tumor, smooth and elastic, anchored above but more or less freely movable from side to side, and, in the absence of adhesions, rising and falling in respiratory rhythm with the liver itself.*

Though seldom larger than the fist, these gall-bladder tumors may sometimes extend as low or lower than the level of the navel and are often mistaken for other conditions.*

May form large tumors.

One often feels a depression or groove corresponding to its emergence from beneath the edge of the liver and a narrow tongue-like projection of hepatic tissue may overlie it or be manifest when the distended gall-bladder itself is not palpable. This is the so-called Riedel's lobe and its presence may prove of value when a loop of gut obscures the outline of the gall-sac itself.

Source of error.

Rarely a palpable crepitation may be obtained bimanually if gall-stones are present and the viscus may be nodular from malignant disease or, in long-standing cholecystitis, may undergo atrophy.

A rare finding.

In cholecystitis, quite apart from sensitiveness and defensive rigidity over the gall-bladder or liver itself, a zone of distinct tenderness may extend from a point 2 to 3 cm. to the right of the vertebral column at the level of the eleventh or twelfth dorsal vertebra across the infrascapular region and sometimes into mid-axilla.

An occasional finding.

THE SPLEEN.—A greatly enlarged spleen and more rarely one scarcely more than palpable may be seen to move with the respiration and, further-

* These tumors in rare instances attain an enormous size. Alban Doran has reported one simulating ovarian cyst (British Medical Journal, June 17, 1905).

more in any case of splenomegaly, a marked prominence of at least the left upper abdominal quadrant is produced.*

As in the case of the liver, auscultation may reveal friction sounds in the presence of perisplenitis, or the organ may be anchored by adhesion, *but it is ordinarily freely and directly movable with respiration, and palpation is the only method yielding dependable and accurate results.*

Proper attitude.

The position of the patient should be right lateral if minor enlargements are to be noted, as in typhoid fever or other acute infections, and the one hand should make pressure over the lower ribs in the post-axillary line while the other makes palpation.

Abdominal distention defeats palpation save in great enlargment and, except in splenoptosis, the normal spleen is not palpable *save in the rarest instances even when the left cup of the diaphragm is forced downward by liquid of serous effusion.*

If greatly enlarged, the dorsal position is to be preferred and it would seem that the condition can hardly be overlooked,† *unless, with a tense wall, the careless or hurried examiner fails to get below the actual border or to distinguish between muscular resistance and the splenic mass.* In these cases the chief notch in the anterior border is sharply defined and quite distinctive.

Students at first palpate too deeply and let the hand rise too slowly with inspiration to detect the minor enlargements.

"Gripping" the spleen.

In the examination of both the spleen and kidney many prefer to grasp the flank with one hand rather than employ bimanual palpation.

The student should use both methods and note and compare the results attained.

Important data.

Differential Factors.—*In all cases where doubt arises as to the nature of a large tumor in the splenic area it should be remembered that a splenic growth or tumor is peculiarly superficial (in close apposition to the abdominal wall) and so accurately applied to the thoracic edge that the finger cannot be thrust between. Its dulness is marked and can be carried directly back to its normal area behind the ninth, tenth and eleventh ribs; it ordinarily moves directly with respiration, and, if the colon be inflated, splenic tumor dulness is not lost.*

Renal tumors extend more deeply, are relatively fixed or immovable, are likely to be reniform or nodular and in nearly every instance will be crossed by an *area of resonance* if the colon be distended artificially.

Obscurant factors.

Emphysematous lung or a pneumothorax may cause an apparent diminution in the *upper* percussion area of the spleen or the pressure of tympanites, ascites, or even a widely dilated stomach or colon, may confuse the findings save in massive enlargement.

Chronic splenomegaly.

Causes of Enlargement.—*Excessive chronic enlargement* may be due to chronic malaria ("ague cake"), the leukemias, splenic anemia, a splenic form of Hodgkin's disease, the Gaucher type of splenomegaly, congenital hemo-

*In a case shown by the author to the Minnesota State Society a leukemic spleen filled the entire abdomen, actually reaching the right anterior superior spine of the ilium, and, as in many similar cases, the patient showed then no impairment of the general health, pallor, or malnutrition, and declined to cease work to take treatment.

†As a matter of fact it is quite often overlooked or mistaken for some other form of abdominal tumor.

lytic jaundice, Banti's disease (splenomegaly with hepatic cirrhosis), chronic tuberculosis, syphilitic hepatitis, amyloid, certain cases of atrophic or hypertrophic cirrhosis, sarcoma, carcinoma, kala-azar, rarely echinococcus cysts or the Vaquez-Osler "erythremia."

Lesser degrees of enlargement may be encountered in ordinary atrophic cirrhosis of the liver, rickets, pernicious anemia, passive congestion or actual portal obstruction. Lesser enlargement.

Acute enlargement is most often encountered in septicemia, acute lymphatic leukemia, malarial fever, typhoid and typhus, erysipelas, acute miliary tuberculosis, tuberculous peritonitis, cerebro-spinal meningitis, smallpox, diphtheria, scarlet fever, relapsing fever, infarct, plague and certain other tropical diseases. Acute engorgement.

Enlargement of the spleen is nearly always uniform, abscess being an occasional, and hydatids or carcinoma an excessively rare cause of irregular outline. Outline.

Splenoptosis.—As a part of extreme visceroptosis, the spleen may be quite freely movable or actually "floating" and readily displaceable through change of posture or manipulation. "Movable spleen."

Its notched border, peculiar soft consistency and oval contour when normal but displaced serve for its identification.

KIDNEYS.—*The normal kidney yields no results to inspection or auscultation and indefinite ones to percussion.*

When enlarged by disease, it may be present itself in the anterior or posterior lumbar region and be readily accessible to percussion and palpation.

The sarcomata in children and cystic, tuberculous or hydronephrotic kidneys in the adult, are the most frequent causes of enlargement.

Palpation.—The dorsal and *semidorsal** postures are the best, and, in the latter, relaxation is assisted by supporting the shoulders and buttocks and bringing the hand and arm of the side under examination obliquely downward and forward to hang relaxed across the abdomen. Posture.

One hand of the examiner makes pressure over the floating ribs or just below them posteriorly, while the other is pressed deeply downward and slightly upward, outside the rectus border at or below the level of the umbilicus and rises *laggingly and slightly* with each forced inspiration or cough. Technic.

If the kidney is abnormally movable, it will slip downward in deep inspiration oftentimes so as to be grasped between the tips of the apposed fingers of the two hands. Degrees of mobility.

The normal kidney yields to palpation only its lower pole and this only under favorable conditions. Any greater degree of mobility constitutes movable kidney.

If the organ is so movable and displaceable as to suggest a mesonephron, it is a "*floating kidney*." Such are often overlooked because at the moment they occupy some distant portion of the abdomen and may fail to move with respiration. Floating kidney.

In extremely rare instances such a kidney may become fixed in its abnormal location. Floating kidneys have been reported as occurring in hernial sacs and, rarely, they may cross the median line. Sources of error.

* The patient lying obliquely on the side *opposite* the one under examination.

A useful posture.

It is sometimes advisable that examination should be made with the patient leaning forward in the standing posture with the elbows or hands supported on a chair or table.

This method sometimes reveals a far greater mobility than had been demonstrated by the usual technic.

One oftentimes may approximate the tissues even above a movable kidney and feel its upward movement as it slips between the finger and thumb in expiration or when the patient slowly assumes the dorsal position.

The head of the patient must be sharply flexed upon the chest if full relaxation is to be secured.

Ballottement Renal.—(*Guyon*).—This special bimanual maneuver may bring the kidney into touch when other methods fail.

Technic.—With the two hands in the usual position the fingers of the posterior (inferior) hand dip sharply into the loin and thrust the kidney forward on its muscular bed and within the reach of the finger tips of the anteriorly placed hand. The short, rapid, regular thrusts, recur in quick succession in order to permit determination of the kidney outline.

Albuminuria of manipulation.

The maneuver need not be excessively uncomfortable to be effective, but as in most instances of renal palpation albumin will appear in the next voiding of urine and should not mislead the observer.

Twelfth Rib Pressure Point.—Firm pressure over the twelfth rib made whilst the patient is sitting erect or *dorsally* recumbent with relaxed muscles will often elicit a pain response or only an abrupt marked defensive rigidity of the overlying muscles, if the kidney is the seat of *active irritation* or inflammation acute or chronic. The sign often occurs in renal calculus lacking all active symptoms.

It is well to bear in mind, however, that this particular area is normally more sensitive than the regions about it.

Of little value.

Percussion.—The semidorsal position is best adapted for percussion *which is usually superfluous, uncertain and often misleading.* Occasionally displacement is suggested by the substitution of tympany for normal dulness or an unusual extension of the latter may point to diseased conditions but the region is ill adapted to dependable percussion results.

Form.

Characteristic Features.—*The reniform shape and peculiar consistence of the palpable or movable kidney are readily recognized unless the organ be markedly inflamed or the seat of a new growth.*

Surface.

Under these latter conditions the tumor is readily palpable usually and may be relatively or absolutely fixed, regular or irregular in outline, smooth or nodulated, according to the nature of the morbid process.

Lagging descent.

If the organ be merely movable, as is so often the case, its maximum displacement occurs near the end of full inspiration, a fact readily explained by its relation to the diaphragm and the directly movable spleen or liver.

Replaceability.

Furthermore, such displaced kidneys are replaceable into the flank or normal kidney position and, as stated previously, in fixed renal tumors artificial inflation of the colon usually establishes a band of resonance crossing or wholly obscuring the percussion dulness anteriorly.

Abscess.

Secondary abscess in the renal region is not uncommon as a sequel of neglected appendiceal abscess, empyema, subdiaphragmatic abscess, caries of the vertebræ, etc.

Points of importance.

One must consider also the globular elastic or possibly fluctuating tumors of hydro- or pyonephrosis; the irregular cystic tumors; the denser more nodular malignant growths and the bogginess or induration of the perinephritic suppurations which tend posteriorly rather than anteriorly, in contradistinction to renal tumors.

Chemic and microscopic examination of the urine, careful consideration of primary factors and the rtional use of tahe aspirating needle assist diagnosis.

THE PANCREAS.—Only in the rarest instances can the head of the fixed, deep-seated, normal pancreas be detected at a point 3 to 4 cm. to the left of the navel, and this only by palpation in those carrying an extremely thin and relaxed abdominal wall and extreme downward displacement of the stomach.

HELPFUL DATA RELATING TO ABDOMINAL TUMORS.—Certain special points of diagnostic value may be stated here with propriety even though the main characteristics of tumors with relation to physical diagnosis are given in the discussion of individual viscera or their diseases.

Fecal Impaction.—If impacted fecal matter is palpably of soft doughy consistence, this fact alone suffices usually for its recognition. Unfortunately this is not always the case and frequently masses of almost stony hardness are encountered and may and do cause serious and damaging diagnostic error in many instances.

"Stony" masses misleading.

These masses may present themselves in any portion of the great gut from the cecum to the anus.

In form, feel and consistence they may exactly simulate malignant growths.

Channeled impactions.

They may be present as channeled masses in patients reporting one or more daily bowel movements though constipation is the rule.

In some instances they resist to an extraordinary degree the action of ordinarily efficient cathartics.

Co-existing with actual new growths of the intestines they may lead to erroneous conclusions as to the form, size and consistence of the true growth itself.

Mobility variable.

They may or may not be quite freely movable. This rather common mobility should not be considered as an absolute finding. Mobility depends upon the segment of gut involved and the presence or absence of marked visceroptosis. Cecal impactions, for example, are usually immobile but may be quite freely movable if the "*cecum mobile*" exists.

The following points aid greatly in the differentiation of fecal masses:

(*a*) *There is usually a history of constipation.*

(*b*) *The tumors are as a rule of doughy consistence and take slowly the forcible sustained impress of the finger much as does the "leathery edema" of the legs in certain cardiovascular cases.*

(*c*) *The most frequent sites are the hepatic, sigmoid, and splenic flexures and the colon. The impactions are often multiple, tend to follow the known anatomic direction of the portion of the gut involved and to assume a cylindrical form. This point is subject to many exceptions but of value nevertheless.*

(*d*) *Hard impactions may crumble under a persistent manipulation.*

(*e*) *They, as a rule, are less tender than either benign or malignant new growths. Exceptionally they are extremely sensitive but in such cases there is likely to be a history of overmuch manipulation antedating the examination.*

(*f*) *They seldom wholly resist free catharsis alone.*

(*g*) *They may show decided change of form and consistency following repeated oil injections or the usual enemata, even if not wholly removed.*

(*h*) *If heavy pressure is made over the mass and gradually released, one may feel the release of the wall from the contained fecal mass.*

This is Gersuny's "adhesiveness sign" ("klebs Symptom"). It is far from impeccable.

(*i*) *However intractable primarily, such tumors disappear almost invariably under such repeated use of enemata and catharsis as is proper in any doubtful case of intestinal tumor, especially if this be combined with the free use of oil injection by mouth and gentle massage.*

Unfortunate precipitancy.

Finally.—Many bellies have been opened only to find a fecal impaction instead of the new growth expected. All cases of *obscure tumor* within the abdomen which resemble new growths but are unassociated with cachexia demand that the measures outlined above be carried out before any positive diagnosis is made.

Indeed an appearance strongly suggesting the cachectic state of malignancy may be present in long-standing cases of fecal impaction.

Segmental spasm.

Phantom Tumor.—Segmental muscle spasm especially of the *rectus abdominis* is sometimes astonishingly like an intra- or extra-abdominal new growth in appearance and, yet more often, a milder transient contraction may falsely suggest *localized defensive rigidity.*

This rare form of phantom tumor is much more deceptive sometimes than the description would indicate, but to the eye and touch of the experienced observer it does not "conform." There is that in its appearance that excites a suspicion which is usually strengthened by its common association with psychasthenia or actual hysteria.

Significant associations.

Crucial test.

Its anatomic situation and limitations are suggestive and helpful, its extra-abdominal nature is usually easily established by the maneuvers already described under "abdominal palpation" and if doubt remains, a few whiffs of ether or chloroform, sufficient merely to produce relaxation, at once removes it.

It is seldom necessary to push anesthesia beyond that early and harmless stage when the arm of the patient, kept steadily at the vertical by repeated command of the physician, drops inertly down.

Confused definitions.

Much confusion seems to exist in the minds of medical writers as to what constitutes "phantom tumor" for there are many transient swellings associated with muscular spasm to which that term may be applied,

and individual choice seems to determine the descriptions given by the authorities.

Spasm of intestinal segments and coincident gaseous distention may account for certain phantom swellings of considerable persistence and size.

Peristaltic knobs.

Mere disorders of peristalsis produce at times a small knob-like projection which may last for several minutes.

Sometimes the name is applied, wrongly one would believe, to extreme but transient general gaseous distention of the abdomen such as is seen often in the psychasthenic and hysterical or psychasthenic "air swallowers."

The "aerophagi."

We all swallow air, but the "*aerophagi*" swallow it excessively and against increasing pressure and may attain thereby alarming abdominal proportions or merely such distention as keeps them uncomfortable, and still swallowing unless corked up. As stated elsewhere this may actually be accomplished by having them hold between the teeth a *large* cork, which makes effective deglutition difficult or impossible.

Colon dilatation.

Idiopathic dilatation of the colon has been described as "chronic phantom tumor," a manifest misnomer, for this condition could never be described as chronic without at least the addition of the words "remittent" or "intermittent."

Mere protrusion.

A not uncommon phenomenon, sometimes described with dubious propriety, as phantom tumor, consists in the protrusion of certain gas-filled intestinal coils without evidence of marked intestinal spasm, but to such a degree and in such locations as may produce a striking and bizarre appearance.

In some such cases voluntary or involuntary diaphragmatic action seems to initiate and maintain the tumor.

The type first described is that to which the term is applied most fittingly.

Visceroptosis or diastasis.

Nearly all of the forms described occur in nervous or hysterical women and usually in such as are congenitally asthenic, badly nourished and carry a thin, relaxed abdominal wall or are afflicted with actual diastases of the belly muscles. Most of the swellings of intestinal origin are easily recognized as ephemeral and in any event all that are due to spasm yield to anesthesia.

Hypogastric Tumor.—*Retention of urine* produces hypogastric tumor which may reach large dimensions and have the elasticity and, perhaps, the fluctuation characteristic of accessible encysted liquid accumulations. The introduction of a catheter resolves all doubt, which is seldom great.

Seldom deceptive.

In stuporous or unconscious patients it must be anticipated.

Enlargement of the uterus produces a solid tumor in the same region and its nature is readily established by bimanual vaginal examination.

Ovarian Cyst.—If large enough this may be detected in the same area as an elastic round tumor showing fluctuation, but it is usually obviously one-sided as to origin and attachment until it attains large dimensions and simulates ascites. It lacks, however, the bulging in the flanks and the changes in percussion resonance and abdominal outline, attending changes of position, and characteristic of free ascitic fluid in the abdominal cavity.

Violent pulsation.

Pseudo-abdominal Aneurysm.—True aneurysm of the abdominal aorta is extremely rare. A more or less violent or prominent pulsation of an obtrusively palpable aorta is very common in exophthalmic goitre cases or in congenital asthenics who are poorly nourished.

Lacks other aneurysmal signs.

In these the aorta shows no such bulging or change in the uniformity of its outline as could be regarded as a true sac or genuine dilatation.

In such patients the subjective sense of pulsation is often troublesome and becomes a source of complaint.

Misinterpretation of a Palpable Pylorus.—If in the upper abdominal palpation the contracted pylorus is encountered as a firm, short tumor-like body, it may lead to mistaken conclusions.

Its relaxation will promptly follow unless it is the seat of spasm, the rhythmic alteration in consistence persists and error is rare if both its normal position and enteroptotic modifications of site are remembered.

Value of inflation.

If spasm actually exists the relation of the mass to the stomach may readily be determined by inflation and evidence of ulcer or erosion should be sought.

DIFFERENTIAL POINTS RELATING TO TUMORS.—Abdominal tumors, apart from mere enlargements, are usually malignant or tuberculous.

Nodular hard tumors are almost invariably carcinomata.

Sarcomata are uncommon and usually present a firm smooth surface offering perhaps, areas of softer consistency.

Benign growths, when such occur, are usually hepatic in seat, though this must not be taken as meaning that the liver is not a frequent source of secondary carcinomatous new growths.

In establishing the anatomic site of a tumor, aside from cases of evident organic connection, its range and type of respiratory movement, fixation, or passive mobility are of chief importance.

Direct movement.

But two abdominal viscera, *the liver and spleen* show respiratory movement *so immediate and direct* as to be practically coincident with the inspiratory and expiratory phases of respiration. Such movement in tumors distinctly suggests an hepatic or splenic origin.

Hesitant descent.

The stomach shows a definite respiratory change of position, less marked if visceroptosis is present, and to a somewhat less degree this is true of the *colon*.

"Lagging."

The kidney shows a *lagging* displacement which, as to extent, varies greatly with the conditions surrounding its anchorage in the individual, though even a normal kidney may slightly increase its range if it becomes the seat of a growth of considerable size.

Kidney tumors, however, whether new growths or of inflammatory origin, show a strong tendency to become fixed.

Fixation.

Pancreatic tumors are fixed at all stages.

True aneurysm, enlarged retroperitoneal glands, bone tumors and the cold abscesses resulting from caries are one and all immovable.

Bilateral kidney tumors associated with urinary findings resembling those of interstitial nephritis are almost certainly cystic.

Bilateral or unilateral chronic renal tumors associated with pus in the urine are often tuberculous and the tubercle bacillus may be demonstrated readily if this condition is present.

The diagnosis should be possible usually and by the same means before any decided tumor is present.

Passive mobility of varying degree is found in tumors of the kidney, transverse colon, small intestine, hepatic, splenic and sigmoid flexures, omentum, stomach, and in growths involving a *cecum mobile*, always provided that such tumors have not been rendered fixed and immovable by adhesions.

A gall-bladder tumor not only moves with the liver in respiration, but characteristically limits its passive mobility to lateral pendulum motion which is lost if adhesions are extensive.

Tumors of the pylorus may show an extraordinary displacement and passive mobility especially in visceroptotic cases. Pyloric tumor.

Expiratory Fixation.—These growths, together with other accessible gastric tumors, may be held fast against their tendency to ascend in expiration. This, in contradistinction to the positive determined expiratory ascent of the liver and spleen and their tumors save in profound visceroptosis.

The Differential Value of Inflation of the Stomach.—It is obvious that the condition of the stomach, with relation to distention and collapse, must influence greatly the accessibility and position of growths attached to it and also affect in some degree the position of tumors involving neighboring viscera if these be movable. This maneuver is especially valuable in the differentiation of pyloric tumors from those of the liver. General rules.

Upon inflation, a pyloric tumor, in the absence of adhesions, moves to the right and downward; those of the lesser curvature, upward usually, sometimes backward and out of reach.

Under the same procedure tumors of the liver directly ascend; those of the transverse colon directly descend; those of the ever-fixed pancreas become obscured; and those of the spleen move somewhat to the left.

Value of Colon Inflation.—This is chiefly of value in relation to its effect when colonic tumors are in question or in cases demanding differentiation between massive renal and splenic tumors.

Usually, as stated, the inflated colon casts a band of anterior resonance over a renal growth whereas the spleen remains unaffected. Kidney vs. Spleen.

Chronic Peritoneal Tuberculosis.—This must be borne in mind always in the presence of tumor *crossing the abdomen* at or somewhat above or below the level of the umbilicus.

The great omentum may form a thickened contracted rounded "bar-like" tumor fixed by adhesion to the intestines and to the abdominal wall itself.

The mesentery may also become thick and contracted and the small intestines may become massed in the middle of the abdomen or to the right side. Mesenteric masses.

Such a mass is sometimes mistaken for the lower border of an enlarged liver but a zone of resonance can usually be outlined between them and the hepatic respiratory movement and the true feel of the liver edge is lacking. Occasional source of error.

Pockets.

In such cases the intestines, omentum and mesentery may be adherent to each other and to the abdominal wall and various pockets may be found containing fluid exudate.

The autopsy picture is oftentimes one of practically universal adhesion.

Fixation of Liver and Spleen.—These organs obtain their movement from the diaphragm and are dependent upon that structure and freedom from immobilizing adhesions for their peculiarly *direct* immediate and powerful descent in full inspiration.

Important point.

Fixation by adhesion is not uncommon in chronic inflammatory conditions, but one may forget that a decided lack of motion may result from *excessive* abdominal distention especially when due to ascitic accumulations under great pressure.

THE STOMACH

An important region.

General Considerations.—Not only has modern research developed a remarkable increase of positive knowledge in regard to the capacity, dimensions and normal limitations of position of the human stomach, but it has greatly extended and modified our conceptions of the vagaries of its secretory activities in health and disease.

Less important than small intestine.

To just what extent its functions of digestion and absorption enter into the digestive process is still undetermined, but, in the light of modern investigations, the chief rôle must be assigned to the small intestine.

Important rôle.

Indeed gastrectomy and cases of achylia taught us long ago that the stomach is not absolutely indispensable to life; yet it plays a most important part in human existence. Nor is its rôle confined to its functions in relation to digestion, absorption and the preparation of food substances for the action of the intestinal ferments. To a considerable extent it is protective in its ability to inhibit the growth of, neutralize, or actually destroy, many kinds of pathogenic organisms and their toxins, or so weaken others as to facilitate anti-bacterial activity of the intestinal ferments.

Normal capacity.

Capacity.—The stomach of the new-born baby has a "voluntary" capacity of but 1 or 1½ ounces, yet in from six to eight years this is increased to 1 quart, and in the adult stomach varies from 1 to 1½ quarts.

Types of Stomach.—We are interested chiefly in the form and position of the stomach in persons standing erect, and a wide divergence of opinion exists as to what constitutes the normal. In individuals wholly free from symptoms of gastric disturbance two chief types are observed, viz: (1) the "steerhorn," carrying its lower border well above the level of the iliac crests, between the umbilicus and the ensiform cartilage, and (2) *the "fish-hook" stomach.*

The Steerhorn Stomach.—This is the normal type according to Holzknecht, but might more properly be designated the "ideal," as it occurs in only about one-third of all adults, almost wholly in men, and in such of these as are powerfully built and carry a large nutritional reserve or actual *panniculus*. Holzknecht maintains, nevertheless, that it represents the normal and necessary form with respect to the functional optimum and the type from which other forms are logically deducible.

Support for his view is found in the fact that well nourished, strongly built children carry such stomachs, whereas undernourished, slender youngsters show the type described below, with the many modifications with respect to latent or demonstrable atony and ptosis, more or less closely reflecting congenital defects of structure and the grade of subnutrition present in the individual case.

The "Fish-hook" Type.—Rieder maintains that the normal is found in the *"fish-hook" stomach*, present in some form in about two-thirds of adult individuals. In this type the long diameter is vertical, the caudal pole sac-like, and ascending to the right in such a manner as to make the *pars pylorica* the terminal of what appears like a syphonage system.

Conditions Influencing "Type."—There would seem to be little doubt that our primitive ancestors carried steerhorn stomachs, but their descendants have developed congenital and acquired defects of structure through the subtle ravages of disease.

It is interesting to recall the fact that 80 per cent. of civilized adults will react to tuberculin or show evidence of this disease at autopsy, and that the type of individual ("congenital asthenic") showing the exaggerated degrees of "fish-hook" stomach in the form of actual gastroptosis or atony in its more readily demonstrable and extreme forms, is identical with that which offers in its tissues the best living medium for the development of the tubercle bacillus. Tuberculosis.

In sucklings the steerhorn type is nearly always present. Between the ages of five and ten it constitutes the type present in two thirds of all children Between the ages of ten and twenty it is found in about 50 per cent. These figures closely parallel the positive results of tuberculin tests for the same age groups. For practical purposes the views of Holzknecht and Rieder both may be accepted if we modify the term "normal" of the former and call it the "ideal normal." In the opinion of the author no such term may be applied properly to any "fish-hook" stomach whose caudal pole sags below the level of the iliac crests, though unquestionably such stomachs, or even lower ones, may be carried without serious symptomatic disturbances in the presence of an adequate general circulation and a balanced nutrition.

The "steerhorn stomach," the powerful build, strong abdominal muscles, ideal normal heart, well developed musculature, wide thoracic aperture, and superabundant nutritional reserve, go together.

The "normal" "fish-hook" type is the commonest one in adult life—may carry its possessor through life without a serious gastric symptom or show extreme degrees of sagging with or without peristolic atony under conditions of nutritional deficit.

This does not mean that individuals with "fish-hook" stomachs must die of tuberculosis or even show or ever have shown clinically active symptoms of the disease.

It does suggest the basic importance of congenital defects of structure and the after effects of acquired infections with associated toxemia and impaired nutrition.

The close association of gastroptosis and cardioptosis observed by the author is dealt with elsewhere. (See Fig. 386, "The Drop-Heart," "Gastric Atony," "Congenital Asthenia," etc.)

Position and Dimensions.—The older views relating to the position of the stomach have been corrected and greatly modified by the result of X-ray work. *When the patient is recumbent and* in the dorsal position or erect, with the stomach fully distended, more than *five-sixths of that viscus lies to the left of the median line. And the lateral distance between the pyloric and cardiac orifice of the uninflated normal organ is but* 1½ *inches.*

Cardinal differential points.

Fundus.

The fundus, when distended, lies in the left cup of the diaphragm; hence the upper limit of percussion resonance normally extends to the fifth left interspace in the parasternal and mid-clavicular lines, while the lower may run from several inches below, to 1 or 2 inches above the level of the iliac crest.

Cardia and pylorus.

The pylorus of the empty normal stomach lies in the median line. When the viscus is distended, the pyloric opening should be midway between the right sternal and parasternal lines 1 *or* 2 *inches below the level of the xiphoid. The cardiac orifice lies, therefore, about* 1½ *inches to the left of the median line opposite the ninth dorsal vertebra posteriorly and the seventh costal cartilage anteriorly. The lesser curvature lies normally deeply under the liver border.*

Lower limit.

Absolute lines cannot be drawn for the limitations of an organ so distensible, so freely movable and so often ptotic.

In general, it may be said that a stomach whose lower border reaches the navel is abnormal, either in size or position.

PHYSICAL EXAMINATION.—Inspection.—This yields valuable results more often than is generally supposed, and, especially in cases with thin, relaxed, abdominal walls, the outline of a distended stomach may be clearly defined.

Growths.

Growths, especially of the pylorus, are often seen to move with respiration, and normal (left to right), or very rarely, reversed (right to left), peristalsis may be present, the latter often affording valuable corroborative evidence of pyloric obstruction. The normal gastric waves are large and deliberate and take from one-fourth to one minute to pass across the field.

Peristalsis.

In cases of gastric dilatation with hypertrophy of the wall due to pyloric stenosis, the condition of intermitting stomach rigidity ("*magensteifung*") may be manifest to the eye as well as to the hand.

As the pylorus intermittently yields, one may hear and feel in many such cases the squirting, spurting or gurgling of expressed fluid ("spurting murmur").

In the congenital pyloric obstructions of children and in carcinoma of the adult stomach, the tumor is often readily seen.

Importance of General Inspection.—The facies of the patient and his general nutrition are matters of cardinal importance in many instances.

A good color and a well-nourished body often go far to aid the physician in excluding organic disease of the stomach. The asthenic habitus points to the existence of visceroptosis and the many functional gastric disorders which are associated with the constitutional inadequacy of such patients and

oftentimes with an outward appearance of suffering and a degree of emaciation which may be most misleading.

On the other hand, it must be remembered that congenital asthenics may carry gastric ulcer or cancer.

Light.—A proper light is indispensable to thorough inspection and sometimes an artificial light is better than daylight for the detection of peristaltic movement and the shadows produced by the respiratory displacements of organs or growths.

Artificial light sometimes preferable.

Teeth and Naso-pharynx.—The condition of the gums, teeth, tonsils and naso-pharynx should invariably be determined and the odor of the breath will often prove suggestive. Loss of the means of mastication may lead ultimately to partial or complete loss of function of the glands which furnish certain important digestive hormones.

Tonsillar and buccal infections.

Chronic infections no less than imperfect mastication may play a large part both in relation to gastrointestinal disturbances and to general health. Peridental abscesses, a persisting sinus infection or chronically infected tonsils may be of fundamental etiologic importance.

The various pictures presented by the tongue have been described elsewhere.

Fluoroscopy and Transillumination.—By giving the patient massive doses of bismuth subcarbonate (20 to 30 grams) suspended in mucilage of acacia porridge, kefir or some similar substance, fluoroscopic or skiagraphic evidence may be obtained.* Occasionally Einhorn's method of transillumination (gastrodiaphany) may slightly assist inspection, if better means are not available.

Bismuth.

In testing the position of the stomach whether by inflation or other methods the patient should be standing.

One of the valuable qualities of the bismuth meal is its weight. We get the position of the stomach as in actual digestion of a full meal.

Gastrodiaphany.—*The gastrodiaphane* is merely a moderately rigid bougie or soft tube carrying an electric light. The patient, who is placed in a dark room, drinks two glasses or more of water, after which the instrument is introduced and the electric circuit completed. In a normal stomach the illuminated surface zone thereby produced indicates the position of the left lower segment of the fundus and is triangular in form, the apex being downward and slightly above the umbilicus. It naturally follows that in gastroptosis or dilated stomach (gastrectasia), the illuminated area is lower and larger.

Of slight value.

The information gained through simple inflation of the stomach or fluoroscopy is usually so much greater that the method is seldom required and fluoroscopy and skiagraphy, when available, replace all such inefficient and insufficient methods.

Better methods.

Türck's Gyromele.—This ingenious old device lends itself to inspection, palpation and auscultation. It consists of a flexible piece of steel, such as a piano wire, inclosed within a rubber tube and bearing a soft sponge. Being introduced into the stomach, it is attached to a little hand instrument which

Ingenious.

* See "Roentgenography" and "Roentgenoscopy."

causes rapid revolutions and will distinctly indicate the position of the sponge as it passes along the lower gastric border. Its inventor uses it not only for diagnostic, but for therapeutic purposes, but at the present time its diagnostic value is not so great, nor is the procedure so safe, in the opinion of many, as to justify its preferential use.

Not wholly safe.

Inflation of the Stomach.—*Methods.*—(1). The oldest and simplest involves the administration of 1 dram of sodium bicarbonate in a wineglass of water, followed promptly by an equal amount of tartaric acid similarly dissolved.

Old method.

Theoretically, it is open to the objection that serious danger from over-distention may result, especially in ulcer; practically, this danger is slight, yet the method is decidedly inferior to the following one.

2. After introducing the stomach tube, air is pumped into the stomach with a Davidson syringe, or, if the patient be tolerant and the physician courageous, the inflation may be accomplished by blowing into the tube. As the degree of inflation is wholly under control under this method, the procedure has almost wholly superseded the older one. The distention should not be great and the *patient's facial expression* quite accurately indicates the safe maximum.

Best method.

Inflation brings into prominence tumors of the anterior wall and obscures those of the posterior wall. In general, gastric growths are cylindrical at the pylorus, broad and flat at the fundus.

Interpretation of Outline.—The normal boundaries have already been given; if abnormal, one should carefully note whether, in the outline established, there is merely a normal or approximately normal area in an abnormally position (gastroptosis); both abnormal position and increased area (gastroptosis with ectasia), or, finally, general enlargement without marked displacement (simple dilatation).

Ectasia and ptosis.

DIRECT INSPECTION

THE ESOPHAGOSCOPE AND GASTROSCOPE

Preliminary Comment.—The diseases of the esophagus are surgical for the greater part and neurologic for the lesser, and can be given only slight attention in this volume.

A misapprehension.

It would appear that some misapprehension exists, however, both as to the best means of direct diagnosis and the relative ease and safety of its application.

The sound.

In diagnosis the esophageal sounds are serviceable chiefly as a means of determining the presence or absence of an obstruction and, approximately, the caliber of a constricted esophagus.

As generally employed, they are doubtless far more dangerous than is the esophagoscope, in the hands of anyone of average skill.

Accessibility of esophagus.

As Lerche says, "through the aid rendered by a proper type of esophagoscope, *the gullet, instead of being an organ difficult of approach becomes the most accessible, barring none.*" He further states that no matter how many cases may have been seen, if the esophagoscope has not been employed

so many mistaken diagnoses will result that any reports made will lack all scientific value and "merely emphasize the frequency of esophageal disorders."*

The Esophagoscope.—There are many esophagoscopes in successful use and the best modern instruments have a rigid tube and a source of illumination distal to the operator and as near as possible to the surface under investigation.

Chief requisites.

This latter desideratum is achieved by the use of the electric bulb, but its position as the distal extremity of the tube ordinarily involves the inconvenience of soiling, with resulting dimming of the light by accumulated mucus and blood, if the latter be present, and the remote danger of lamp breakage.

Lerche's esophagoscope.

The instrument here shown carries a suction tube which can at will be extended by the operator, not only to cover, but to extend far beyond the lamp.

Keeping the field clear.

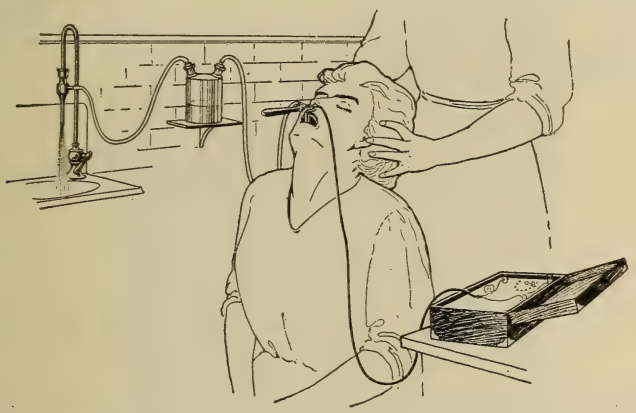


FIG. 428.—Patient in correct position with the esophagoscope introduced and the suction-pump at work.

For office or hospital work the attachment of the simple Chapman suction pump keeps the field of operation itself entirely dry.

The tip of the instrument is provided with a blunt protective lip and enables the operator to introduce it without an obturator and inspect the entire tract traversed by the instrument.

Safety tip.

In its introduction the suction tube is drawn back and the instrument entered with the outer surface of its lip facing the epiglottis and the anterior wall of the esophagus, thus bringing its greatest diameter into relation with the most distensible diameter of the canal it traverses.

Technic of introduction.

The detachable handle, if used at all, should be on the side of the instrument which carries the lip if the patient is to be examined in the sitting posture and on the opposite side if the posture is that of recumbency.

Technic of Introduction.—If, in a case to be examined, there is a large quantity of fluid and remnants of food retained in the esophagus, this material must first be drawn off by the stomach tube and the esophagus washed out

*"Diseases of the Esophagus," Wm. Lerche, N. Y. Med. Journal, June 19, 1915.

before the esophagoscope is introduced. The suction tube is for the removal of fluids accumulating during the operation.

Posture of patient.

Esophagoscopic work in adults is done usually under local anesthesia with 10 per cent. solution of cocain and with the patient in the sitting position. If a general anesthetic is used the patient is examined while lying on the back or on the right side. In children general anesthesia is always required. Fig. 360 shows the patient in the correct position on a low chair with straight back and with the head supported by an assistant. The esophagoscope is shown in position and the suction tube *A* is connected by a long rubber tube with the Chapman suction pump attached to the water faucet and having a Wolff's bottle interposed for the reception of the mucus and blood.

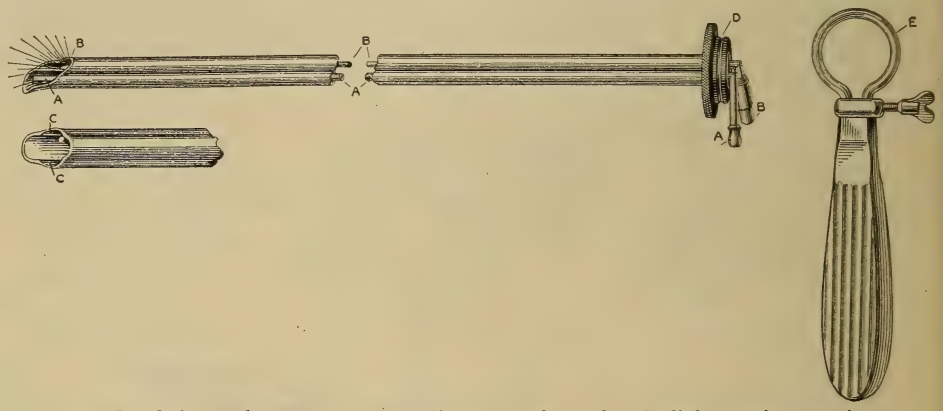


Fig. 429.—Lerche's esophagoscope. *A*, Indicates suction tube; *B*, light carrier, running in the grooves *C*; *E*, detachable handle to fit in groove *D*.

Ease of introduction.

The introduction of this instrument is easy. The operator stands in front of the patient, and, holding the illuminated esophagoscopic tube near the proximal end with the right hand, places the lip of the instrument on the back part of the patient's tongue pointing somewhat toward the patient's right. While the operator now looks through the instrument it is passed over the epiglottis and down to the inlet of the esophagus. On gentle pressure the cervical part is entered and the open thoracic part of the esophagus soon comes into view.

No lubricant need be used on the instrument and one does not guide the instrument by inserting a finger into the patient's mouth. A dry cell-battery is required for the lamps. The street electric current should not be used when the instrument is connected with the water faucet.

Tubes required.

Three tubes are required. One 10 mm. diameter and 50 cm. long, and one 10 mm. diameter and 25 cm. long, both for adults; one 8 mm. diameter and 35 cm. long for children.

THE GASTROSCOPE

This, the newest of the specific instruments devised for the exploration of the stomach is capable of yielding most interesting results, but only in

the hands of a thoroughly experienced and practised operator, and, in most instances, under profound anesthesia. This last procedure is doubtless to some degree avoidable under the skilful technic of a past master of the art.

The difficulties in gastroscopy also are manifestly greater than those of esophagoscopy by reason of the obstacles to be overcome in bringing the part to be examined into view. The path from the teeth to the stomach must be made direct and hence the normal curves must be straightened. Obviously the head must be thrown far back to raise the level of the upper jaw and overcome the curve normally present and considerable delicacy of technic is required to secure the entrance of the instrument into the cardia and beyond that point as is actually accomplished by the expert.

It occasionally happens that in individuals with a very prominent upper jaw and short thick neck the introduction of the tube may be well nigh impossible. The passage of the tube from the esophagus into the stomach may

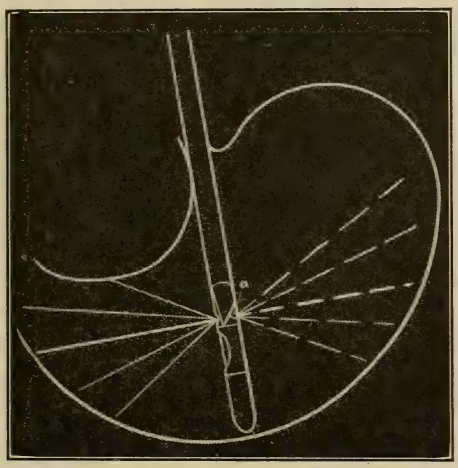


FIG. 430.—Usual gastroscopic field. It is manifest that the accessible area will be greatly increased in ptotic stomachs.

also be blocked by reason of great deformity of the spine, abnormalities of the gastric musculature itself, large aneurysms of the thoracic aorta, or existing heart disease greatly distending the left auricle and producing compression upon, and stenosis of, the esophagus.

Some of the modern forms of the gastroscope have largely overcome any danger inherent in its use by dispensing with the uniformly rigid tube such as easily led to perforation of the esophagus in clumsy or inexperienced hands when the attempt was made to traverse that structure at its lower portion opposite its point of passage through the "*hiatus esophagus*" of the diaphragm. The use of a flexible tip which in its passage conforms to the curves of the esophagus offers an advantage, though adding somewhat to the complication of the instrument.

Much difficulty has also been experienced in devising an instrument which would secure a clear field of vision and freedom of the area under

examination from obscuring secretions. This is accomplished by the newer instruments of Jackson and of Elsner.

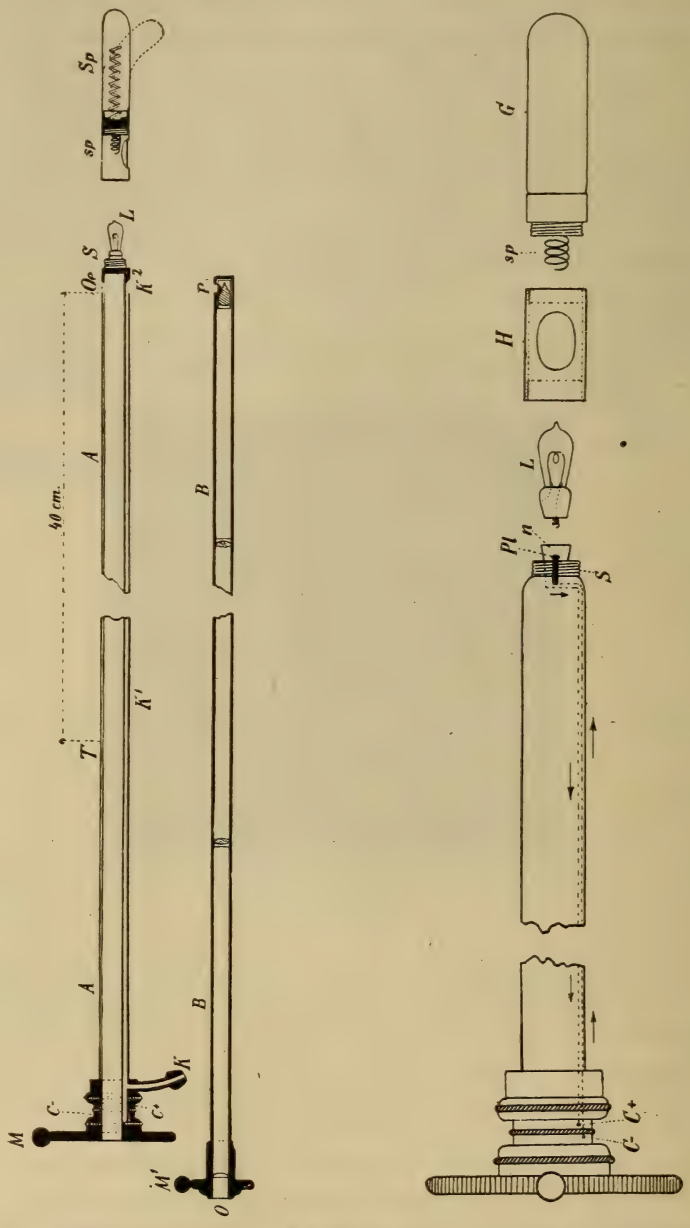


FIG. 431.—Elsner's gastroscope. *AA*, Outer tube (11 × 76 cm.); *Oe*, objective lens; KK^1K^2, air passage; *T*, mark to indicate distance tube should be passed (40 cm.) to just enter stomach.

The greatest difficulty of all doubtless lies in the limitation of the working field and the difficulties encountered in bringing into view all portions of the stomach and this is emphasized by the many variations in form and position

which this organ may assume, no less than by the almost constant peristaltic movements which are likely to be present.

Even under full anesthesia, such as is required almost invariably for the examination, it is seldom thought wise to continue the procedure for more than a few minutes and during that period only the expert operator can secure much information. He must work rapidly, intelligently and with the full knowledge of both the possibilities and limitations of the instrument.

As stated, the two chief forms of gastroscope are: First, that consisting of the rigid tube, such as that devised and used with excellent results by

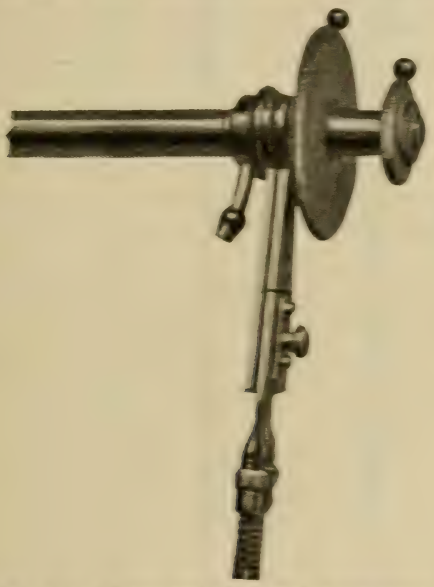


FIG. 432.—Upper end of Elsner's gastroscope.

Dr. Chevalier Jackson, and second, that represented by the instrument of H. Elsner shown here, which combines the rigid tube body with a flexible tip.

Dr. Jackson's instrument has its light at the distal end of the tube on a carrier which permits rapid removal for cleansing, should this be necessary during an examination.

By the ingenious device of placing the light bulb itself at one side of the visual orifice, the very considerable advantage of oblique illumination is secured.

A drainage device is provided in order that the field of examination may be kept clear of blood or mucus and this drainage tube itself may be replaced and removed by another as desired.

An obturator is furnished to aid the passage of the tube through the constricted areas.

Elsner's gastroscope embodies the principle of the Nitze irrigation cystoscope and consists of an outer tube containing the optical apparatus which is freely movable upon its axis, the usual optical apparatus and an outer tube. The two tubes are so placed as to leave between them a half-moon-shaped

passage. This last serves as a canal for the introduction of air and likewise carries the light conducting mechanism. The light is carried upon the lower end of the main tube, is fully protected and the instrument carries a flexible tip which Elsner believes greatly facilitates its primary introduction. Of the same caliber as the gastroscope and carrying an elastic spiral within, this tip is intended to fulfill the double purpose of meeting gently and passing

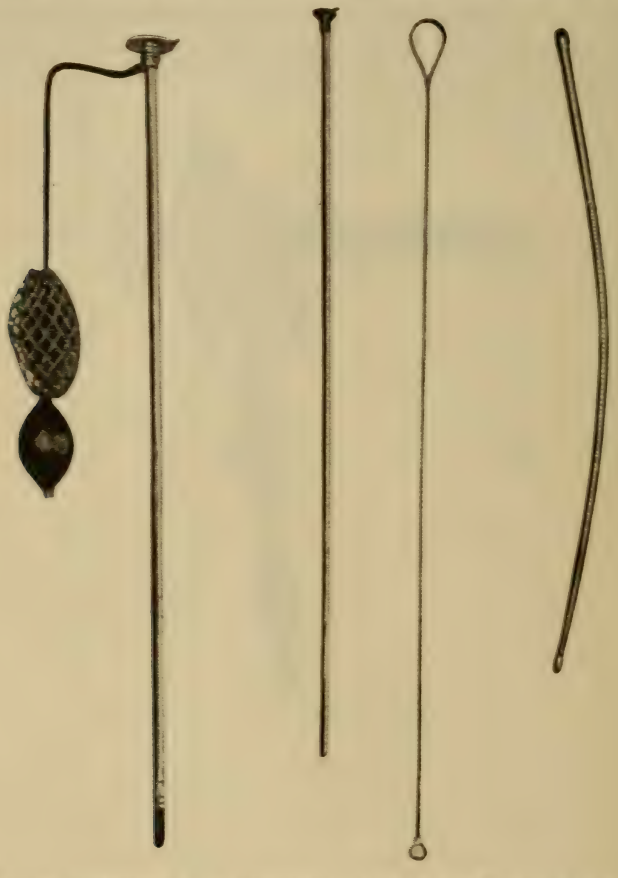


FIG. 433.—Elsner's gastroscope. A sponge carrier and a metallic sound are shown at the right. Outer tube and that containing the optical apparatus are shown on the left.

easily the diaphragmatic junction point and also of preventing any possibility of perforation of the stomach attending the actual introduction of the tube. With such an instrument in many cases it is possible for the *expert* to observe directly the more important regions of the stomach as is indicated by the figure, viz., the anterior wall, the region of the pylorus, the entire fundus and the greater part of the lesser curvature and posterior wall. This procedure, of course, demands the proper handling of the light both through the manipulation of the movable shaft governing it and the proper raising and lowering of the tube itself. The pyloric antrum is clearly seen but unfortunately the pylorus itself is usually unattainable. Only when the pylorus is greatly lowered or unusually high does it come into the gastroscopic field.

The introduction of the gastroscope should be preceded by a most thorough and careful physical examination of the patient and especial attention should be paid to the possible presence of aneurysm or mitral disease or advanced arterial sclerosis, especially if this be associated with high arterial tension. The question of possible deformities will usually be determined and the constitutional type of the patient may or may not be such as is favorable to the introduction of the instrument.

Victims of congenital asthenia furnish the very best material on account of the general slackness of their tissues and ligaments and in them one is frequently able to see the pylorus itself.

The author deals thus briefly with this ingenious device for the reason that he considers it wholly unadapted to general use and, assuming equality in skill and perfection of technic, more dangerous than exploratory incision.

Palpation of the Gastric Area.—This is chiefly used to determine three points: (1) *The presence of tumor.* (2) *Localized tenderness.* (3) *Succussion.* Seldom palpable.

The palpable tumors of the stomach oftenest involve the pylorus and lie to the right of the median line between the margin of the ribs above and the navel below.

Areas of thickening over the distended stomach, often plaque-like in feel, may be encountered *in rare instances* and indicate an old ulcer base. Old ulcers. Adhesions frequently occur, anchoring the stomach to adjacent organs and greatly complicating and obscuring diagnosis. Adhesion. Accessible tumors ordinarily move freely and indeed may be so far from their usual site as to suggest growths of distant organs. Their movement and position in connection with inflation then becomes of special importance. Tumors. In very thin flabby abdomens one must be on his guard against mistaking other structures, perhaps normal, for gastric growths. Useful method.

Intermittent stomach rigidity as described under "*palpation*" over the fundus may be encountered in cases of pyloric stenosis.

Through relaxed thin abdominal walls such as are encountered often in cases of visceroptosis or "hanging belly" the pylorus may sometimes be directly palpated as a cylindrical body 4 to 5 cm. long and felt to harden and relax with an associated "squirting" sensation (already mentioned) and if a soft tube be used the point perhaps may be distinctly felt as it is coaxed along the lower border to the pylorus. Pylorus. If pylorospasm occurs, the "feel" is that of a hard egg-shaped mass. Pylorospasm.

If bimanual palpation be employed over a thin, relaxed abdomen with the tips of the fingers deeply pressed against the posterior wall of the abdomen at an angle of 45° with the external surface, one may sometimes feel the duplicated lower edge of the collapsed stomach as it passes upward at the end of deep expiration. Palpating border. It gives the impression of a cord rolled under the fingers and may be associated with a gurgle if any fluid is present in the viscus. A slight downward glide of the finger-tips at or immediately preceding the height of expiration is necessary to the success of the maneuver.

Below the stomach one may feel the transverse colon as a similar cord, *unaffected* by respiration. Transverse colon.

The procedure is interesting and constitutes an excellent exercise for the development of the tactile sense, but otherwise its value is almost nil.

Of slight value.

The levels of the visceral borders will vary with the grade of any visceroptosis present and palpation is seldom successful with a normally placed stomach.

Succussion.—Any splashing sound *suggests* dilatation or gastroptosis with marked atony if it occurs more than six or seven hours after a meal.

Overestimated sign.

The value of this sign has been overestimated and is practically limited to its occurrence before breakfast or on a fasting stomach. *It may be present in wholly normal individuals and, in general, merely indicates slight motor insufficiency, or, if it be obtained within narrower limits but by very gentle tapping, it strongly suggests decided gastric atony.**

In asthenic dyspepsia the sign is undoubtedly in a large degree dependent upon the condition of the abdominal wall itself and is peculiarly common in connection with the relaxed abdominal musculature and visceroptosis of asthenic individuals.

Finally, rarely, actual dilatation of the stomach may occur without superficial splashing.

Succussion is obtained by placing one hand over the left ribs posteriorly while making sharp dipping movements anteriorly.

Precautionary Pelvic and Rectal Examinations.—Many disastrous errors in diagnosis would be avoided if physicians would adhere more scrupulously to the rule which demands that in married women such examination be made if any substantial reason exists for suspicion. Pelvic growths or abscesses especially are potent causes of obscure gastric symptoms.

The author does not wish to be understood as advocating routine pelvic examinations. Sound common sense is necessary. In young girls especially these should be made only when clear indications are present.

Percussion.—Ordinary percussion is of little use and need not be discussed.

Slight value.

It was formerly the custom to introduce a pint or more of water and percuss the dull area produced, but both the boundary and the muscle tonus are better established by inflation.

To attempt to outline the stomach by percussion or even to differentiate the stomach tympany from that of the intestines, in the absence of fortuitous or artificial inflation, is absolutely futile.†

Auscultatory percussion is distinctly useful though nevertheless fallible; useless if not preceded by inflation. Normal gastric tympany, as heard over a moderately inflated stomach, is represented by the segment of a circle. It is limited above by left lobe hepatic dulness, but includes Traube's space and has a curved border below, which normally should *not* reach the navel.‡

* Sahli quite properly distinguishes between "deep splashing" and "superficial splashing," for these differences are quite readily recognized by the examiner.

† The empty stomach is, in fact, often practically inaccessible to percussion because of the overlap of the liver and left lower thorax.

Furthermore, the stomach tympany varies greatly with the amount of food or gas content and the same statement applies in the case of the hollow viscera surrounding it.

‡ The word "not" is given emphasis because of a present tendency to be misled by

Contraction of the left lobe of the liver, shrinkage of the left lung, gastroptosis, and marked distention of the stomach tend to enlarge it. By careful auscultatory percussion, the upper right and left limits may be somewhat extended though the change of note due to the lung and left hepatic lobe will be distinctly marked.

Technic.—*The bell of the stethoscope is placed just below and to the left of the ensiform cartilage and light percussion is carried from it in radiating lines, in accordance with the boundaries outlined above, either from the stethoscope outward until the note changes, or from the surrounding negative areas toward that of gastric resonance.*

It is well to shift the position of the stethoscope and repeat the percussion in order to check results. So also in the case of a growth, if the stethoscope be placed over it and a similar method employed, one may often determine its connection with the stomach. An elaborate description of the tones is unnecessary as the veriest tyro can distinguish the abrupt change that indicates that the border of the *inflated* stomach has been passed.†

Auscultation.—Aside from the procedure mentioned below, this method finds its only usefulness in the detection of esophageal "swallowing sounds" "the succussion sound" already referred to, the "sizzling" or "fizzing" of active fermentation, and the test for hour-glass (bilocular) stomach.

Swallowing sounds.

Auscultation of Inflation Murmurs.—While the stomach tube is in the stomach the outline of that organ may be determined sometimes with considerable accuracy by forcing jets of air into it with the bulb and noting the limits of audibility of the brûits produced during the process of inflation, but the value of this procedure is very limited.

Revidtzef's Sign.—It would seem that this might well replace the older method of noting delay in the second element of the double pharyngo-esophageal sound attending deglutition.

A valuable test for esophageal stenosis.

Revidtzef points out the fact that in stenosis of the esophagus the retained food requires more than one act of swallowing for its complete removal and that in such instances the normal double sound will be duplicated, or repeated several times, if the patient attempts to complete the act of swallowing without taking more into the esophagus.

If the interval between the primary and secondary sound exceeds the normal limit the older test gives added information but cases of stenosis may present a normal interval (five to seven seconds) and the newer test would seem the more reliable.

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the frequency of gastroptosis into the belief that a *decidedly* low stomach is a wholly negligible condition.

Its chief importance lies in its definite assignment of its possessor to the congenitally asthenic group, a matter of no little importance in many instances, whether as a contraindication of all major surgery save that of genuine need or actual emergency, or, as clearly indicating the proper line of medical treatment to be undertaken. (See "Congenital Asthenia.")

† The note is uniform in quality and pitch so long as the stomach is percussed, though intensity depends upon the distance of the stroke from the stethoscope.

ROENTGENOGRAPHY AND ROENTGENOSCOPY IN THE DIAGNOSIS OF GASTRIC AND DUODENAL DISEASE

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Modern technic.

Historical Note.—Professor Roentgen announced his discovery of the X-rays in 1895 and stated that not only metals but the salts of metals, either solid or in suspension, would obstruct the passage of these rays. This suggested the procedure of filling certain hollow organs invisible to the ray, with solutions or suspensions of these metallic salts, thus to render these organs roentgenizable. Dr. W. B. Cannon of Boston was probably the first to employ a bismuth salt mixed with food in a roentgenologic study of the gastrointestinal tract and the method has grown with time and experience until today it occupies the most important place among physical methods of gastrointestinal diagnosis.

The bismuth meal.

Great importance.

TECHNIC.—The patient takes a morning meal prepared as follows:

Meal No. 1.—Fifty grams of bismuth subcarbonate is well mixed with a small quantity of raspberry syrup and water. This mixture is added to 150 grams of breakfast gruel and thoroughly mixed therewith. Six hours later, the patient steps behind the fluoroscopic screen in a properly darkened room. The examiner notes whether the stomach is empty or contains a residue, and what progress the meal has made in its journey through the intestine.

Meal No. 2.—A suspension of 50 grams of bismuth (subcarbonate or oxychlorid) in fermillac 500 c.c. is then given and observed as it passes through the esophagus into the stomach to fill that organ.

The stomach is studied to discover any accessory pocket, niche, incisura, or permanent contraction, any evidence of spasm, or abnormality of peristalsis, any "filling defects," deformities, or pyloric obstruction.

The first part of the duodenum, known as the cap, is examined from all possible angles to determine whether it is deformed or regular in outline.

Necessary Procedure.—To this end one must observe from various angles each portion of the gastric wall, through a reduced diaphragm. As the food enters the stomach it is observed to pass along the lesser curvature, filling the stomach by distending it gradually in all directions. *The normal stomach contracts upon the ingesta so that the organ constantly appears filled except for a variable quantity of gas which is always present at its upper pole.*

Concurrent palpation.

The stomach is palpated under direct observation to determine the mobility and patency of the pylorus.

The duodenum is also palpated and the degree to which this stimulation increases gastric peristalsis is noted. Following the examination the patient is observed fluoroscopically at intervals as these two bismuth meals progress through the intestinal canal. Roentgenograms may be made from time to time, as a permanent record of any pathological condition noted, or, possibly, to reveal something overlooked on the screen.

Variable.

Appearance of the Normal Stomach.—The normal stomach varies rather widely with the sex and habit of the individual. *The most common type is*

the perpendicular, hook-shaped organ, with equidistant borders, the lower pole reaching a point about 2½ inches below the pylorus. "Hook-shaped" type.

Another type is the semihorizontal, steerhorn-shaped stomach which lies high in the abdomen with the pylorus at its lowest extremity. "Steerhorn" type.

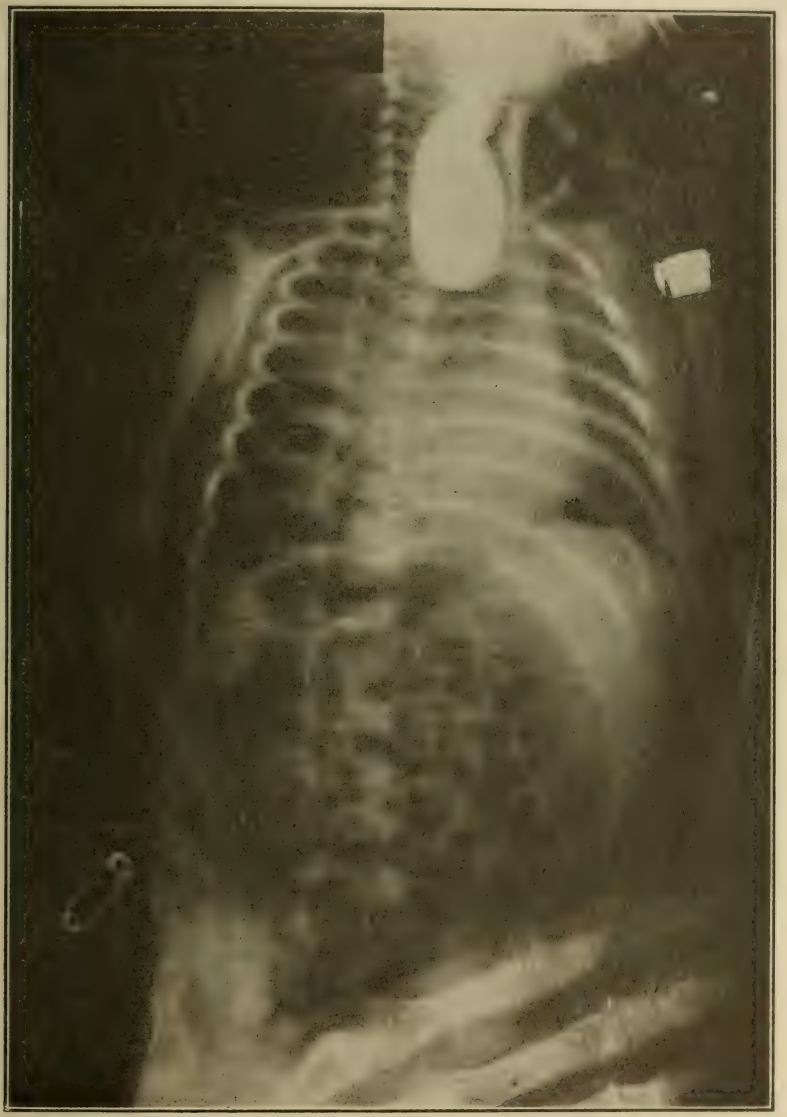


FIG. 434.—Congenital atresia of the esophagus. Note distention of stomach and intestine with air. This was explained at autopsy by a union of the distal end of the esophagus and the main bronchus. (*Dr. Frank S. Bissell.*)

This is the type usually observed in the male of "stout" habitus. It is rarely seen in the female. Various types, depending upon the muscu-

lar tone of the stomach, as well as the length of its mesentery are described as: (1) *Hypertonic.* (2) *Orthotonic.* (3) *Hypotonic.* (4) *Atonic.*

Normal retention periods.

Motility.—*An unobstructed stomach will, with rare exceptions, clear itself of bismuth meal No. 1, within six hours after ingestion. The time of complete evacuation varies from two to six hours, depending upon muscular tone, and the degree of acidity of the gastric contents. A delay of more than six hours dis-*

Significance of delay.

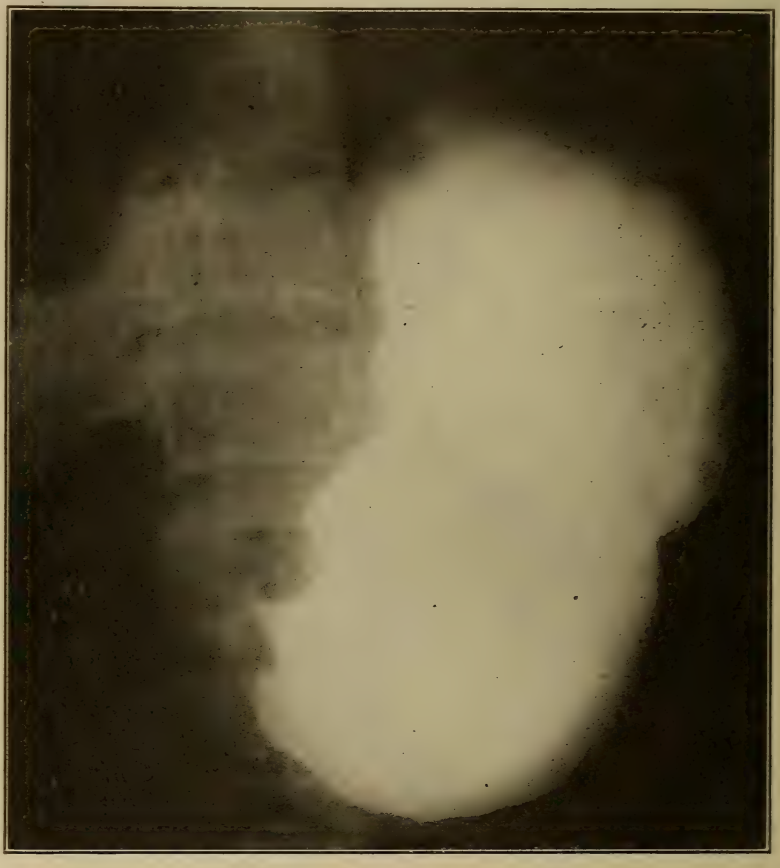


FIG. 435.—Carcinoma of the pylorus. Note area of gross filling defect, *i.e.*, absence of bismuth shadow. (*Dr. Frank S. Bissell.*)

tinctly suggests a lesion of the stomach or duodenum, less frequently of the gallbladder.

Peristalsis.—In the normal stomach we can observe, usually, but one or two superficial peristaltic waves at the same time. They begin near the cardia increasing progressively in depth until they attain the antrum, and are usually seen distinctly only upon the greater curvature.

Interruption of peristaltic waves occurs at the site of any gastric tumor or induration. Spontaneous peristalsis may be wholly absent during an examination, even when no organic disease exists and this is especially true in the presence of extreme fatigue, nervous depression and certain cases of gastro-

ptosis and atony, though in the latter they are sometimes apparently strong and vigorous.

Bulbus Duodeni.—The first or horizontal portion of the duodenum resembles stomach more than intestine in roentgenologic appearance as well as in many other respects (acid contents, motor phenomena, embryology). As a rule, it is the only portion of the duodenum which can be clearly visualized

FIG. 436.—Early carcinoma of the pylorus. Note slight pre-pyloric filling defect. (*Dr. Frank S. Bissell.*)

with the X-ray. It has been called the "cap" because of its appearance, resting upon the pylorus, or separated from the pyloric end of the stomach by the sphincter pylori. While the size and shape of the cap may vary considerably in different individuals, it is always regular in outline unless deformed by ulcer or adhesions. It is often necessary, however, to continue observations over a long period or to make a large number of roentgenograms before its normal regular contour can be demonstrated.

The "cap."

Gastroptosis.—This concomitant of congenital asthenia may exist in any degree, with or without marked atony and is detected at once by the use of the bismuth meal.

Even in extreme grades of ptosis, the pylorus is usually found fixed in its normal position at the level of the second lumbar vertebra. The important points for the roentgenologist to determine are (1) the tonus of the gastric muscle, (2) the motility of the stomach under variable conditions, (3) the effect of various positions upon gastric motility, and (4) the ability of the abdominal muscle to aid in gastric motility.

Important points.

Tonus.

1. The tonus is determined by the degree to which the content is equally distributed from fundus to pylorus. Sometimes the tone will appear excellent at first, but the stomach muscles seem to tire quickly, allowing the contents to fall to the lower pole.

Motility.

2. A stomach which is abnormally slow in emptying itself when the patient is nervously or physically tired may function in a normal manner after rest. Likewise it may readily be determined whether a stomach functions better upon a full meal or upon a partial one.

Effect of posture.

3. It has been frequently observed that a hypotonic stomach will empty very slowly while the patient retains an upright position, but when in the prone, supine, or right lateral decubitus, the apparent obstruction is lifted. It is important to learn which position suits a given case best.

Aid of abdominal muscles.

4. Another point which may be of importance is the ability of the patient, by voluntary action of his abdominal muscles to raise the intragastric food column, or even the lower pole of the stomach itself, thus aiding materially in the emptying process.

A knowledge of these various factors may prove of much value to the clinician in his management of the case. Gastroptosis is usually accompanied by a ptosis of one or both flexures of the colon, and the X-ray examination is not complete until the meal has been followed through the intestinal tract.

Dilatation.—When the stomach becomes dilated in any degree, its borders no longer appear equidistant.

Atony.

The lower pole tends to bulge like a distended bag. *If atony is associated with the dilatation the ingesta tends to fall to the bottom of the stomach, leaving a very large gas filled space above.*

CARCINOMA VENTRICULI.—Probably the most important province of the X-ray in gastric diagnosis is that of the early detection or exclusion of cancer of the stomach. A negative diagnosis, if made by an experienced examiner, is practically conclusive. A positive diagnosis too is rarely in error although there are certain pitfalls which must be avoided and certain non-malignant conditions which may closely simulate cancer. In such cases, however, a careful consideration of the clinical evidence will usually save one from a false diagnosis.

Roentgenological Signs.—Cancer developing within the gastric wall soon encroaches upon the lumen producing thereby a defect in the normal outline. This distinction between cancer and ulcer should be constantly

borne in mind: *that while in ulcer the barium column encroaches upon its surroundings, in cancer the column itself is encroached upon.* Before an actual filling defect is demonstrable, the gastric wall within the involved area becomes so infiltrated that normal peristalsis is interrupted at that point.

FIG. 437.—Carcinoma of stomach. Note filling defect on greater curvature. (*Dr. Frank S. Bissell.*)

When the peristaltic waves are not present within a certain limited part of the greater curvature, therefore, carcinoma should be strongly suspected.

Before a diagnosis based upon a filling defect is made, one must be certain that it is not

1. Artificial—produced by pressure of stomach against spine.

2. Syphilitic—produced by gumma or specific infiltration. Here one notes a relative well-being and freedom from symptoms on the part of the patient, and a positive Wassermann aids somewhat in the differentiation.

3. Extrinsic—produced by a tumor, organ, or mass without the stomach, or by skeletal deformities.

4. Spastic—due to persistent contraction of gastric musculature. Not difficult to differentiate with careful observation.

FIG. 438.—Perforating gastric ulcer with hour-glass constriction. No hæmorrhages previous to the examination. Death from hæmorrhage one week later. (*Dr. Frank S. Bissell.*)

Pyloric Patency or Obstruction.—The pyloric ring may become so infiltrated that it fails to close, the meal passing through into the duodenum with great rapidity. On the other hand, the infiltrating mass may so encroach upon the pylorus that an obstruction of variable degree is caused. Hence a six-hour retention may result.

Differentiation.

The differentiation between carcinoma of the pylorus and adhesions of the gallbladder is not always easy. Usually, however, carcinoma produces a characteristic deformity of the pylorus. When an obstruction is due to carcinoma, the stomach is more likely to appear inert, showing little or

no visible peristalsis, or perhaps an occasional peristaltic wave. Obstructions due to adhesions or chronic duodenal ulcer are more prone to cause active or hyper-peristalsis.

2. Scirrhus Carcinoma.—In this form of carcinoma, filling defects and obstructive phenomena are less marked, and a shrinkage of the gastric wall occurs, thus developing the "leather bottle" form of stomach. The area of invasion may have a worm-eaten appearance with overhanging edges. The growth may be annular, small and freely movable, merely constricting the pylorus to form the apex of a cone.

The early roentgenologic diagnosis of carcinoma must depend, not upon gross filling defects which are late manifestations, but **upon minute defects,** *the finer functional and motor disturbances, perverted, irregular or interrupted peristalsis and the occurrence of unexplained residues.*

GASTRIC ULCER. (*Ulcus ventriculi*).—A large percentage of active, *i.e.*, symptom-producing gastric ulcers will manifest one or more roentgenologic symptoms. These are: (1) *Delayed evacuation.* (2) *Presence of an* **incisura** *in gastric outline.* (3) **Presence of an accessory pocket.** (4) *The niche.* (5) *Hour-glass contraction.*

1. Delayed evacuation, resulting in a residue in the stomach after a six-hour period, frequently occurs in cases of gastric ulcer, even though the ulcer is situated at a distance from the pylorus. *Hyperchlorhydria is not necessarily a concomitant symptom and this residue may be due solely to pylorospasm.*

Other causes of delayed evacuation are: carcinoma of the pylorus, duodenal ulcer, or pylorospasm incident to gall-bladder disease. *Practically all cases having a six-hour residue are found to have some surgical lesion.*

2. An incisura *on the greater curvature, which persists despite palpation and the previous administration of belladonna, is a cardinal symptom of gastric ulcer.*

It is probably due to spasm of circular muscle fibers and hence occurs at a point directly opposite an ulcer on the lesser curvature.

3. Accessory Pocket.—As the bismuth suspension enters the stomach, it may be seen to run into a pocket or miniature stomach outside of the gastric wall. This always indicates a gastric ulcer, perforating most frequently into the liver or pancreas.

4. The niche is a pathognomonic sign of *penetrating* gastric ulcer. It is observed as a small fleck-like bismuth shadow on the lesser curvature. Its presence is usually confirmed by an incisura on the greater curvature.

5. "The hour-glass constriction" of gastric ulcer must be found present upon repeated examinations to avoid the possibility of error.

When it is observed, the patient must be re-examined after the administration of belladonna. Twenty drops of the tincture will usually serve to determine the tolerance of the patient and if no reaction follows the same dose may be given every two hours until the physiologic limit is reached. If the "hour glass" persists upon re-examination it is positive evidence of a lesion.

Such a contraction may disappear under anesthesia so that only the ulcer is found at operation.

DUODENAL ULCER.—The most important roentgen signs of duodenal ulcer are: (1) **cap deformity**; (2) **diverticulum, or accessory pocket.**

1. **Deformity of the bulbus duodeni, pilleus ventriculi or cap** as it is variously styled, is constantly observed when duodenal ulcer is present. The defect in outline may be difficult to demonstrate since it sometimes occurs

FIG. 439.—(See Fig. 440.) Chronic duodinal ulcer.

on an inaccessible part of the cap. *When, however, a normal cap is clearly seen either on screen or roentgenogram, a negative diagnosis is justified.*

Failure to visualize the cap should not be accepted as evidence of ulcer. Here one must rely upon secondary gastric signs such as (1) *hyperperistalsis*

and (2) *delayed emptying time*, to make a probable diagnosis. The differentiation usually lies between duodenal ulcer and gallbladder disease.

Gallbladder disease may cause cap deformity, either by direct pressure of an enlarged gallbladder or by adhesions. Such deformities have not,

FIG. 372.

FIG. 439 AND 440.—Chronic duodenal ulcer. Note irregularity of *bulbus duodeni*, constant in a series of roentgenograms. (*Dr. Frank S. Bissell.*)

as a rule, the usual characteristics of ulcer deformities. The latter usually assume one of the following forms:

(*a*) Incisura—single or multiple.

(*b*) Excavation—basal or on posterior wall.

(*c*) Niche or accessory pocket.

2. A diverticulum, when observed, is distinctive evidence of perforating duodenal ulcer.

Minor or confirmatory signs of duodenal ulcer *are:* (1) *Hypermotility with speedy clearance of the stomach.* (2) *Lagging of bismuth in the duodenum even after the stomach is empty. This is especially significant if it is associated with a* (3) *tender pressure point.* (4) *Intestinal hypermotility, which may occur with duodenal irritation despite normal or increased acidity of gastric contents.* (5) *Gastric hypertonus.* (6) *Hyperperistalsis.* (7) *Spasmodic hour-glass con-*

FIG. 441.—(See Fig. 442.) Old duodenal ulcer.

tractions of the stomach which are occasionally produced by intense duodenal irritability. (8) *Delayed gastric evacuation resulting in a six-hour residue frequently occurs in duodenal ulcer.*

It should be noted that hypermotility or rapid clearance may occur in duodenal ulcer without obstruction, whereas delay occurs when spasm or adhesions interfere with evacuation.

The writer's experience has convinced him that it is not safe to make a

diagnosis of duodenal ulcer upon any of these minor symptoms but that the presence of either one of the major signs is conclusive.

The gastric hyperperistalsis observed in duodenal ulcer is probably a reflex from duodenal irritation. It varies in intensity from a slight exaggeration in wave depth to most energetic, *cramp-like contractions. The lesser curvature participates in these contractions so that the waves appear in symmetrical pairs.* Three or even four of these may be observed at once whereas normally only one or two relatively superficial ones are seen.

FIG. 442.

FIG. 441 AND 442.—Adhesions from old duodenal ulcer. Deformity of *bulbus duodeni.* (*Dr. Frank S. Bissell.*)

True hyperperistalsis rarely, if ever, occurs except in conjunction with delayed gastric motility, but partial pyloric obstruction due to a lesion on the gastric side is not accompanied by this symptom. Hence it is apparent that it is a manifestation in some way dependent upon duodenal irritation. Since it occurs only intermittently, it may be readily overlooked in a given

case. When a six-hour retention has been demonstrated, however, persistent observation will usually be rewarded. The concomitant occurrence of true hyperperistalsis and a six-hour retention is conclusive evidence of obstructive duodenal ulcer.

FIG. 443.—Chronic ulcer of the duodenum, after gastro-enterostomy. Note that the *bulbus duodeni* contains barium-laden ingesta, showing also constant irregularity, despite a properly functionating gastro-enterostomy. Hyperperistalsis is evident. (*Dr. Frank S. Bissell.*)

A gastric residue after six hours occurs in about 40 *per cent. of the recognized cases of duodenal ulcer. Hence it is a very important sign. Other conditions in which it occurs are actual pyloric stenosis, gastric ulcer and, less frequently, in gallbladder disease.*

Of the minor roentgenological signs of duodenal ulcer, the writer believes the most important one is intestinal hypermotility.

It is especially significant in the presence of hyperacidity of gastric contents which when due to other causes tends to *retard* intestinal motility. The degrees of intestinal motility are determined by the position of the head of the bismuth column at a given time following ingestion.

Normal Intestinal Motility.—Under normal conditions the first part of the bismuth meal should reach the cecum in four hours. To attain the first portion of the transverse colon eight to ten hours are required and in from twenty to twenty-four hours it should be in the rectum or appear in the stool.

A portion of the bismuth may remain in the colon for forty-eight hours.

Constipation of all grades may be recognized roentgenologically and oftentimes the information furnished is of great value, a statement especially applicable to the misleading spastic cases characterized by a striking state of colonic spasticity and *marked bismuth delay in the cecal region.*

The Colon.—While various obstructive phenomena may be observed as the meal passes through the colon, the bismuth or barium clyster offers the best means for the roentgen study of the colon. The suspension employed should be warmed to body temperature and observed on the screen as it finds its way from ampulla, via sigmoid, descending colon, splenic flexure, hepatic flexure and ascending colon to cecum.

Filling defects due to carcinomatous infiltration are carefully searched for and when observed are very characteristic.

Palpation during process of filling may elicit evidence of adhesions. Small pockets or diverticuli may be seen to fill and may remain filled for days after the evacuation of the colon.

The position of the flexures is carefully noted as evidence for or against a diagnosis of splanchnoptosis. The position of the transverse colon is less important since it may appear in various positions at as many examinations.

The appendix may frequently be visualized upon screen or plate twelve to twenty-four hours after ingestion of a fermillac-barium meal. When it fills and empties as the cecum empties, and is apparently non-adherent, a negative roentgen diagnosis should be made. The tip of the filled appendix may be fixed (adherent) or it may remain filled for many hours or days after the contents of the cecum have passed on. Here a positive diagnosis is indicated. A retrocecal appendix is usually pathological.

Indirect evidence of chronic appendicitis may be of considerable importance. It consists of (*a*) *ileal stasis* (delay in the terminal ileum for 18 hours or more); (*b*) *cecal stasis* (retention of a barium residue in the cecum after the rest of the column has passed on through the colon; (*c*) *pressure*, tender-point discovered while manipulating the appendix under the screen.

ILEOCECAL TUBERCULOSIS.—Lawrason Brown has recently pointed out that the most characteristic roentgen sign of this disease is a failure of the cecum to fill and remain filled in a normal manner with an opaque

enema. Whether this sign is due to spasm, to a hypermotility, or to a diffuse infiltration producing a rigidity of the part of the bowel affected, is undetermined. The speed with which this phenomenon manifests itself from time to time seems to indicate that it is due to spasm.

FIG. 444.—Gall bladder disease. Note thickened gallbladder producing deep concave impression upon bulbus duodeni. (*Dr. Frank S. Bissell.*)

Tuberculosis elsewhere in the intestine manifests itself in no typical manner but is indicated by obstructions or localized narrowing of the bowel lumen.

CHRONIC ULCERATIVE COLITIS is characterized by an absence of haustral markings and at times by a marked narrowing of the affected colon.

GALL-BLADDER.—The degree of precision with which chronic cholecystitis can be diagnosed roentgenologically seems to vary directly with the effort and persistence devoted to the problem. The modern trend seems to be toward an attempt to demonstrate thickened and hence pathological gallbladders rather than gallstones which may be contained therein. If one accepts the statistical reports of an increasing number of men of large experience, one reaches the conclusion that it is possible to demonstrate from 85 to 95 per cent. of all chronically diseased gallbladders.

The only direct sign, of course, is a diffuse shadow extending below the liver margin somewhat simulating gallbladder in outline. If within this shadow gallstones can be outlined, the diagnosis is much more conclusive.

More indirect signs are (1) pressure deformities of the cap, (2) displacements of the duodenum upward and to the right, (3) persistent filling of the second and third parts of duodenum during barium meal examination, (4) unexplained pylorospasm or gastrospasm, and (5) six-hour gastric residues unexplained by other lesions.

THE STOMACH TUBE AND ITS USES

The Tube.—One should use a soft rubber tube carrying ample fenestrations at its extremity and a large terminal perforation. It should be surmounted by a glass funnel, below which some physicians introduce a glass tube as an indicator. For diagnostic purposes, *the simpler the stomach tube the better are the results, and the very elaborate and complicated special tubes are unnecessary.*

Simple requirements.

Contraindications.—*Under certain conditions the introduction of the tube is unjustifiable and dangerous, and it is always necessary to balance the value of the result to be obtained and the possible danger experienced.**

The following conditions usually forbid the use of the tube in those not habituated to its use: (*a*) *Extreme anemia; weakness and exhaustion from whatever cause.* (*b*) *Advanced myocardial weakness.* (*c*) *Recent hematemesis or tarry stools.* (*d*) *Advanced arteriosclerosis or past cerebral hemorrhage.* (*e*) *Advanced pregnancy.* (*f*) *Aortic aneurysm.* (*g*) *Terminal pulmonary tuberculosis, especially if hemoptysis has occurred.* (*h*) *High grades of emphysema and severe bronchitis.* (*i*) *Cases showing excessive arterial tension.*

Furthermore, in elderly persons of apoplectic build and tendency, the first passage of the tube usually involves an amount of straining and congestion that is extremely dangerous. False teeth should be removed before attempting to introduce the tube.

In the case of patients who have become habituated to the use of the tube previously these restrictions need apply only to recent hemorrhagic cases and aneurysm.

Technic of Introduction.—By word, manner and method the operator should minimize the procedure in the mind of the apprehensive victim. The

Important suggestions.

* That this is not imaginary was shown not long since in a case observed by the author in which a *large soft* tube passed directly through the base of a *carcinomatous ulcer*.

patient should be reassured and told to obey orders absolutely, swallow when told to do so *and breathe deeply throughout.* He should *not* be instructed *primarily* to breathe through the *nose*, for any such *conscious* effort in that direction tends to increase his initial sensibility.

If, however, his breathing is checked as the tube enters the esophagus, he should be told *to close his lips upon the tube* and breathe through the nose. Sharp commands are often successful and effective in shutting off nervous gagging or retching or attempts to pull out the tube, which should be warmed by placing it in hot water, held like a pen and introduced promptly and firmly without haste and without regard to temporary obstruction.

The patient receives the tube with the head inclined somewhat *forward*, the mouth *comfortably open, not stretched to the limit*, and the tongue unprotruded. He may be asked to swallow as it reaches the pharynx and there need be little fear that it will enter the larynx; if it does, the fact is at once evident, the tube is removed and no harm done. No considerable force is at any time necessary nor should it be employed.

The depth to which it should normally be passed is from 16 to 22 inches from the teeth and the *normal* distance is usually marked upon the tube, but is of slight use.* In gastroptosis and ectasia it will need to be deeply introduced before results can be obtained and that fact is diagnostically suggestive.

In all individuals showing the stigmata of *congenital asthenia* it may be taken for granted *primarily* that the point of the tube must descend well below the level of the navel.

On the other hand, one should not fail to try the effects of aspiration at different levels and in different positions of the body before accepting the fact that the stomach is emptied.

The tube may fail to reach the surface of any residual content or may coil or tilt upward in such a way as to maintain its point above that level.

Keep fingers out of patient's mouth.

In the introduction of a stomach tube there is not the slightest reason for introducing the finger into the patient's mouth, *a procedure which increases "gagging" and distress and often involves well-deserved injury to the physician's finger.*

Expression of contents.

To obtain the contents, the patient is asked to lean sharply forward with the knees pressed together, take a deep breath and strain as at stool. Either the physician or the patient may assist this movement by firm pressure over the region of the navel or at a lower level if visceroptosis exists.

Aspiration.

In some cases it may be necessary to attach a syringe bulb or Politzer's bag to the tube and make suction.† From time to time a tube may be

Tube blocking.

blocked and the bulb may be used to force in jets of air, or the obstruction

* Most of the stomachs investigated are of the vertical, *low-lying* (ptotic) type.

† Such a bulb can readily be fitted with a glass tube that accurately fills the caliber of the stomach tube and the device is better than the use of a compression bulb on the tube itself. The fortunate ones who can milk a tube as if it were a cow's udder require neither excessive straining on the part of the patient nor the use of a suction bulb.

may disappear if the tube be slightly withdrawn and reintroduced; this latter procedure being often necessary in any event.

The first attempt to introduce a stomach tube is usually associated with more or less violent retching and straining and is greatly dreaded by the patient, but he may be assured that after three or four introductions the discomfort will be slight. Habituation rapid.

If the patient's head is thrown back, the tube may enter the glottis and stridor at once follows. As stated previously, no harm is done except from the fright the patient may suffer.

The use of the X-ray and "exploratory incision" has displaced the systematic examination of the gastric contents to an unjustifiable degree.

The stomach tube should still hold first place, not only because it is readily available to the use of all practitioners, rather than the fortunate few, but also because the X-ray, valuable and often indispensable as it is, cannot and does not fill its place.

Roentgenography and roentgenoscopy show certain pathologic conditions with a promptness and certitude which is most gratifying, but on the other hand the stomach tube alone shows many of these quite as definitely and many more which the X-ray does not reveal. The one supplements the other and often aids in confirming findings. Both are fallible.

The exploratory incision is at once the most valuable and most abused and overdone of diagnostic procedures. If it were made an invariable rule that, save in actual emergency, none should be undertaken until all other modern diagnostic procedures had been exhausted, it is probable that the present number of operative short cuts to diagnosis would be reduced by 80 per cent.

In relation to the stomach itself the tube properly used is one of the most valuable of our aids to diagnosis and therapy alike.

THE DIGESTIVE FERMENTS.—It should be clearly understood that the gastric digestion is but part of a complex process in the completion of which the following *non-gastric* ferments are concerned to a considerable degree.

(*a*) **Ptyalin.**—This constituent of the saliva is an important enzyme serving to convert starch to maltose, doubtless acting for an hour or more upon such portions of a full meal as are contained in the fundus of the stomach and for a considerable time protected from the action of the HCl which inhibits its activity. Prolonged action in stomach.

(*b*) **Amylopsin.**—This enzyme is contained in the pancreatic secretion and converts starch to maltose and achroödextrin.

(*c*) **Maltose.**—A constituent of the saliva, of the secretion of the small intestine, and of that of the pancreas. This converts maltose to dextrose.

(*d*) **Invertase.**—An enzyme of the small intestine which converts cane sugar into dextrose and levulose.

(*e*) **Lactose.**—This occurs in the secretions of the small intestine and converts maltose into dextrose and galactose.

(*e*) **Trypsin.**—This derivative of the pancreatic juice acts upon proteins.

(*f*) **Erepsin.**—Derived from the small intestine, this prepares peptones for absorption.

(*g*) **Steapsin.**—This substance splits neutral fats into fatty acids and glycerin.

Gastric Digestion not Indispensable.—*It will be seen readily that gastric secretions are not absolutely indispensable to digestion as is well demonstrated by cases of functional achylia and other conditions in which the gastric secretory functions are wholly lacking.*

Prepare food for absorption.

The ferments acting in the small intestine upon ingested food are peculiarly efficient in that they carry the processes to the point where actual absorption may take place.

Indirect Gastric Aids to Digestion.—The stomach permits a certain amount of absorption of water, dextrins, salts and peptones and by its acid content stimulates the production of secretin in the duodenum, which substance in turn increases the secretory activity of the pancreas, hastens the neutralization of the acid chyme and promotes the activity of the ferments.

Bile.—The bile assists in the conversion of the neutral fats and is believed to exert some inhibitory influence upon intestinal putrefaction.

Extent of Digestive Processes.—Enzyme activity extends from the buccal cavity to the ileocecal valve and absorption is especially active in the duodenum and jejunum, though the colon is also an important channel for the completion of absorption of the residuum of prepared substances.

The remainder of the ferments will be considered in connection with the examination of the gastric contents.

Genuine importance.

EXAMINATION OF DUODENAL CONTENTS.—Much information of genuine importance is obtainable by this procedure and although some of the finer reactions sought are somewhat time-consuming, others are extremely simple.

Both gastric and duodenal.

Present Field of the Duodenal Tests.—This is both gastric and duodenal for the tube lends itself admirably to *fractional** analysis of the stomach contents if swallowed immediately after the test breakfast is taken.

Ease of introduction.

Furthermore it is comparatively innocuous from the patient's point of view, especially if his attention is directed to the fact that it is no larger than the bulky drug capsule, or large food fragments, swallowed without difficulty. It is evident that in many instances the gastric "emptying time" will be defined very sharply and it should also prove useful in determining the presence of persistent hypersecretion.

Shows retention period.

Not the least of the advantages claimed for the duodenal tube, is the fact that it may be left *in situ* for as long a time as is desired.

* Fractional analysis demands that specimens of stomach contents be taken every 10 or 15 minutes throughout the entire period of digestion of the test meal; the tube remaining *in situ* until the stomach is empty.

Detects blood.

With respect to both the stomach and the duodenum it may be of considerable value in determining the presence of blood.

Occult blood, however, might be due to the procedure.

Yields pure content.

In the duodenum itself, its chief specific value is derived from the facility and promptness with which one may obtain the pure or relatively uncontaminated duodenal content.

Most of tests simple.

The clinically essential tests for pancreatic ferments and bile pigment and bile acids are extremely simple, and quickly performed. Cultures may be readily made if desired and have proven useful in cases of recovered typhoid with persisting cholecystic infection.

Cultures.

It is conceivable that the same procedure might prove useful in the puzzling first stages of typhoid fever with dominant primary cholecystic and duct symptoms.

The Duodenal Tube.—Many modifications of the original "Einhorn tube" are now available, one of the best of which is that of Rehfuss, which instead of a *perforated* bulb has one which is elongated and carries longitudinal slits, each of which in length is equal to the diameter of the tube and sufficiently roomy to prevent plugging in most instances.

Duodenal tubes are so marked as to indicate certain distances from the mouth of the patient to the terminal bulb, these corresponding, in the ordinary position of the stomach, to the distance of the cardia, the pylorus, and the duodenal portion itself, from the teeth of the patient.

In duodenal feeding one may need to introduce the tube 100 or even 125 cm. Tests for duodenal content begin ordinarily when the tube has entered for a distance of from 75–80 cm.

Method of Introduction.—This is simplicity itself in most instances.

The bulb is placed at the back of the tongue; the patient takes a few swallows of water and with tightly closed lips continues to swallow until the tube is fed in well beyond the 40 cm. mark indicating its passage through the cardiac orifice and entrance into the stomach.

Passes rapidly to stomach.

This consumes but two or three minutes if the patient is ordinarily docile and sensible.

He may *sip* water at will during the process, but the ingestion of any considerable quantity is undesirable. Little gagging or straining is likely to be encountered, but if this should appear, it is a simple matter to anesthetize the pharynx by a local application.

Anchoring the tube.

If considered desirable for any purpose to leave the tube *in situ* for several hours or days it may be made fast to the patient's clothing or to his cheek by a strip of adhesive plaster, and he may read, write, or sleep without discomfort.

The average passage time.

About one-half hour is required usually for the passage of the tube through the stomach and into the duodenum. Its passage through the pylorus is readily determined by touching a piece of red litmus paper to the liquid which presents at the orifice of the tube (if the "capillary-size" tube is used) or is secured by aspiration.

Alkaline secretion.

The reaction will be alkaline if the duodenal secretion has been attained.

Stomach contents may be acid or neutral, but never alkaline unless some such substance as magnesia or soda has been ingested.

Postures, during introduction.

It is customary to introduce the tube while the patient is sitting and, when 75–80 cm. have been swallowed, to place him in recumbency on the right side with the hips elevated.

Occasional delay.

More elaborate postural procedure is recommended by Holzknecht and other authorities but is seldom necessary. In some cases the passage of the tube from stomach to duodenum requires several hours or even a whole night.

Empty stomach necessary.

The patient must have eaten no food during the twelve hours preceding the test.

Sufficient stimulation of duodenal secretion is secured by the introduction of the bulb and the older Volhard-Boldireff method (oil breakfast) is no longer desirable.

The capillary (very small) tubes are to be preferred for diagnostic purposes.

Tests for hemolysis.

The work of Eppinger, Bondi and J. P. Schneider has demonstrated the extent to which the hemolytic process is reflected in the duodenal secretion in cases of plastic pernicious anemia and hemolytic jaundice *and Schneider has devised a relatively simple method of demonstrating the great increase of urobilinogen peculiar to these conditions.*

The qualitative tests for the various gastric ferments, for blood and for bile, have been given already elsewhere and the method of Dr. Schneider is here quoted from his interesting paper,* together with such portions of his "summary" as directly affect this procedure.

Color.—This is of prime importance and presents four distinct shades. *The chocolate-yellow* secretion is characteristic of pleochromie and it always yields a + + + value bilirubin. *The normal yellow* secretion may vary in opacity according as it is rich in bile acids admixed with hydrochloric acid. This opacity is by many erroneously regarded as mucus. The lighter shade of yellow is significant of a duodenal secretion poor in biliary pigment, just as the colorless secretion is proof of absence of pigment.

Bilirubin.—To 10 c.c. of duodenal contents are added 10 c.c. of an alkaline solution of calcium chlorid. After vigorous shaking this is filtered. The precipitate is dissolved under gentle heat in 10 c.c. of acid alcohol, and the resulting green solution concentrated to a given volume. By colorimetric comparison with a standard green solution† the quantity is indicated as +, ++, or + + +.

Urobilin and Urobilinogen.—To 10 c.c. of duodenal contents are added 10 c.c. of Schlesinger's solution, the whole thoroughly shaken and allowed to filter. The filtrate should be slightly alkaline; if not, a drop or two of dilute ammonia solution may be added. The filtrate will in the presence of urobilin show a more or less pronounced green opalescence. To 10 c.c. of this filtrate are added 1 c.c. of Ehrlich's benzaldehyd solution (paradimethyl-

* Archives of Internal Medicine, Vol. XVII, pp. 32–41, January, 1916.

† Ten grams of zinc acetate in 100 c.c. of absolute alcohol. A turbid solution which must be shaken.

aminobenzaldehyd, 2 gm.; acid hydrochloric, 15 c.c.; aq. dest., 15 c.c.). In the presence of urobilinogen a red color will develop. This solution is allowed to stand in a dark place for fifteen minutes, when it is subjected to a spectroscope* study for the values of both urobilin and urobilinogen. The acid character of Ehrlich's solution enhances the absorption bands of both these pigments many fold, the urobilinogen band being a very dense, narrow one in the yellow and the urobilin a wide one covering all of the blue into the green, the acid shifting the latter to the blue side.

The above solution is read in a graduate, and dilutions made with 95 per cent. alcohol, until a point is reached for each pigment where the absorption band will have disappeared at a fixed median position of the aperture, to again reappear with two revolutions of the aperture toward closure. The number of dilutions required for each element multiplied by twenty (5 c.c. of the original duodenal contents) will be the dilution value per 100 c.c. Following the basis used by Wilbur and Addis, the value has been reduced to the scale of 1000 c.c.

In developing this method as applied to the duodenal contents it would appear that bilirubin being so concentrated would present a practical difficulty in that it gives a diffuse absorption of the spectrum. Wilbur and Addis, working with the products of gall-bladder fistulæ, etc., made use of fullers' earth to remove bilirubin. The writer found that it did so but that it also removed especially urobilin. If the original 10 c.c. are well shaken with Schlesinger's solution before filtering, practically all of the bilirubin is removed, the retained quantity causing no error or inconvenience owing to the fact that the end-stages are read in a highly diluted medium where any slight diffuse absorption of the spectrum no longer operates.

That the above simple method is quite as accurate as one first removing bilirubin, was established by comparing the values thus secured with those prior to adding Schlesinger's solution.

A report of nineteen cases showing the enormous increase in urobilinogen in the five cases of plastic pernicious anemia included is followed by the summary given, in part, below.

Summary.—1. The early icteric feature of certain types of pernicious anemia is an expression of the primary fundamental hemolytic process of which the fully developed disease is a late bone-marrow exhaustion.

2. The excessive hemolysis of pernicious anemia is attended by both a pleochromie and urobilinocholie.

3. Pleochromie is an expression of the immediate hemolysis; urobilinocholie of the heaped-up pigment in the portal system.

4. Urobilinocholie varies directly as the portal system is surcharged or becomes relatively empty of the plus of pigment. It is highest in crises regardless of whether or not gross liver changes can be demonstrated.

5. Normally the duodenal secretion contains a certain level of bilirubin, occasionally urobilin, *but never urobilinogen in considerable amounts.*

6. The most constant blood finding in genuine pernicious anemia is the

* Kirchhoff and Bunsen, large model.

high index. This is an expression of the overplus of hemoglobin-building material heaped up in the liver.

Significance of Duodenal Findings.—*Impaired trypsin activity* is evidence of pancreatic disease and, in its persistent absence during several days of tests, the other ferments of importance need not be sought and the existence of pancreatic disease is established.

A very rough estimate of its activity, when it is present is furnished by the relative slowness or imperfection of its digestion of a disc of lightly boiled egg, as compared with the normal, exactly as in the test for pepsin.*

More accurate testing demands the use of a more elaborate procedure.

F. W. White finds that normal trypsin content is indicated by the digestion of 10 c.c. of a "$\frac{1}{10}$ per cent. alkaline casein solution"† in one hour in a water bath at 37°C. by the trypsin contained in 1 c.c. of duodenal content in dilutions of from 1 : 1,000 to 1 : 10,000.

Indicates pancreatic disease.

Bile.—The presence of bile in the duodenal content does not prove an unobstructed common duct for it may be present in marked degrees of constriction and also in cases of impacted stone or catarrhal jaundice despite its apparent or even actual absence from the feces.

Digestion test.

Its entire absence from the duodenal content strongly indicates occlusion of the duct by malignant growth; more rarely, complete block from other causes.

Therapeutic Value.—The value of the duodenal tube in gastric intolerance or subnutrition with absolute anorexia or repulsion is very great.

Many authorities state that as compared to rectal feeding, both being in skilful hands the actual increase of potency gained is as three to one.

It may be retained *in situ* for two weeks if necessary and is well borne by the stomach in most instances even though food and even liquids are promptly ejected.

When so used the food must be only warm, not hot, and is introduced, strained and in liquid form, every two hours, by means of a funnel or other attachable appliance, at a rate of flow of about 60 drops per minute, until 250 c.c. have been given.

Important clinical bearing.

To prevent overcooling during the half hour required for feeding by this drop method, a hot water bag may be laid upon the tube (F. W. White).‡

Duodenal feeding.

EXAMINATION OF GASTRIC CONTENTS.—Aside from the methods described under "Examination of the Abdomen," our knowledge of gastric

* Any pepsin present will be inactive in the alkaline medium and produce no confusion. Undigested casein is shown by a clouding of the solution upon the addition of acetic acid, at the end of the test period (one hour).

† Grübers pure casein 1 gram; dissolve in 1000 c.c. of a 1 per cent. solution of sodium carbonate; boil. No single test suffices by reason of the known variability of the trypsin content.

‡ Milk, cream, cane sugar or lactose and eggs, simply *stirred* into milk are the food substances most employed. Of these milk and cream are the most dependable and reënforcement by sugar and eggs must be cautiously carried out.

One cannot with propriety discuss the therapeutic aspect of this procedure in a work on diagnosis but this brief reference will serve to suggest the possibilities of the duodenal tube.

ailments still depends largely upon a consideration of the case history in connection with a chemical examination of the stomach contents taken after a test meal.

It should be clearly understood that the old belief in the sacrosanct inviolability of the stomach, upon which many tests have been based, can no longer be maintained. Quite apart from our recognition of the considerable part played by the salivary diastase in the stomach itself, stands the fact that, normally, the pancreatic secretion enters the stomach through regurgitation and initiates the digestion of the fats within the stomach itself. Pancreatic ferment activity.

We know also that in connection with malignant growths of this viscus special peptolytic and proteolytic agents become active and that similiar substance may be present in other gastric ailments. Nor does the chief ferment of the stomach itself confine its activity to the parent viscus. It is known to be active in the small intestine and even within the lymph stream itself. We are chiefly concerned nevertheless with the simpler phenomena of gastric digestion.

The test meal serves the purpose of stimulating the stomach to a degree which should produce the physiologic phenomena of digestion in full measure, if the stomach is normal, and reveal its weakness if it is abnormal.

Period of Rising Acidity.—*After the ingestion of food there follows a period of rising acidity. The secreted hydrochloric acid combines promptly with the proteins and alkaline bases leaving no free acids to inhibit fermentation by bacteria, hence from fifteen to forty minutes after a meal one would find hydrochloric acid abundant in combination, but absent as a free acid, while starch transformation would progress unhindered and lactic acid would appear because of fermentation or introduction with the food.*

Second Period.—*A second period follows during which the affinities of the proteins are satisfied, free hydrochloric acid appears and fermentation ceases. After certain test meals, this period should be attained an hour after the meal is ingested and one thus has a definite normal by which abnormal variations may be measured. This second period is followed after two hours or more by a third characterized by a fall in the acidity. An excess of proteins requires much acid for combination and means in all cases a delay in the appearance of free hydrochloric acid. An excess of carbohydrates acts in the opposite way, their acid affinities being promptly satisfied.* Third period.

Points to be Determined.—Primarily one seeks for *free and combined hydrochloric acid*. The latter represents a loose chemical combination with alkali-albumin and undergoes a progressive transformation by means of the acid and the action of the digestive ferments which results in the production of: (1) *acid-albumin;* (2) *proto- or hetero-albumoses;* (3) *deutero-albumoses; and finally;* (4) *peptones.*

Certain standard test meals are here given, but it should be remembered that any meal will give good *clinical* results if its constitution is known and that finical exactitude is not essential. The chief purposes of the test meal are to stimulate secretion and determine motility, one or both, and the *type* of meal is more important than the exact amount of food introduced. Basic purpose.

The Test Dinner of Von Leube and Riegel.—Four-hundred c.c. (12 to 14 ounces) of soup, 50 grams (2 ounces, or two ordinary slices) of wheat bread, 100 to 200 grams (3 to 6 ounces) finely divided beefsteak and 200 c.c. (6 ounces) of water constitute the meal. It is removed four hours later.

Ewald's Test Breakfast.—This consists of two slices of stale white bread or toast, or one or two baker's rolls, and from 300 to 400 c.c. (8 to 12 ounces) of water.*

Improved Meal.—*Two shredded wheat biscuits make a still better test breakfast, being wholly free from lactic acid and yeast cells.*

Best test breakfast.

These meals represent 35 to 70 grams (1 to 2 ounces) of solid, but the exact amount is not material. They should be taken in the morning and be removed one hour after the *commencement* of the meal.

Boas' Test Breakfast.—One tablespoonful of oatmeal is added to 1 quart of water and boiled down to 1 pint. It should be taken plain save for a little salt and removed in one hour.

This breakfast and the shredded wheat meal are especially valuable in cases of suspected cancer of the stomach, as they introduce no lactic acid, the determination of which is so important in this disease. In such cases the stomach should be washed out the night before or in the morning an hour before the meal is taken.

Ewald's breakfast seldom causes any confusion, lactic acid not being introduced in sufficient quantity to produce the ordinary clinical reaction suggesting carcinoma.

Effective Propulsion and Free and Timely Discharge.—An extremely simple test of freedom of discharge or patency of the pylorus consists in allowing a patient to eat a few cooked cranberries, raisins, prunes, black berries or other thick-skinned or stony fruits at night after the stomach has been completely emptied and thoroughly washed. The contents are removed in the morning one hour after a "shredded wheat" or "oatmeal" breakfast when the residue will show seeds, or skins, if actual decided stenosis is present, and also permit a chemical examination of the ingested morning meal.

A simple retention test.

Yet another readily available and informative method for general purposes requires the *removal of the stomach contents on the morning following the ingestion of a full test dinner or ordinary meal which includes some of the substances mentioned above.*

Bourget and Kemp's Retention Meal.—This extremely convenient test meal is composed of 200 c.c. of soup, 100 grams of chopped or shredded beefsteak, 50 grams of bread, and 6 stewed plums (prunes or raisins).

An excellent test breakfast.

This meal should be passed out of the stomach wholly and completely in *three* hours. By the amount and character of residue the degree of retention is roughly measurable.

* Tea is often recommended but should not be used. It to some degree modifies the activity of ptyalin and the production of hydrochloric acid, two points which, as Fuld puts it, overweigh both the lack of savor and the suggestion of prison fare embodied in the plain test breakfast.

Repeated observations at varying periods after the ingestion of the test meal will further establish the grade of retention present.

The grosser meals advocated have little or no advantage and some decided disadvantages as compared with a light mixed one of this type. Many cannot be retained by patients of the very type in which such test procedure proves most helpful.

Point of importance.

Much ingenuity has been wasted, vast amounts of time consumed and many diagnostic errors incurred through the highly creditable but wholly futile attempts made in the past to measure the propulsive power of the stomach and certify the adequacy and normal reactivity of the pyloric outlet by means of special and specific chemical tests.

So far as the author can determine each and every one of these has proven vitally defective and should be retained no longer in the text-book or in practice.

The death blow of the best of them was delivered by Pawlow in his discovery of the fact that regurgitation of pancreatic secretion into the stomach is not uncommon even under normal conditions and the effective check interposed by the roentgen-ray has done the rest.

Aside from the agency last named, one must place his reliance upon properly controlled and, usually, repeated test meals associated with such other data as are yielded by the history of the case and the physical signs elicited.

The X-ray has proven invaluable in relation to the determination of effective motility and is by *far the most reliable method.*

All procedures in which the determination of muscular and pyloric potency is dependent upon the amount of residuum recoverable by the tube is fallible even though very valuable, in that quite frequently complete removal of the stomach contents cannot be achieved.

It constitutes nevertheless the best method available to the generality of practitioners, but is better fitted to establish than to disprove gastric disability.

On all such occasions the patient should thoroughly masticate the meal, brush the teeth and cleanse the mouth after taking the retention meal in order that no coarse lumps may delay the passage through the pylorus and no indigestible particles remain in the buccal cavity, become detached at some later period and lead to spurious findings and false conclusions.

Two hours after the shredded wheat or Ewald test breakfast or six hours after the Riegel dinner, a stomach should be either entirely or approximately empty.

In actual extreme stenosis with pronounced secondary ectasia solid food remnants representing the residue of several previous meals may be present.

In mere atony such prolonged retention of undigestible remnants seldom or never occurs.

It is obvious that the contents must not only be expressed but further sought for through a thorough stomach washing.

The Macroscopic Appearance of the Normal Stomach Contents.—The normal fasting stomach should show only a few cubic centimeters of fluid of variable reaction and color, containing mucus and oftentimes a bile-stained mixture of gastric and duodenal secretions.

One hour after an Ewald test *breakfast* the appearance should be that of thin porridge, the bread in particles rather than distinct fragments, and little or no mucus present. The filtrate should be clear or faintly yellow, yielding a slight odor, and from 20 to 70 c.c. in amount.

A residue of less than 20 c.c. *suggests* too rapid emptying of the stomach; an amount exceeding 30 c.c. suggests retention, if rich in food remnants; otherwise hypersecretion.

The first inference is rendered uncertain by the difficulty often encountered in *completely* emptying the stomach. If, however, the stomach be washed before the tube is withdrawn a somewhat more accurate inference is obtainable through the appearance of the washings.

Abnormal.—*Anacidity* or marked hypoacidity is indicated by unchanged or slightly modified bread fragments, slow filtration usually, and a musty odor.

Hyperacidity is suggested by greater fluidity, foamy surface, stale, acid odor, rapid deposit of sediment and a larger liquid residue (70 to 100 c.c.).

In catarrhal gastritis the thick slimy content with evidence of subacidity is characteristic and in hyperchlorhydria with excessive motility or in achylia gastrica in which gastric digestion is *nil* owing to the entire absence of HCl and ferments, the stomach may be entirely empty.

Suggestive Odors.—Normal gastric contents yield no odor, a slight "bready" aroma, or an acid smell.

In cases of stasis one may recognize the alcoholic odor like that of *fermenting wine*, if yeasts are present in quantity; of *rancid butter*, if the fatty acids dominate; of sulphureted hydrogen, if albuminous putrefaction prevails.

Elaborate Chemical Tests and Instrumental Procedure.—Many elaborate and complicated procedures have been devised by enthusiastic specialists, and necessarily and properly by research workers, but fortunately for the student and practising physician every necessary and reliable test whether chemical or instrumental may be satisfactorily and oftentimes even more reliably performed by extremely simple methods.

Hence, throughout this section, the author will pay scant or no attention to methods or instruments whose chief merit is delicacy or super-refinement.

GASTRIC CONTENTS—CHEMICAL TESTS

QUALITATIVE TESTS.—Free HCl.—The following procedure is simple and convenient: (*a*) Test filtrate with congo-red paper or solution (congo-red 1, distilled water 100), or add to a portion of the filtrate 1 or 2 drops of *an alcoholic solution* of di-amido-azo-benzol (0.5 per cent.).

Simple tests for acidity.

Free hydrochloric acid is indicated by a decided blue discoloration with the former, or red with the latter reagent.

Theoretically, both tests show only that a free acid is present, but practically, *a decided color* change proves the presence of free hydrochloric acid.

Organic acids can only produce it when present in quantities never encountered in the stomach. Saturated and dried filter paper may be used in the di-amido-azo-benzol test if desired.

Tropeolin Test.—Tropeolin test paper is made by soaking strips in a saturated alcoholic solution of tropeolin oo. Dipped into the gastric contents and *dried over a flame*, it strikes a lilac blue color if free hydrochloric acid is present.

Günzberg's Test for Free HCl.—A few drops of resorcin solution (resorcin 5, cane-sugar 3, alcohol 100), or phloroglucin solution (phloroglucin 2, vanillin 1, alcohol 100) may be mixed with an equal amount of filtered gastric contents in a porcelain capsule or on any white non-absorbent surface and evaporated slowly to dryness, a *beautiful marginal rose color indicating free HCl.*

The Boas Test for Free HCl.—This test is applied in exactly the same manner as is that of Günzberg. The test solution is more stable, the color reaction less vivid.

Reagent.—Resublimed resorcinal..... 5 grams
Cane-sugar..... 3 grams
Alcohol..... 100 c.c.

Lactic Acid.—*In the absence of HCl, lactic acid should be sought by placing in the test-tube 2 drops of carbolic acid and 6 drops of neutral tincture of ferric chloride, adding water until a deep but clear amethyst color appears. The gastric filtrate after being treated as directed below is then added drop by drop and if lactic acid be present, a clear yellow reaction appears.*

The reaction is intensified by shaking up 5 c.c. of the filtrate with ten times its volume of ether, evaporating, adding 5 c.c. of water and applying the test. The use of unprepared filtrate makes the reaction very uncertain.

No attention is paid to doubtful reactions. Lactic acid should *disappear* as free HCl *appears* in the process of digestion and is always abnormal if found one hour or more after a test *breakfast.*

Simon's Modification of Kelling's Test.—This should replace the classical lactic acid test of Ueffelmann, because of its comparative immunity to the disturbing influence of glucose, the albumoses and the phosphates. Best test for lactic acid.

Test.—Fill a test-tube with distilled water and add a sufficient number of drops of a saturated aqueous solution of ferric chloride to give the solution a *faint* yellow tint. Divide into two equal portions, add to one a small amount of the gastric filtrate and compare with the control. *A distinct yellow indicates lactic acid.*

Quantitative Test for Lactic Acid.—The least complicated quantitative test is the simplified Boas procedure which gives results slightly lower than the true figures, but is sufficiently accurate.

Test.—To 10 c.c. of filtered stomach contents add a few drops of dilute sulphuric acid. Filter to remove albumin. Evaporate to syrupy consistence on water bath. Add water to regain original volume. Again evaporate to small volume to remove fatty acids. Shake up thoroughly with 200 c.c. of ether which takes up the lactic acid. Evaporate. Raise to original volume with distilled water. Titrate with a decinormal solution of sodium hydrate using phenolphthalein as an indicator. One uses the same notation as in the test for HCl, *i.e.*, multiply the number of cubic centimeters used by 10. In clinical work the game is not worth the candle.*

Other fermentation acids.

Acetic and butyric acids are revealed by the odor of vinegar and rancid butter produced upon heating. Butyric acid appears as small oily drops of characteristic odor if a bit of calcium chloride be added to an ethereal residue.

A more exact test for acetic acid demands treatment with ether, evaporation, neutralization of the ethereal residue with dilute sodium hydrate and the addition of a drop or two of dilute ferric perchlorid, but the clinical results are not worth the labor.

Fermentation present.

These volatile fatty acids redden litmus-paper, held over the tube during boiling and *indicate fermentation. Lactic acid is the only fermentation acid of marked clinical importance.* It proves the absence or extreme attenuation

HCl absent.

of HCl and the presence of a stasis and fermentation strongly suggesting carcinoma.

Pepsin and Pepsinogen.—*With normal or increased hydrochloric acid no test is necessary* as the presence of this acid proves also that of the ferment and in any event pepsinogen may be disregarded.

The entire absence of pepsin in the gastric contents clinically is essentially characteristic of so-called "functional achylia gastrica," true gastric atrophy or carcinoma.

Simple tests.

Test.—Prepare three tubes. (1) *Water* 10 c.c. + *pepsin* 5 *grains* + HCl *dil.* 3 *drops.* (2) *Filtrate of gastric contents* 10 *c.c.* (3) *Filtrate* 10 *c.c.* + HCl *dil.* 3 *drops*, add to each a disc of egg albumen about 1.5 mm. thick, and 10 mm. in diameter.

These should be constantly on hand and are readily prepared from slowly and *lightly boiled* egg, preserved in glycerine and washed before using. Cooking over a water bath is an excellent method.

Tube No. 1 serves as a standard of comparison. Digestion in No. 2 indicates both pepsin and HCl; in No. 3, No. 2 failing, it shows the presence of pepsin or pepsinogen alone. Digestion should be complete in about three hours at blood heat, but considerable variation is allowed.†

Relation to HCl.

As a rule, pepsin activity and acidity run parallel, but this rule may be

* Each c.c. of the decinormal solution used represents 0.009 of lactic acid. Hence, multiplying by 10 gives the actual amount present; by 100, the percentage.

† The quantitative methods of Thomas and Weber depending upon the digestion of a solution of casein in 0.2 per cent. solution of hydrochloric acid, and the still more complicated one of Mett, are too cumbersome for the general practitioner. The still newer *ricin method* of Jakoby-Solms and the edestin method of Fuld are described in the later literature but are open to the same objection.

departed from in extreme hyperacidity and certain functional cases of subacidity. If motor sufficiency is present or increased even a total absence of acid and ferments may cause few symptoms as is shown in many cases of "achylia gastrica" over long periods.

Rennin Test.—Add to 10 c.c. of milk 3 to 5 drops of gastric filtrate keeping mixture at blood heat. The appearance of coagulation within fifteen minutes indicates rennin. It may be entirely absent in carcinoma, achylia gastrica and atrophic gastritis, but is the most resistant of the ferments. Most resistant.

A positive test for rennin is almost absolute proof of the presence of pepsin and of free hydrochloric acid.

Lipase.—The elaborate test reaction for lipase is of interest only to laboratory workers as its only clinical importance lies in the diminution or entire absence of this ferment in achylia gastrica or atrophic gastritis, and this may be assumed if both HCl and the ferments are absent. In any event it is but slightly active. Slight value.

Albumin.—This should be absent one hour after a test breakfast.

Starch.—The digestive transformation of starch into *amidulin* by the action of ptyalin begins in the mouth. This later becomes *erythrodextrin* which strikes a violet or mahogany brown color with Lugol's solution. This in turn becomes *achroödextrin* (unaffected by iodin) and maltose (readily detected by Fehling's solution). Transformations. Tests.

It is evident that if the gastric secretion be normal the hydrochloric acid will prevent more than a partial conversion of the starch, despite the rapid action of the ptyalin, the extent of which depends primarily upon the thoroughness of mastication and secondarily upon the character of the food in relation to the penetration of the acid encountered and the time certain portions of an ingested meal are held in the fundus and protected in great measure from the acid. It is checked when the appearance of the free acid proclaims the satisfaction of proteid affinities and this is able to reach the food then being acted upon by the ptyalin. This ferment is doubtless more of a factor in gastric digestion than we had supposed until Cannon set us right.

Clinical Inference.—*A marked starch reaction in the stomach contents suggests hyperacidity; a marked achroödextrin reaction, diminished hydrochloric acid, and a marked Fehling's reaction, decided subacidity or absent HCl.*

Trypsin.—The pancreatic ferments are of interest chiefly in the examination of the duodenal secretion but may be found in the gastric contents even under normal conditions and especially after the injection of oil. They do not reveal their presence save in an alkalinized fluid. (See "Duodenal Content.")

Test.—A 1 per cent. solution of sodium carbonate may be used for this purpose and to the alkaline gastric juice contained in a test-tube, one adds a few flakes of fibrin which have been previously stained with Magdala red.

The fluid is then incubated and the digestive action of the trypsin progressively liberates the dye which in turn colors the solution.

Steapsin.—A few drops of neutral olive oil and gastric contents are mixed upon a watch glass and the mixture colored by the addition of alcoholic rosalic acid. It is then hermetically sealed by covering it with another glass and placed in a water bath for an hour when the disappearance of the original pink color will indicate the presence of steapsin.

Amylopsin.—Tests for amylopsin are of slight value in relation to the gastric contents because of the presence of the ptyalin of the saliva the action of which has been inhibited by the HCl ordinarily present.

The test is merely that of adding 1 or 2 c.c. of the filtrate to four times the amount of dilute starch paste and keeping the solution warm. In a short time Fehling's solution will show the conversion of starch to sugar.

Iodide tests.

The absorptive power of the stomach may be tested by the administration of 10 grains of potassium iodide, in capsules, which should produce in the sputum the characteristic iodin reaction with starch paper within ten or fifteen minutes. *Obviously the test is of very limited value.*

Solution No. 1.

QUANTITATIVE TEST FOR HYDROCHLORIC ACID.—*Take three portions (10 c.c. each) of the unfiltered gastric contents. Add to one 3 or 4 drops of phenolphthalein solution (1 per cent. alcoholic solution).*

Total acidity.

This substance, being the indicator for organic acids, acid salts and both free and combined HCl, upon titration with an alkaline solution of known composition will measure the total acidity.

To the second portion add 3 or 4 drops of alizarin solution (1 per cent. aqueous sol. alizarin monosulphonate of sodium). This, being the indicator for free acids and acid salts, measures the degree of acidity less the combined HCl, *i.e.*, the *physiologically active acid loosely combined with the proteids;*

Combined HCl Solution No. 2.

hence by subtracting the reading thus obtained from the total acidity (*which does include the combined acids*) one gets the amount of the combined acids.

Free HCl Solution No. 3.

To the third portion add 3 or 4 drops of dimethyl-amido-azo-benzol (0.5 per cent. alcoholic sol.), this being the indicator for free hydrochloric acid.

Color of mixtures.

Solution 1 is cloudy, white or grayish. Solution 2 is bright yellow. Solution 3 is cherry-red if free acid be present.

*The three solutions representing the gastric contents and the respective indicators are now each to be titrated with decinormal sodium hydrate solution.**

1 c.c. of which will neutralize 0.00365 grams of HCl.†

This neutralization is indicated by a maximum depth of the color obtained; therefore *the titration is continued in each case as long as color deepens, but no longer.*

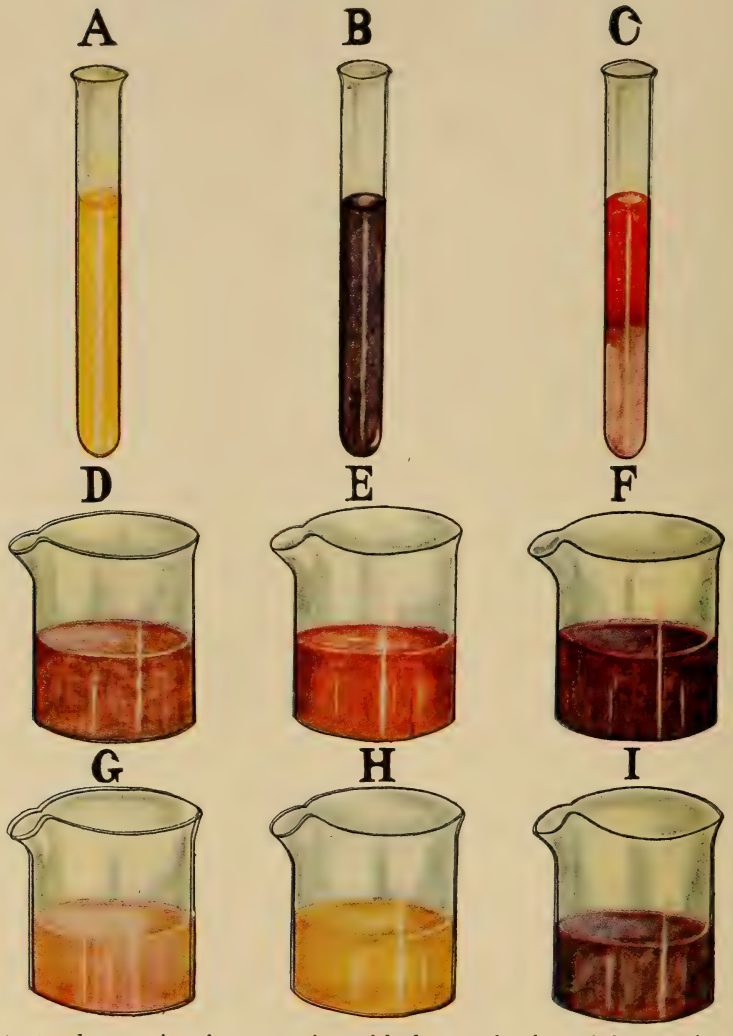
Red-total acidity.

END REACTIONS.—*These are: In solution 1 (for total acidity) a distinct and persistent red which does not deepen on further titration.*

* A decinormal solution = $\frac{1}{10}$ of the molecular weight of the substance, in grams, in 1000 c.c. of distilled water, hence in the case of NaOH the molecular weight being 40 the solution contains 4 parts in 1000 of distilled water. (A normal solution = the molecular weight of the substance in 1000 parts of distilled water.)

† The fact that the three right-hand figures correspond to the number of days in the ordinary year is an allowable crutch for a limping memory.

PLATE III.



Important color reactions in connection with the examination of the gastric contents. (Drawn from actual specimens.)

DESCRIPTION OF PLATE III

A. *Lactic acid* reaction.
B. Test fluid before adding stomach contents in Uffelmann's *lactic acid* test.
C. Di-amido-azo-benzol reaction for *free HCl* (qualitative test).
D. True terminal reaction for *total acidity* (phenolphthalein).
G. Antecedent color change in same test, *not* the terminal reaction.
E. Color of stomach contents after adding test solution for *free HCl.*
H. Terminal color reaction same test. (Color also nearly represents that assumed by stomach contents after adding test fluid (alizarin sol.) for combined HCl.)
F. Terminal color reaction *combined HCl.*
I. Antecedent color, *not* true end reaction same test. Test really represents all acids in stomach contents except combined HCl (see text), the result obtained being subtracted from the previously determined total acidity.

Violet-combined HCl. Yellow-free HCl.

In solution 2 (for free acids and acid salts) a pure violet.* In solution 3 (for free HCl) a *fixed yellow.*

One must carefully note at the commencement of each titration the exact reading indicating the height of the solution in the burette and again take it repeatedly on and after the first suggestion of the end reaction.

The number of cubic centimeters used in exact titration multiplied by 10, represents the degrees of acidity of the specimen in each case, for the amount of the decinormal sodium hydrate solution actually used represents the number of cubic centimeters found necessary to neutralize the acidity of 10 c.c. of the given stomach contents and multiplying by 10 expresses the amount which would be required to neutralize 100 c.c. This simple and convenient notation is the one in common use.

Example.—If the burette reading shows the use of 3.5 c.c. of the NaOH solution, we say that the acidity is 35 or 35 degrees; if 5 were used, 50, etc.†

Example.—0.00365 × 50 (degrees of acidity) = 0.182.

Total acids.

RECAPITULATION.—Sol. No. 1 (*phenolphthalein*) *reacts to free acid, acid salts, and the loosely combined acids, is grayish-white and cloudy and gives a red terminal reaction.*

Combined acid

Sol. No. 2 (*alizarin*) *measures the same acid contents as No. 1 less the loosely combined acid, thus necessitating for an estimate of the combined acid the subtraction of the result obtained, from the figures representing the total acidity. The original color of No. 2 is yellow, the end reaction violet.*

Free HCl.

Sol. No. 3 (*dimethyl-amido-azo-benzol*) *reacts only to free acid, is originally cherry-red, and its end reaction is a brilliant yellow.*

Normals.

Finally, *the normal total acidity is* 40–60 (0.146 to 0.22); *the combined acidity* 25 (0.09); *the normal free HCl* 20–40 (0.073 *to* 0.146) *after a test breakfast.*

After a Riegel dinner the total acidity is 110; *free HCl,* 44.

Sources of Error.—Töpfer's method as here given is by far the most accurate and convenient for clinical work, but is slightly inaccurate when free hydrochloric acid is absent, especially in the presence of an excessive amount of the organic acids.‡

These errors are not sufficiently great to disqualify this simple and convenient test for practical clinical use.

Hydrochloric Acid Deficit.—If free HCl is absent 10 c.c. of the unfiltered gastric contents may be titrated with a decinormal solution of this acid using

* A red-violet first appears and as suggested by Todd, the true color is best appreciated by comparison with a separate control solution composed of 5 c.c. of a 1 per cent. sodium solution to which 2 or 3 drops of the indicator have been added.

† The figures may be readily converted into terms of hydrochloric acid; for each degree represents the amount required to neutralize 0.00365 grams of hydrochloric acid.

Multiplying 0.00365 by the acidity expressed in degrees or by its exact equivalent the reading in cubic centimeters multiplied by 10 expresses the result in terms of HCl percentage.

‡ The methods of Sahli, Reissner, Martius, Hayem and Winter, Leo and Luttke, may be found in works devoted solely to clinical diagnosis, but are too complicated for he uset of the practising physician.

di-methyl-amido-azo-benzol as an indicator and continuing the titration until the red color indicates the presence of free acid. The result is expressed in exactly the same terms as are used in the preceding tests and represents the amount of HCl shortage, *i.e.*, the amount required to saturate the alkaline bases and proteins present. In cases where HCl is wholly absent this may replace the test for combined acids.

Effects of Diet.—Variations in the hydrochloric acid content, particularly in that of free acid will, of course, depend somewhat upon diet, and an excessive amount of saliva may neutralize the gastric contents; in the latter case the addition of a few drops of dilute HCl and 2 or 3 drops of ferric chloride solution to the stomach contents causes a red color reaction.

As the weak acid affinities of starch foods are readily satisfied and the demands of proteid substance is much greater, the highest acid values are found in meat eaters and the lowest in vegetarians.

Within normal limits, the more liberal the diet and the better the nourishment, the greater is the acidity. It is evident also that the real value of our figures is represented by the "combined" and "free" acid percentages.

The Diagnostic Bearing of the Foregoing Methods.—For hasty work the mere determination of free HCl is most readily achieved by the use of congo-red paper easily procured of any chemist, though it tells us nothing save the bare fact that free HCl is present. This being the case, lactic acid need not be considered or sought in the presence of a sharp reaction and the presence of normal ferments is usually a fair presumption. Too much dependence must not be placed upon the first test meal, for by reason of the emotional disturbance, the result may be misleading. The same rule may be applied to several successive tests, if, as seldom happens, the nervous disturbance continues to be excessive. Ordinarily we have to consider four conditions:

Rough deductions.

Get more than one test meal.

1. **NORMAL ACIDITY** (*Euchlorhydria*). (HCl 20 to 40)—This excludes achylia gastrica, chronic gastritis and usually carcinoma of the stomach. It does not exclude gastric atony and dilatation, nervous dyspepsia or the rare cases in which carcinoma develops upon an old ulcer base.

2. **HYPERCHLORHYDRIA** (Free HCl 40 +, *i.e.*, 0.146 per cent. or higher).—We distinguish between the secretion of excessively acid gastric juice during digestion (hyperchlorhydria proper) and the same condition appearing in the fasting stomach and representing more or less continuous *hypersecretion*.

As regards *hyperchlorhydria proper*, its chief clinical importance depends upon its frequent but far from constant association with ulcer of the stomach and the *possibility* that it is a factor in the production of that lesion.

Ulcer.

Simple hyperchlorhydria, moreover, is common in gastric neuroses without ulcer symptoms, *in the gastric crises of tabes dorsalis*, disease of the gall-bladder, and as a temporary condition due to exhaustion, emotional disturbance, constipation, dietetic errors, overuse of alcohol and tobacco or even to special articles of food.

Various associations.

Symptoms.—*Its symptoms are probably due chiefly to an associated hyperesthesia of the mucosa often attributable to distant sources and especially to ulcer or chronic appendicitis.*

Suggestive signs.

The most characteristic feature is the combination of slight or decided digestive disturbance (epigastric oppression, pyrosis, regurgitation, nausea, perhaps vomiting, relative or actual intolerance of starches, and occasionally diffuse pain and headache) with a good appetite and a clean tongue.

Dietetic and drug test.

The attacks appear from one to three hours after a meal, evidently marking the height of digestion and the maximum of free hydrochloric acid. They are relieved if vomiting occurs, or often (temporarily) by taking nitrogenous food and are in all cases ameliorated by the administration of an alkali. The attacks may also recur at the end of the digestive period and when the patient has been for many hours or even a day without food. In such cases starches may be almost wholly eliminated from the diet by the patient himself. Slight diffuse tenderness may be present and constipation is the rule though diarrhea is sometimes a troublesome symptom.

The stomach content is usually clear, acrid in odor and of low specific gravity. It will of course show high acid values and unconverted starches.* The urine at the height of an attack is usually neutral and deposits phosphates.

Pain.

In acute attacks the pain may be severe and strongly suggests ulcer save that it is more diffuse. As in many cases of ulcer it is relieved rather than increased by taking nitrogenous food, but the condition itself in most cases is corrected readily by attention to diet, *constipation*, and proper rest and recreations. Anemia and other evidence of marked malnutrition are frequently absent, but may result from limitation of ingesta. The appetite is frequently unimpaired, more often variable

Appetite.

Hypermotility is common and pylorospasm occasionally occurs.

There is a broad zone between normal acidity and actual hyperacidity which is symptomless or symptomful according to the irritability or non-irritability of the individual stomach and the line cannot be strictly drawn.

Asthenic Cases.—In general, the nutrition of the patient suffers but little, but one meets the condition frequently in the visceroptotic congenital asthenics and such are likely to show more or less decided weight loss or even a striking emaciation, such as is often present also in ulcer.

Pain Variants.—The pain is extraordinarily diverse in type and especially in severity, varying from mere discomfort to the severe attacks described above. "Hunger pain" is not only common but itself varies greatly in character in different cases and is sometimes constantly associated with nausea.

Hunger pain.

Epigastric Tenderness.—This is usually wholly absent or if present is diffuse and slight.

A word of caution.

Secondary or Complicating Hyperchlorhydrias.—The frequency of this condition as an accompaniment of some organic disease must be remembered.

* After a test breakfast the free acid which is the determining factor in the diagnosis will exceed 40, the total acidity 70. The stomach is frequently entirely empty one hour after the meal because of coincident increased motor activity.

Establishing the fact that hyperchlorhydria is present is but a single step in the diagnosis in many instances.

The condition of the gallbladder, hernial openings, female pelvic organs, appendix, the deep reflexes and duodenum deserve primary consideration.

The existence of the stigmata of congenital asthenia, evidences of psychasthenia, the excessive use of stimulants and narcotics as well as the question of overwork must receive attention.

One does not know how to strike a balance between the errors of commissivn on the part of the surgeon as represented by many futile and unnecessary operations and those of omission on the part of those physicians who treat the condition as a primary clinical entity.

Many cases, so regarded and treated, carry gastric or duodenal ulcers or a diseased gall-bladder.

Many others who might be cured by mere rest and bettered nutrition or proper treatment for an existing chlorotic anemia go to the operating table.

The history of the case must be carefully studied, due weight, but not blind faith, given to the absence of sharply localized tenderness, the absence of occult blood in the stool, to the X-ray findings *especially*, and to any and every means available for application to the differential diagnosis.

Fortunately these cases are for the most part distinctly unlike the urgent operative cases of the conditions they simulate and usually, therefore, can be given the benefit of careful observation and more or less extended medical treatment without incurring serious danger. (See" Gastric and Duodenal Ulcer.")

Under proper conditions the great majority make a prompt recovery. The congenitally asthenic individuals with visceroptosis and an atonic musculature should be shielded especially from hasty operative interference, for they are poor subjects for the knife.

CHRONIC HYPERSECRETION.—This includes two chief *syndromes* of hypersecretion usually marked by decided hyperacidity.

Continuous.

In **Teichmann's disease** *hyperacidity in the fasting stomach is continuous.* Though closely following the etiology of simple hyperacidity, it is often associated with ulcer, may occur in pyloric stenosis, simple dilatation or chronic gastritis, with impaired motility, and is a mere syndrome.

A mere syndrome.

Rossbach's disease (*nervous gastroxynsis*), also frequently associated with gastric ulcer, may be merely a chronic periodic or irregularly intermittent form of what was primarily simple hypersecretion. The symptoms are those of the ordinary "bilious attack" with nausea, bilious vomiting, epigastric distress and *diffuse* pain and tenderness. It is recurrent, lasting from one to several days at a time, frequently accompanies the gastric crises of tabes, but hardly deserves a place among actual diseases.

Recurrent.

Between the simple hyperchlorhydria and chronic hypersecretion stands the so-called "*alimentary hypersecretion*" which occurs only at the normal

digestion periods, the contents showing often an excess of 50 to 200 c.c. over the amount of ingesta.*

Caution.

Comment.—Admitting that for the present the foregoing classification of hyperchlorhydria and hypersecretion must be preserved, the utmost caution is necessary in accepting it in the individual case, because of the frequency of unrecognized gastric or duodenal ulcer and the tenuousness of the differential lines drawn. *It is true that in typical active ulcer the pain and tenderness are more distinctly localized;* yet often both these symptoms and hemorrhage are entirely absent, the ulcer going quietly on to spontaneous recovery or perforation. *The crises of locomotor ataxia,* moreover, may be associated with either hyper- or hypochlorhydria with or without hypersecretion.

In every case of periodic recurrent hypersecretion the first thought should be of pyloric ulcer with spasm, the second of locomotor ataxia.

3. **HYPOCHLORHYDRIA** (HCl under 20).—Low HCl values point to subacute or chronic gastritis, but are frequently met with in asthenic dyspepsias, early carcinoma, certain late forms of gastric ulcer, duodenal ulcer, dilatations *with or without stenosis, the early stage* of achylia gastrica, *and also in chronic disease of the gallbladder, pancreas and vermiform appendix.* Its value as a positive symptom is, therefore, slight.

4. **ANACHLORHYDRIA** (HCl absent).—As a symptom this is of positive and decided value, though met with in certain cases of asthenic dyspepsia, advanced chronic gastritis, as well as nearly all gastric carcinomas, pernicious anemia, achylia gastrica and true atrophic gastritis.

Unfortunately, according to the author's experience, many cases of dyspepsia apparently purely nervous, and especially those of congenital asthenia accompanied by marked malnutrition due usually in part at least to the common cause (viz., chronic starvation induced by foolishly restricted and monotonous diet), show this condition, the acid reappearing after a short period of treatment.

The sign is, therefore, chiefly valuable in connection with the associated symptoms (see "Acute and Chronic Gastritis," "Cancer of the Stomach," and "Achylia Gastrica") and the general discussion of the stomach contents.

Heterochylia (HCl variable).—This is merely a convenient term covering the cases of decided intermittent variations in secretion from day to day.

It may occur in health or in any form of gastric disturbance, functional or organic *and emphasizes the inadequacy of single examinations.*

CERTAIN PROMINENT GASTROINTESTINAL SYMPTOMS

Aside from the functional or secondary hypersecretion previously discussed, we may encounter so-called neuroses relating to pain, tenderness, peristaltic action, eructations, vomiting, motor spasm or relaxation, appetite and various paresthesias (see "Combined Gastric Neuroses").

* One of the most marked of the cases of continuous hypersecretion ever seen by the author was caused and maintained by the hourly or two hourly introduction of the stomach tube by the patient himself during the entire day and at intervals during the night.

APPETITE.—Anorexia.—Loss of appetite, though related to many conditions, may be described under this head.

It is present in the active stage of all fevers, in many chronic exhausting diseases and under the stress of violent emotion (grief, worry, anxiety). It may be purely hysterical or psychasthenic and take the form of mere loss of the hunger sense, actual repulsion, a sense of repletion, actual perversion, or nausea upon taking a slight amount of food.

Chlorosis, chronic alcoholism, hysteria, nervous and emotional overstrain, influenza and septic processes, including advanced tuberculosis are some of the best known examples, but again the author would emphasize the frequency of chronic anorexia as the result of the malnutrition of congenital asthenia, of dietetic fads and fancies and a narrowed monotonous regimen.

Again and again the most obstinate cases of dyspepsia with complete anorexia and excessive emaciation are promptly relieved under the rest cure and the gradual introduction of a varied and generous dietary.

Bulimia.—An insatiable and inordinate craving for food is one of the marked symptoms in certain cases of true diabetes and occurs normally in convalescence from fever such as typhoid and the exanthemata. It is met with as a nervous or hysterical manifestation as well as in cerebral disease, epilepsy, exophthalmic goiter, etc.

In the hysteric and psychasthenic cases it may be associated with an absence of the sense of satiety (*acoria*) or there may be a craving for unusual articles or such as are seemingly or actually injurious. Occasionally it assumes the form of a morbid desire for disgusting substances, as is sometimes observed in insanity, hysteria, pregnancy and chlorosis. To a less degree it appears in childhood, where it no doubt oftentimes represents in normal children a genuine structural need, in so far as it relates to special starches or proteids, too often combated by parents.

"Heartburn" (*pyrosis or "water brash"*) covers a regurgitation of stomach contents common to many forms of gastric disturbance, and "*merycismus*" (rumination), usually observed only in psychasthenia, hysteria, idiocy or epilepsy, covers voluntary regurgitation and remastication of the food.

Peristaltic Unrest (*Supermotility; Hyperkinesis*).—This troublesome excessive motility is accompanied by noisy gurgling and rapid emptying of the stomach, but may occur in the empty organ. It is a common transient phenomenon, but may be persistent in hysteria and neurasthenia.

Eructation (*Pneumatosis, Belching, Flatulence*).—This common phenomenon needs no explanation. Ordinarily negligible, it is a persistent, troublesome and characteristic symptom in certain "*asthenic dyspepsias*" associated with marked psychasthenia or hysteria.

The disturbing sense of distention and frequent "audible eructations" may humiliate a perfectly innocent patient and disgust and annoy those about him.

The term "pneumatosis" is properly applied to an extreme distention of the stomach by air, swallowed persistently by certain dyspeptics or by malingerers. The cause may be detected by placing a *large* cork between the teeth

The aërophagi.

of the patient and maintaining it there while the patient is kept under observation. The cure may often result from the same procedure, for this condition is often unconsciously produced by the patient.

"Corking up" usually convinces the aërophagi of the blamelessness of their stomachs with respect to such excessive gas formation.

The mouth should be kept *wide-open* to prevent the abnormally excessive air swallowing. *This condition is extremely common in a minor but troublesome form in the psychasthenic dyspepsias of the asthenic type.*

Nervous mechanism.

NAUSEA AND VOMITING.—*Vomiting may be either central or peripheral in origin, its center being closely related* to the respiratory center in the medulla.

In gastric disease the act is distinctly reflex, in toxemic conditions chiefly central. Purely psychic influences are capable of producing it, as is illustrated by hysterical vomiting and that associated with profound emotion or mental shock. Its relation to special sensation is shown by the involuntary response to excessively foul odors, disgusting tastes and shocking spectacles.

Bacterial or non-bacterial.

Toxemic Vomiting.—The toxins may be bacterial, as in the onset of the exanthemata, pneumonias, malaria, or severe acute infections in general; or non-bacterial, as in uremia, diabetes, Addison's disease, profound anemia or certain autointoxications. Furthermore, one must consider the action of such drugs as apomorphia, ether and chloroform, the effect of deleterious gases and the overuse of alcohol.

Long list of causes.

Reflex and Central Vomiting.—As a precursor or accompaniment of apoplexy, so also as a result of brain tumor, meningitis, thrombosis and embolism, vomiting may be a marked and somewhat suggestive feature.

In anemia both autointoxication and cerebral disturbance must be considered. It occurs in shock, collapse and occasionally in severe concealed hemorrhage, Ménière's disease, migraine, ocular muscular insufficiencies and refractive errors. Vomiting attending disturbance of equilibrium as typified by sea-sickness is well known and in diseases of the abdominal organs the stomach is of course the most frequent cause of reflex vomiting, whether there be actual inflammation, irritation, flatulence or mere disturbances of secretion. Under similar conditions it may attend ailments of related or neighboring organs, such as the large and small intestine, the appendix, the rectum, liver, gallbladder, pancreas, kidney and peritoneum.

As regards the respiratory tract, vomiting is readily induced by direct titillation of, or foul secretion adherent to, the naso-pharyngeal mucous membrane and may be associated with paroxysmal cough as in pertussis or pressure from mediastinal tumors.

Circulatory causes, aside from anemia and cerebral congestion or hemorrhage, may result from direct pressure, as in pericardial effusion or from secondary congestion of the abdominal viscera in valvular or myocardial disease.

Aside from the uremic vomiting of Bright's disease, the urinary tract is an infrequent source of vomiting, but the genital tract furnishes many examples, as in the vomiting of pregnancy, uterine displacement and ovarian or testicular disease.

Obscure Hernias.—According to the author's observation vomiting of an irregular, urgent, paroxysmal type, or a recurring causeless and equally erratic nausea result frequently from incipient inguinal hernias or tiny umbilical ruptures. Pain is often present but widely diffused.*

Points to be Especially Noted.—*These are (a) frequency; (b) the time of day or night; (c) pain and its persistence or intractability; (d) the presence or absence of any associated nausea; (e) its relation to meals both as to the character of the food and the time elapsing after ingestion; (f) the character of the vomitus, viz.: the amount, color, taste, odor, consistence, stage of food conversion, the presence of remnants of remote meals, and blood whether bright red, dark or of the "coffee-grounds" variety.*

EXAMINATION OF THE VOMITUS.—The vomitus affords information of great importance and vomiting sometimes occurs in such a manner and at such regular periods as to render the use of the stomach tube unnecessary in the very type of cases in which its use is contraindicated.

Test meal sometimes unnecessary.

On the other hand, its usefulness with respect to the exact determination of *secretion* is decidedly limited, for one can draw few inferences from a quantitative or qualitative analysis of the vomited material in cases characterized by an *acute* suspension of digestion.

Acute indigestion.

In some instances the regulation test meals of various types can be used with advantage, but only when the time interval between food ingestion and vomiting is approximately of the proper duration.

Test meals may be used.

The general appearance of vomited materials is too well known to need description and it is the significant variations that demand our attention.

The amount, scant or excessive, suggests hyperacidity and excessive motility on the one hand, hypersecretion, dilatation or atony upon the other.

Vomitus from a fasting stomach, if considerable in quantity, clear and of a sharply acid taste and odor, suggests hypersecretion; if the mucous content be large and the tongue coated, gastric catarrh.

Distinctly bilious vomiting is a common indication of acute indigestion, but if persistent may also point to pyloric incompetence and excessive regurgitation or to duodenal stenosis, simple, or associated with ulcerative or malignant infiltration. Such vomiting occurs in incipient or developed hernias, peritonitis and intestinal obstruction. If the two latter conditions persist, the vomitus soon presents an unmistakable fecal odor.

Bile.

Mucus may be present in all degrees, according to the nature of the lesion, but is rarely absent entirely even if HCl be present, and may take the form of *snail-like* masses.

Mucus which comes from the stomach itself is incorporated with the food, does not rise to the surface, and usually markedly slows filtration, if present in excess.

Gastric mucus.

In hyperacidity it is scant and soon dissolved and so usually points to sub- or anacidity if present in considerable quantities at the height of digestion.

That which floats on the surface is of no clinical significance, being derived

* As stated elsewhere, a mere relaxation of the ring may be sufficient to cause such distress.

Surface mucus.

from the pharynx and buccal cavity. This fact is important because of the false conclusions so often based upon the excessively large quantities so often present, especially in vomitus.

Pus may be visible in rare instances of gastric abscess or because of fistulous communication with abscesses of other organs.

In cases of poisoning the characteristic odor of the toxic substance may be evident and even in cases of gastric catarrh or the ejection of a meal rich in butter fat, the rancid-butter odor of fatty acids may be detected.

In advanced actively ulcerating carcinoma or in the presence of foul purulent material from whatever source, the odor may be peculiarly and offensively putrefactive.

In benign pyloric stenosis *with pronounced gastric stasis* the familiar odor of sulphureted hydrogen is often peculiarly characteristic.

Parasites.

Various parasites may be evident in rare instances to the naked eye. Such are round worms, thread worms and segments of tape worm.

Peculiar tints.

The color aside from food admixture and discoloration is light yellow in anacidity, green, if bile contamination is present or the development of certain yeasts has taken place; but is ordinarily dirty gray or greenish brown and, rarely, rose color from bile decomposition with high acidity, or, in the crises of tabes dorsalis (Lorenz) with hypersecretion.

Bile may be present in small quantity even in a normal stomach and in health and occur in any vomitus if the efforts are prolonged. If present in quantity at the outset or in repeated slight attacks it suggests, as stated, pyloric insufficiency or duodenal infiltration.

If, in the case of vomiting several hours after a meal, the gastric content, when set aside in a high glass jar, shows a lower chyme stratum equal to or exceeding one-half the total height of the mixture, it suggests stenosis or acute retention.

Coffee grounds.

HEMATEMESIS.—If the blood be recently effused, it is bright red and fluid; if partially digested, it resembles coffee grounds; if in an intermediate stage and in sufficient quantity, it may be clotted.

How simulated.

The appearance of blood is simulated by jellies and jams, cocoa, coffee, beef juice, such drugs as iron and bismuth, the dark red wines, grape juice, and fruits, such as currants, cranberries and cherries.

Amount.

The amount of blood may be large or small and the small amounts may be derived from any source; indeed a mere streaking of mucus may accompany excessive and persistent vomiting and of itself indicates no serious lesion. On the other hand, hemorrhages of all degrees occur in ulcer and carcinoma, splenomegaly, certain cachectic conditions of the hemorrhagic type (such as advanced leukemia, hemophilia, scurvy, and purpura), in esophageal lesions, in virulent infections chiefly associated with tropical fevers, such as yellow fever and pernicious malaria, and as the result of the action of irritant and corrosive poisons. Blows and wounds, the varices of portal obstruction, chronic heart disease and vicarious menstruation, also must be considered.

Source of Error.—In nosebleed, hemoptysis, ruptured tonsillar abscess, and similar conditions the blood may be swallowed and its source may be

recognized only through the presence of the causative factors named. In hemoptysis as opposed to hematemesis, the bright red finely frothy blood of alkaline reaction is raised by coughing and such bloody sputa ordinarily persist for several hours. There are usually significant or even localizing physical signs and oftentimes distinctly or characteristic subjective sensations referred to the thorax.*

Chemic and Microscopic Tests for Blood.—The following test will apply equally to blood in the stools and in the stomach contents and it must be remembered that an examination of the stools is of the utmost importance in all cases of supposed carcinoma or gastric ulcer, vomiting being frequently absent and slight hemorrhages unrecognized.

Furthermore, any effused blood is usually promptly removed from the stomach and, in general, the feces usually prove a more satisfactory medium for its detection than the gastric contents. **Stools or stomach contents.**

The Weber-Müller Guaiacum Test.—If solid, a small portion of the suspected material should be thoroughly subdivided and shaken up with a little distilled water. **Extremely accurate.**

Transfer 2 c.c. of the suspected mixture to test-tube; add a few drops of acetic acid and 4 c.c. of ether. Shake up thoroughly and carefully decant the supernatant ether into a clean test-tube. Add a pinch of guaiac resin and shake thoroughly but briefly. Allow an amount of ozonized turpentine equal in volume to that of the remaining ethereal mixture to run down the side of the test-tube and underlay. If blood is present a blue ring forms at the junction of the ether and turpentine layers and becomes diffused upward.

Teichmann's Test.—This requires the careful evaporation of the suspected material on a glass slide, after which a crystal of sodium iodide or sodium chloride is added, followed by a drop of glacial acetic acid. Too rapid evaporation is prevented by the use of a cover-glass and it is then gently heated to the exact boiling point, fresh acid being added as evaporation goes on. When a brown color appears, indicating the formation of hemin, complete evaporation is allowed and a drop of water run under the cover-slip. The microscope will then show the characteristic black or brown rhomboidal crystals of hemin. **Valuable test.**

Benzidin Blood Test.—Mix fresh each time the following solution: Benzidin, an amount that will stay on the point of a pen knife; glacial acetic acid, 2 c.c.; hydrogen peroxide 1 c.c. If solid material is to be tested, mix it thoroughly with a small amount of water. Put a drop of the mixture on a slide and place the cover-slip over it. Then allow a few drops of benzidin solution to flow around the edge. *A blue or green color will develop at the line of contact in the presence of blood.*

For liquids, if opaque, proceed as above; if translucent, place a few cubic centimeters in a conical glass and allow the benzidin solution to flow slowly down inner edge. *A blue ring will develop in presence of blood.*

* Though in hemoptysis the blood is raised by coughing, vomiting is a frequent accompaniment and may predominate, the cough being slight. Hence too much stress should not be laid upon this as a differential factor.

Weakness of Benzidin Test.—Potatoes, milk and farina or similar substances may yield the benzidin color reaction but the Ewald meal does not

FIG. 445.—(See Fig. 447.) Boas-Oppler Vacilli.

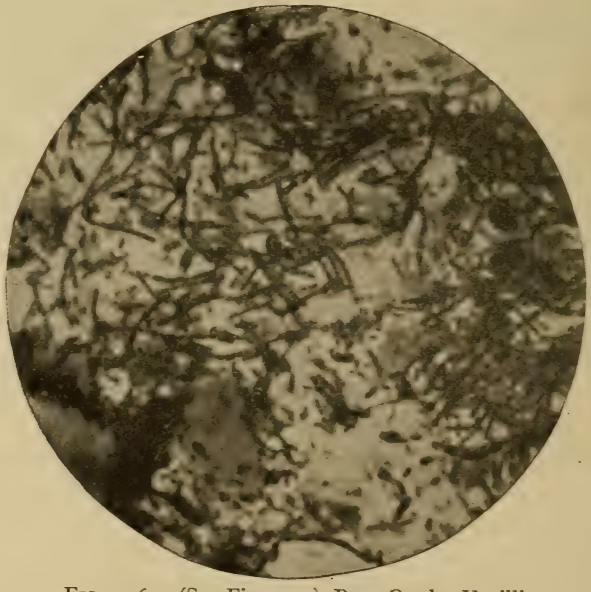


FIG. 446.—(See Fig. 447.) Boas-Oppler Vacilli.

produce it. Unfortunately, the ingestion of cooked meat even if taken forty-eight or seventy-two hours previously may cause a false reaction. *The Weber-Müller test should be given preference.*

GENERAL MICROSCOPIC FINDINGS.—The food detritus depends upon the character of the meal previously taken and the time elapsing since its ingestion, but, in general, consists of muscle fibers and starch granules, more or less changed by cooking and digestion, the former being recognized by their markings, the latter by their peculiar form and structure or their blue reaction with Lugol's solution. Skins of fruit, seeds of berries or even the more digestible substances, if remaining from a remote meal, point to impaired motility or actual stenosis.

Starch and muscle fibers.

Indigestible residue.

Saliva is indicated by pavement epithelium and the so-called salivary corpuscles, and the various vegetable cells are usually so peculiar and bizarre as to at once suggest their nature.

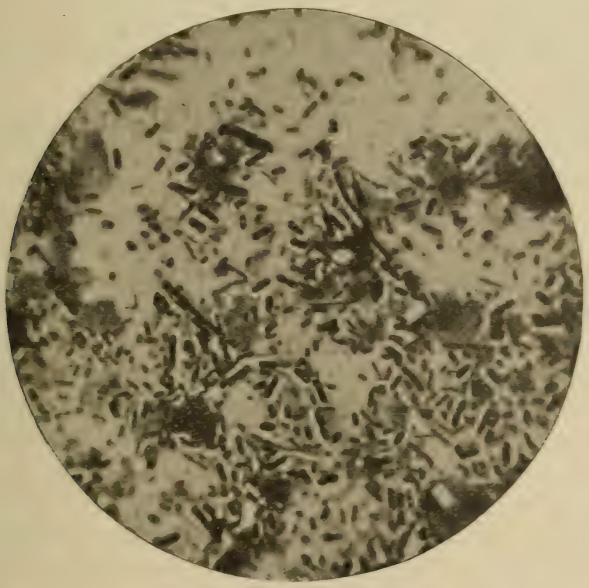


FIG. 447.

Photomicrographs of feces from three cases of advanced pyloric carcinoma showing Boas-Oppler bacilli in the stools. In all of these the organisms were most abundant in the gastric contents. The Boas-Oppler bacilli are seen as large segmented organisms slightly different in form in the three specimens.

In Fig. 445 they are shown with many of the other forms of fecal organism, among which their size easily distinguishes them, and in Fig. 446 they are the predominant bacilli.

Fig. 447 shows many short, thick-rodded bacilli that may be mistaken for the Boas-Oppler, but the difference in size between these (bac. aërogenous capsulatus) and the Boas-Oppler will be noted by comparing the first with the second forms, about half a dozen of the Boas-Oppler being shown in the same field. × 1000. (Bassler's "Diseases of the Stomach and Alimentary Tract," copyright, F. A. Davis Company, 1916.)

Fat may appear in the form of crystals or of the highly refractile droplets soluble in ether, staining orange-red with Sudan III, and black with osmic acid (1 per cent.). The presence of a few red blood corpuscles after vomiting or the passage of a stomach tube is not necessarily pathologic.

Vegetable Parasites.—The Boas-Oppler bacillus, the *sarcina ventriculi* and the tubercle bacillus are the most important of the organisms found in the stomach though it seems possible that in the near future the streptococcus

may be found to play an active part in the production of some of the more important organic lesions both gastric and duodenal.

Boas-Oppler vs. Leptothrix.

The Boas-Oppler bacillus is apparently a pleomorphic microörganism but if present, is commonly seen in the form of *large long rods* frequently arranged in chains or zigzags and staining brown with Gram's solution, whereas the leptothrix which resembles it stains blue.

High degree of stagnation with impaired motility, absence of free hydrochloric acid and the presence of lactic acid represent the conditions most favorable to this microörganism.

It is obvious that gastric carcinoma of the stenotic type is the ailment which oftenest and to the highest degree produces the conditions which satisfy these requirements. Moderate degrees of stenosis, malignant or not, are not sufficient to permit the development of these bacilli.

They are always present in large numbers if found at all and usually abound in the large clots sometimes met with in cancer.

*They invariably co-exist with lactic acid and with rare exceptions the acid is present in large quantities.**

They never co-exist with hydrochloric acid and the finding of the one proves the absence of the other.

In advanced stenotic gastric cancer they are almost invariably present. It is doubtful if they are ever present in the benign stenosis or in primary atony in any considerable number or otherwise than as an accidental or fortuitous finding.

The Sarcinæ.—*These indicate stasis and fermentation processes, require the presence of hydrochloric acid and though present in large numbers in many benign cases of that type, are seldom or never found in cancer of the stomach showing an absence of HCl. Both the large and small varieties usually assume the form of bales.*

Yeast fungi are present in large numbers in marked degrees of atony or ectasia with absence of HCl, and in hyperchlorhydria unchanged starch granules will be plentiful in cases showing decided subacidity and muscle fibers will be correspondingly numerous in their definite and unchanged form.

Cancer Cells.—Rarely one finds detached particles of new growths, but more often such fragments are absent or unrecognizable.

DISORDERS CHARACTERIZED BY SENSORY, MOTOR AND SECRETORY DISTURBANCES

GASTRIC SPASM.—Spasm may occur either at the cardia or pylorus, and both may be purely nervous phenomena, the former almost invariably so, the latter being most frequently associated with hyperacidity or actual ulcer; in either, there may be a subjective localized sensation of obstruction. Both may be painful and the latter may produce visible or even reversed peristalsis, and, if long continued, lead to actual dilatation. If the two conditions are simultaneous (invariably neurotic), the marked overdistention may be evident. (See "Roentgenography.")

* Fuld states that in rare instances the bacilli may be found when only the finer tests reveal lactic acid.

Gastric Hyperesthesia.—Marked gastric intolerance with a sense of weight, fullness or burning and without an organic basis may be encountered in hysteria, asthenia, exhausting and debilitating disease, mental and physical overstrain, profound anemia, shock and forced or voluntary fasting over long periods. It may be associated with marked diffuse tenderness corresponding to the stomach outline and is probably a chief cause of symptoms in simple hyperchlorhydria, and in many cases of hypersecretion.

Often misleading.

Gastralgia has been previously referred to as a misused term representing in most instances appendicitis, gall-stones, gastric ulcer or cancer, the gastric crises of locomotor ataxia, splanchnic arteriosclerosis or angina pectoris.* A true neuralgia of the stomach is exceedingly rare, and whether such a condition exists save as a secondary ailment is questionable.

A rare condition

ANOREXIA NERVOSA.—This curious condition, rare but by no means a clinical curiosity, is characterized by absolute loss of appetite and frequently by abhorrence, and prompt ejection and rejection of all foods. It may follow prolonged fasting (starvation "cures"), shock, grief and the like, or, as is usually the case, be associated with hysteria or profound psychasthenia.

In hysterical cases, especially, the patient may be profoundly or but slightly anemic, will *promptly* vomit all food administered, complain of vague and often shifting abdominal pain and tenderness, and frequently pretend to vomit blood, or, if posted, imitate the painful crises of ulcer.

Emaciation may be extreme and a fatal issue is possible, if proper treatment is not available.

Absolute rest, isolation, suggestion and forced feeding is necessary and firm control by a competent nurse and physician will usually clear up the diagnosis and cure the patient.

One of the most characteristic features is *the almost instant vomiting of* the ingested food, though, usually, water will be retained and very frequently the act is obviously little more than mere regurgitation.

All cases so far encountered by the author have proven amenable to the effect of firm insistence combined with complete change of environment and absolute exclusion of the sympathetic oversolicitous family and friends.

In some cases the simulation of gastric ulcer has been absolute until the opportunities for deception with respect to hematemesis were removed and ocular demonstration demanded in place of the hearsay evidence previously imposed upon family and physician alike.

ACHYLIA GASTRICA. (*Apepsia Gastrica*).—**Definition.**—*As the name indicates, the achylias represent a total lack of digestive ferments and of hydrochloric acid free or combined in the gastric juice.*

This interesting condition as at present represented in medical literature is one of the most blurred of all clinical pictures, but, in practice, it resolves itself into a reasonably clear and definite grouping.

Two main groups.

1. *Those achylias which yield no evidence of being distinctly secondary to or associated with any other organic disease.*

Primary.

* With epigastric maximal pain localization.

Secondary.

2. *Such achylias as are clearly secondary to or associated with a dominant chronic organic ailment.*

Group I.—SIMPLE ACHYLIA.—This contains cases of achylia of special interest and primary importance from the standpoint of their apparent clinical independence.

Subdivisions.

In these we find two chief divisions based upon the physical type of the patient; (a) the depressive or asthenic type; (b) the active type.

In the former may be placed the most interesting examples of this peculiar condition and it probably embraces the greater part at least of the *congenital cases* which may be far more numerous relatively than we can assume justly at the present time.

The "Active Type."—This needs no special description, but is remarkable in that it presents absolutely no clinical, and but little, if any, pathologic, anatomic, or physiologic, basis for the presence of the achylia itself.

The signs in cases of this type, aside from the gastric findings, are usually negative, in the clinical sense, to an extraordinary degree. The patients often show no striking subjective symptoms, in many instances suffer little if any weight loss over long periods, and wholly lack the general anatomic physiologic and psychic stigmata of the "passive" type of achylia.

Actual but transient simple achylia may occur during profound emotional crises, on the first day of menstruation, or in association with severe diarrheas. Many of the psychasthenic victims of asthenic dyspepsia seem to pass from hypochlorydria to achylia and back again under their own exaggerated play of emotions.

We are concerned here only with the persistent types, yet must not forget the liability to erroneous conclusions in the heterochylic cases and the proof it offers of the absolute indispensability of repeated examinations.

The Passive Type of Achylia.—This is the form occurring in those carrying the stigmata of "congenital asthenia," elsewhere fully described.

In these individuals achylia *may* exist for years without symptoms if their general nutritional balance and reserve are well maintained, but the congenital tendency to instability and inadequacy of function as reflected in anatomic structure and in the labile psychomotor sphere usually make them ultimately more or less typical "nervous dyspeptics."

In nearly all of them visceroptosis and muscular relaxation of varying degree are *easily demonstrable* and not a few show distinct evidences of gastric atony.

General Symptoms.—There are no subjective symptoms peculiar to achylia, but in its advanced stages the various and variable manifestations of so-called "dyspepsia" may individually or collectively appear and disappear or become relatively fixed and permanent. Flatulence, however, is rarely troublesome unless motility is impaired.

As time passes, periods of more or less severe diarrhea supervene, due apparently to a progressive inability of the intestinal digestive ferments to carry the overload represented by an entire absence of gastric digestion, together with the premature and excessive expulsion of the gastric con-

tents replacing the proper rhythmic fractional release of the gastric content which, in health, is maintained by the "*acid reflex.*"

Doubtless the stimulus to pancreatic secretion is also measurably impaired by the achlorhydria and a secondary state of *achylia pancreatica* may possibly occur in rare instances.

The decided value of small, frequently repeated feedings is in accord with the facts outlined above.

As time passes, the intestinal inadequacy and irritability often increase, the patient's nutrition suffers, and decided emaciation and cachexia may develop.

Such periods may come early, very late, or never. They may at times be held in check for many years by proper treatment or by it be rendered of only rare occurrence.

Cases showing repeated, more or less frequent diarrheal attacks, of somewhat prolonged duration, should be suspected of, and examined for, achylia, and also, its chemical opposite, hyperacidity. Such seizures occur in both conditions and show a predilection for an early morning maximum and an afternoon recession.

The author has yet to see an actual death chargeable to simple achylia though he has watched certain cases for many years, but such patients may and do become weak and anemic and many fall readily under the attack of some intercurrent ailment.*

Stomach Findings.—*The diagnosis of achylia rests wholly upon repeated examinations of the stomach contents, both following test meals and when wholly empty.*

The differentiation of simple from secondary achylia is attained only through the careful elimination of primary dominating organic disease not alone through the examination of stomach contents, but by complete and thorough general investigation.

Examinations of the stomach contents should not be made during severe diarrheal seizures, or, in the case of women, on the first or second day of a menstrual epoch.

During such periods the wholly normal individual may present a typical achylia.

The characteristic findings are the following:

First.—The recovered stomach content one hour after the test breakfast shows macroscopically no digestive change, the bread fragments appearing as if just taken into the stomach.

Second.—The gastric content is small in amount and, in fact, is often wholly absent, after the interval usually observed, owing to the absence of the normal "acid reflex" which adjusts pyloric activity and consequent premature discharge of the contents into the duodenum.

It is frequently found necessary to express or aspirate the meal within three-quarters or even half an hour of its ingestion and the combination of

* Two cases of achylia simplex at present under the observation of the author are not only well nourished but plump and have absolutely no gastric or intestinal discomfort.

evident reduction of content and unchanged food is in itself almost pathognomonic of achylia.

Third.—The fasting stomach after a "retention test meal" shows no residue indicative of obstruction or stasis.

Fourth.—Under chemical tests a faint acidity *may* be demonstrated which is due to traces of organic acids, but hydrochloric acid whether free or combined is wholly lacking.

Fifth.—The usual tests for pepsin and for labferment are wholly negative or yield only such slight traces as are clinically negligible.

Sixth.—The amount of mucus present is extremely slight or only moderate in amount, in striking contrast to the achylias of certain rare types of true chronic gastritis.

Heterochylia.—In certain cases of that form of achylia associated with chronic congenital asthenia and its associated nutritional instability and psychomotor lability, no less than in achylias of the "active type," associated with convalescence from acute illness, overwork, worry or emotional strain, such findings as have been given may alternate with, be interrupted by, or, in the latter group, actually replaced by periods of hydrochloric acid secretion and ferment secretion.

This constitutes the so-called "*heterochylia*" referred to elsewhere which, as stated, leads to many errors both of commission and omission if diagnosis is based upon the result of a single test meal.

Such contradictory findings also may precede for long periods the full development of a persistent achylia.

Motility.—The motor activity of the stomach may be wholly normal, increased, or distinctly diminished in some of the asthenic cases with atony and gastroptosis, but such impairment seldom or never is excessive.

Hence, "achylia" with true stasis and food stagnation, even though these three conditions may stand alone, demands surgical exploration.

Anatomic Changes in the Stomach.—In many cases the stomach itself has been found to be wholly normal, but in others *an extremely low-grade* granular gastritis may exist which is not believed to be primary or to merit the name of an actual disease.

Friability and Vulnerability of the Mucous Membrane.—In many cases of achylia the stomach is extraordinarily susceptible to bruising and abrasion, and this is doubtless especially true of those cases which present the degenerative changes described in the foregoing paragraph.

Fragments of the friable mucosa often may be recovered readily by the stomach tube in such instances, and submitted to microscopic examination.

Hemorrhage.—Erosions are not uncommon and may be quite symptomless or sufficiently severe and extensive to cause occult blood to appear in the feces or produce an actual hematemesis. Pain sufficient to suggest round ulcer of the stomach is occasionally encountered, but the gastric findings and case history are usually sufficient for differential purposes.

The Stools.—The stools of such patients as show diarrhea are usually highly suggestive if meat is taken over a period of several days. They con-

tain, as a rule, undigested muscle fiber and a considerable excess of unchanged connective tissue.

In severe cases the evacuations are liquid and highly offensive. Mucus may be present in quantity if the small intestine has become inflamed and, in this event, will be mixed intimately with the food remnants.

Frequency and Age Incidence.—Achylia is probably far more frequent than clinical statistics would indicate and is found in about equal percentage in each decade beginning with the third and ending with the fifth. In the second it is about one-half as frequent; in young children, supposedly extremely rare, perhaps because of lack of tests.

It must be said that all such figures fail to show true incidence when applied to an ailment so silently long-enduring and so generally overlooked.

Pernicious Anemia and Achylia.—Atrophic gastritis has been described as a separate ailment associated with a blood picture of anemia.

It would seem more rational to say that in pernicious anemia practically all cases show a decidedly low hydrochloric acid content, and many an entire absence of both hydrochloric acid and ferments, *i.e.*, a secondary *achylia*.

In rather more than half of these cases an actual atrophy of the stomach is demonstrable *post-mortem*.

This apparent atrophy is regarded by many as a post-mortem phenomenon, but the observations upon the fragments of mucosa obtained from the stomach of living cases of pernicious anemia would indicate that the change actually exists *intra vitam* to a considerable extent.

It is certain, nevertheless, that many cases of that ailment run their course lacking both achylia and atrophy and equally so that achylia often goes to the final exitus without gastric atrophy.

High grades of secondary or chlorotic anemia may accompany true simple achylia and few achylic patients carry wholly normal blood findings.

Differential Diagnosis.—The differential diagnosis of simple achylia depends primarily upon an analysis of the stomach contents and the absence of high grades of stasis and stagnation of either the malignant or benign type.

The utmost caution should be observed with respect, not alone to the primary examination of such patients, but also the course of the ailment after discovery, for early carcinoma (the only operative stage) may present for a time a strikingly parallel picture.

Chronic gastritis, save for the rare, terminal atrophic type, is at once ruled out by the absence of mucus in excess or in quantity in the recovered test meal.

Terminal chronic nephritis or diabetes and advanced pulmonary tuberculosis may be associated with achylia but are easily recognized.

Senile achylia represents apparently nothing more than the usual secretory and circulatory deficiencies of old age carried to an unusual degree. *Senile hypochlorhydria* and achlorhydrias are a common event.

Disease of the gallbladder and chronic appendicitis must also be borne in mind always as possible causes.

The achylia of malignant disease of the stomach is associated with the signs and symptoms of that ailment as fully described under its proper heading, but, as just stated, great difficulty may be encountered in relatively early stages of the malignant growth at which period stasis and stagnation of high grade may be absent.

The old observation of Fenwick, that achylia may result from the presence of progressive carcinomatous growths in regions other than the stomach has been in some measure confirmed by modern workers and should at least be held in mind in estimating the importance of the achylia in the individual case and as a stimulus to thorough and complete examinations.

GASTROPTOSIS.—The general topics of "visceroptosis" and "congenital asthenia" cover this condition.

Gastroptosis may and usually does exist without dilatation or even demonstrable peristolic atony. It may be symptomless or associated with psychasthenic and asthenic dyspepsia or decided motor insufficiency, though the latter is rare save in the severer forms or in the presence of the other recognized factors.

The tendency to both persistent and recurring *tonus-inadequacy* is nevertheless marked by reason of the basic constitutional defects. (See Congenital Asthenia, "Drop" Heart, etc.)

Among the commoner symptoms of active cases are: subjective distention; troublesome borborygmi, especially if tight clothing or bands are worn; tenderness over the solar plexus; subjective aortic pulsation, and, in the presence of sacro-iliac relaxation, a dragging sensation or dull pain referred to the region of the sacro-iliac joint or lower lumbar region.

Such symptoms are not due to gastroptosis which in the greater proportion of cases is relatively or wholly symptomless, but is chiefly a reflection of states of depressed nutrition and impaired circulation.

As stated elsewhere, ptosis of the stomach is but one manifestation of the general visceroptosis of congenital asthenia. According to the author's observations, finding a gastroptosis one may predict the presence of the "drop" heart (or *vice versa*) and, less constantly, some demonstrable degree of abnormal renal displaceability. (See Fig. 386.)

Floating kidney, a typical "drop" heart and gastroptosis of the 3d degree (pelvic) go very generally together.

Thence one ascends through the 2d and 1st degrees of definite gastroptosis to the common fish-hook type, lying above or at the navel in the standing posture, and finds the ideal stomach in the transverse "steer-horn" type whose pylorus constitutes its lowest portion.

Grades of "drop" heart, closely following, in the degree of their departure from the normal size and outline, the grades of descent in gastroptosis, are quite clearly demonstrable.

Exceptions of course may occur especially in men for reasons stated elsewhere, and the modification of the "drop" heart outline due to actual disease of the heart or kidneys, together with an unfortunate adherence to the term "small heart" as descriptive of a hitherto nondescript type, have served to

confuse the picture hitherto and prevent the recognition of a clinically interesting and important grouping.

Succussion is often present and low in site. The displacement is detected by the low position of the lesser curvature readily determined by inflation,* as is any coincident dilatation, or still more definitely by the X-ray.

DISORDERED MOTILITY.—Hypermotility has already been touched upon and is marked in many cases of gastric or duodenal ulcer, some of cholecystitis, hyperacidity, achylias, tapeworm and the ingestion of certain drugs, such as pilocarpin and strychnin.

Motor insufficiency of varying degrees may result from simple atony and the term is less properly applied to the stasis produced mechanically by pyloric stenosis, as in these cases there may be actual hypertrophy of the muscular layers and, for a time at least, partial compensation. Dilatation may result from the stasis of either myasthenia or stenosis.

With respect to the retention time, motility may be affected by many conditions other than mere muscular sufficiency or insufficiency: such are the presence of gastric or duodenal ulcer, occasionally in cholecystitis, the character and consistency of the food ingested, the position of the stomach itself, the activity or non-activity of the abdominal muscles, the amount of material in the great and small intestines, the attitude and activity of the individual, etc.

GASTRIC ATONY AND CHRONIC DILATATION.—The modern roentgenologist divides the stomach into the *corpus ventriculi* which sustains the food column, preparing it for passage into the intestine, and the antrum pyloricum which is chiefly concerned in projecting it into the duodenum.

Each of the first-named divisions may share the work of its fellow but its chief function is as above stated.

The corpus ventriculi may also be subdivided into *a fundus* occupied by gas and a *pars media*. The actual movement of food is concentric rather than a mere grinding or mixing process.

The "gas bubble."

As in the case of the ptotic heart it may be evident as a mere narrowing of reserve and hence of the symptomless performance of function. The author has repeatedly encountered gastroptotic patients in whom no gross roentgenoscopic signs of atony were demonstrable, but who experienced a most striking relief from or amelioration of symptoms when they were instructed to lie upon the right side for an hour or more after each meal.

Impaired tonus or "atony" actually means in large degree, though by no means wholly, impaired power of the *concentric contraction, i.e., perisystole.*

Peristalsis is the term applied to the vermicular waves of annular contraction which run in series from left to right. The two types of contraction are normally correlated, but so far structurally independent as to permit the one to be impaired without a parallel degree of involvement of the other.

The third important variety is that by virtue of which the pylorus relaxes and contracts under the operation of the hydrochloric acid reflex in such a

* Best employed when the patient is standing erect, inasmuch as posture greatly influences the degree of descent.

manner as to propel its contents into the duodenum in such fractional portions and at such intervals as best conserves the maximal effective digestive activity of the latter. Under normal conditions the duodenum inhibits the relaxation of the pyloric sphincter until the acid chyme previously received is completely neutralized.

The fluoroscope has demonstrated the fact that the structurally and physiologically normal stomach, when receiving a bismuth meal, so grasps it through the power of concentric contraction (tonus) inherent chiefly in its fundus and central portion as to hold and compress it and more or less roughly presents the profile of a horn of plenty.

This is filled to about two-thirds or more of its capacity, the upper surface of the fundus content being capped by air.

If tonus be impaired *decidedly*, the column is illy maintained, the meal carries down the weakened inferior portion and finds its level far below the normal. *Atony may be marked without* any *demonstrable* serious involvement of peristalsis, and, moreover, with no serious interference with the efficient regulation of the pyloric sphincter so long as hydrochloric acid is present in adequate amount.

In extreme cases, of the asthenic type especially, peristalsis gradually may become involved and delayed emptying results, but true stasis and high grades of food stagnation such as characterize pyloric stenosis, do not occur in cases of simple atony. Even in high degrees the stomach is empty of all food the morning after an evening's "retention meal."

Any attempt to sharply classify the various conditions associated with muscular insufficiency or actual dilatation of the stomach leads to much confusion and it is better to consider the stomach and its musculature exactly as one would the heart.

Weak musculature resulting in impaired motor activity with either decrease or increase of demonstrable peristalsis, and relaxation of the muscular walls constitutes true atony of the stomach and, as in the case of a chronically weak heart, leads to lessened effective functional activity.

In certain instances a misleadingly active peristalsis occurs from a heightened irritability which results probably from sub-nutrition and overstrain due to impaired muscle tonus.

If an actual obstruction exists at the outlet, the stomach and the heart act alike in that they try to overcome the mechanical obstacle and the *initial* or gradually induced dilatation by increased action and hypertrophy of the muscle, or, if this is not possible, to undergo passive dilatation with consequent stasis.

The parallel is sufficiently exact and helps to clarify this complex subject which is involved in a maze of contradictory opinions even at the present time.

Disregarding a suppositious megalogastria we have first to deal with gastric atony or myasthenia, *i.e.*, decreased concentric contractility due to selective muscular insufficiency.

This may exist without marked or permanent dilatation in any degree so long as peristalsis remains unaffected, but persistence of atony plus im-

paired peristalsis to an extent involving prolonged retention of the stomach contents tends to produce a secondary dilatation (ectasia) varying greatly, but seldom attaining the extreme form encountered in cases of actual mechanical obstruction.

Hence, as stated in the opening paragraphs, we find that in atony considerable degrees of dilatation are possible without the extreme manifestations of gastric stasis and fermentation encountered in pyloric stenosis. *In this last condition*, as in stenosis of the aortic orific of the heart, unless myasthenia or atony preëxist, there is primarily an hypertrophy of the muscular coats which may for a long time prevent extreme dilatation or marked stasis. Ultimately, however, either because the stenosis becomes more extreme or the muscular weakness predominates over hypertrophy, marked dilatation occurs with an increased degree of myasthenia and resulting stagnation.

Stasis

Ectasia

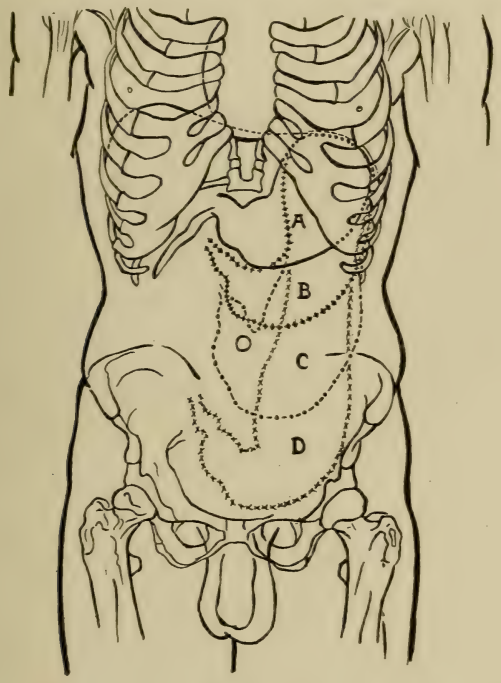


FIG. 448.—The normal and the ptotic stomach. A, the "steerhorn" type; B, the "fishhook" type; C and D, two of the several grades of gastroptosis. According to the author's observation a grade of descent (sagging) lying between C and D is extremely common. (*After Holzknecht.*)

Gastroptosis.—Any case of dilatation, if marked, *may* be associated with varying degrees of displacement (*ectasia with gastroptosis*), but marked ptosis may and usually does exist without dilatation (*gastroptosis proper*), is one of the commonest of clinical conditions and usually is wholly symptomless save during periods of depressed general nutrition and diminished myocardial tonus. Having the preceding factors in mind, one may proceed to discuss the conditions under three chief heads.

Chronic or acute.

Gross Atony.—(*Myasthenia, "Relative," "Motor or Facultative Insufficiency"*).—This covers the non-obstructive form and may be primary, secondary, acute, or chronic and associated with mere temporary dilatation, abnormal distensibility, or a true ectasia. (See Section on Roentgenography.)

FIG. 449.—Decided gastroptosis with impaired tonus. Patient decidedly asthenic and complaining of decided gastric discomfort of the asthenic-dyspeptic type. Lower pole of stomach just above white strip at right.

Fatal cases.

In rare instances acute, fatal dilatation occurs, a condition of special importance in connection with heart disease, arteriosclerosis, the acute infections and major operations, particularly of the abdominal type.

Chronic dilatation of an extreme form may be seen in the employees of breweries or others who are gross eaters and heavy beer drinkers. In its lesser degrees it is not uncommon, especially in association with the visceroptosis of congenital asthenia.

Etiology.—No disease is richer in *asserted* etiologic factors, chief among which are *hereditary structural defect and functional inadequacy or instability and chronic or recurrent nutritional depression*, often resulting from unconscious voluntary chronic starvation, from nervous shock, grief, worry, mental or physical overstrain, or exhausting diseases, *chronic cryptogenetic sepsis and*

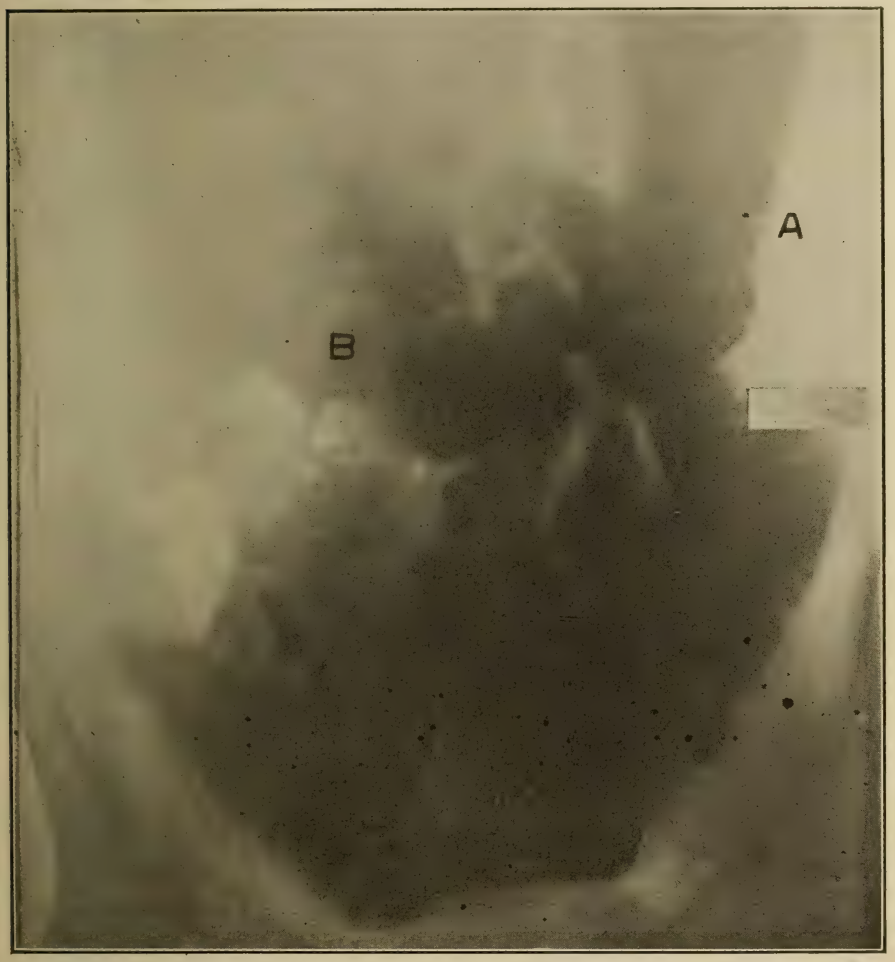


FIG. 450.—Same patient (Fig. 380) showing the effect of his improved condition. Position of lower pole indicated by upper level of white strip. Tonus manifestly greatly improved. Peristalsis active in both cases. Emptying time somewhat *shortened*, suspected duodenal ulcer. Diagnosis not confirmed by surgeon.

certain acute prostrating infections, and obscure toxemias. A host of other causes are given usually, but probably are, for the most part, of slight importance.

Inefficient musculature.

The more one studies the manifestation of congenital asthenia and its visceroptoses in both the complete and the still more interesting imperfect forms, the better he realizes the dominant part played by it in simple atony.

The Symptoms.—Subjectively the gastric symptoms are those described

Appetite.

under "nervous dyspepsia." In the early stages at least, appetite is often well maintained, though in others and especially in cases of long standing, the slightest quantities of food may give the sensation of repletion and distention.

Vomiting unusual.

Vomiting is relatively infrequent, irregular or wholly absent in marked contrast to stenotic atony and ectasia and there is usually more or less obstinate constipation.

It is commonly stated that these patients are intolerant of food and that its ingestion immediately excites distress, pyrosis, eructation, heartburn and the like.

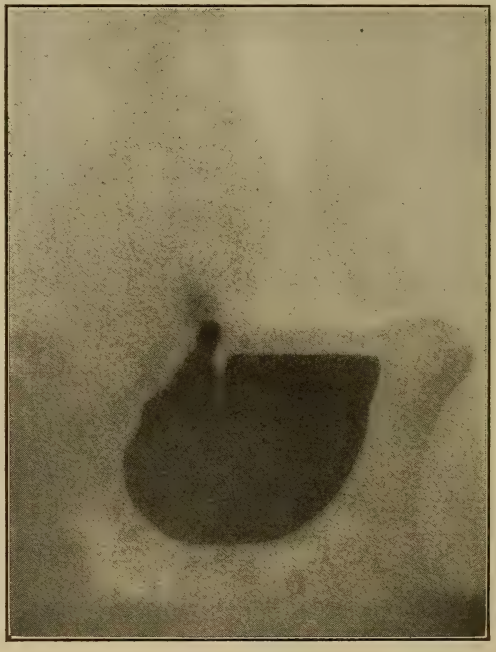


Fig. 451.—Atony of the demonstrable type. Note failure to grasp the bismuth meal in a normal manner. The great elongation of the stomach represents usually the congenital deficiency of tonus which is largely symptomless so long as a certain nutritional level is maintained. Ectasia is manifest in this case.

This is certainly not to be taken as wholly expressive of the behavior of cases or applying to food in all forms and quantities.

Liquid food is often badly borne, whereas light solids and semisolids, especially if taken six rather than three times a day in less quantities may give great relief and bring about the desired improvement in nutrition.

Effect of rest.

The prompt effect of rest and isolation is often demonstrated in cases of this type, and large daily quantities of food given at even shorter intervals together with rest for the heart are well borne and rapidly overcome the basic subnutrition.

Objectively, one notes usually only the filling abnormality (not "defect" in the roentgenologic sense) heretofore described as determinable by the X-ray to which may be superadded oftentimes gastric enlargement. (Fig. 451.)

Gastroptosis of some degree is an almost invariable finding according to the author's personal experience.

The milder cases may show merely excessive distensibility, as revealed by

FIG. 452.—Decided congenital defect with no *demonstrable* atony of the concentric muscle fibres. Such a patient may be free from symptoms or profoundly dyspeptic, the question being largely one of a sustained adequate level of nutrition.

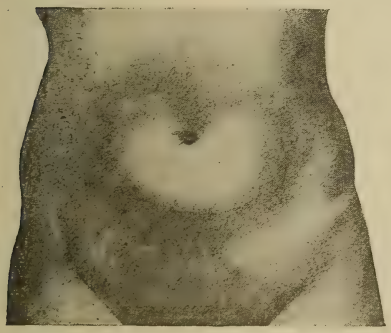


FIG. 453.—Dilated and displaced stomach (inflated) (crescentic form). Outline—personal observation. (*Figure after Riegel.*)

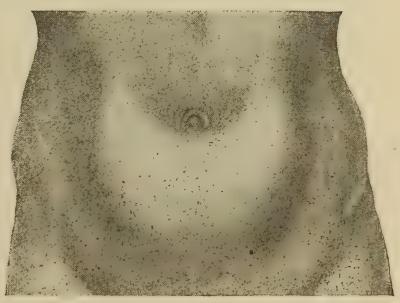


FIG. 454.—Simple gastroptosis. Personal observation. Stomach tensely inflated. (*Figure after Riegel.*)

inflation. The test of low level of dulness resulting from the ingestion of a pint of water sometimes advocated proves only a gastroptosis, not a severe atony.

Contrary to the findings in many stenotic cases excessive or reversed peristalsis is usually lacking.

Succussion is sometimes apparent even six or seven hours after a meal and is abnormally low in position from associated gastroptosis.

Residual food may or may not be found seven hours or more after the Riegel dinner, *but not if the meal has been retained overnight.*

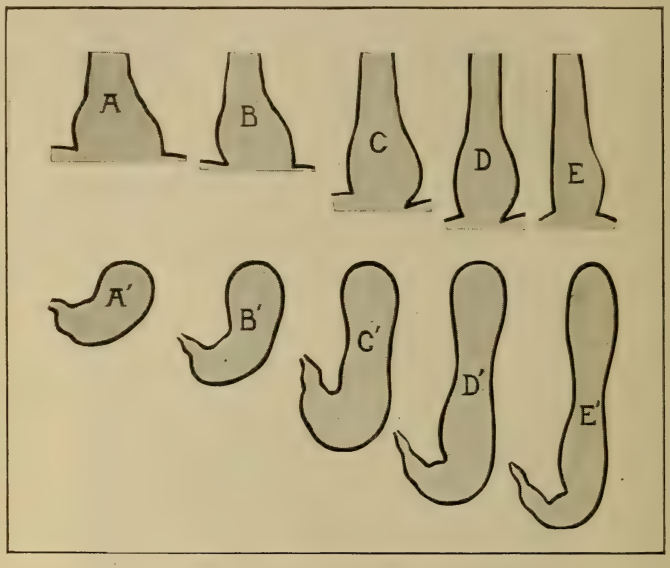


FIG. 455.

A = Heart of robust athletic male (ideal normal).
A′ = The high transverse stomach (ideal normal) "steer-horn" type.
B = Feminine type of heart. Commonest of types. Frequent in males.
B′ = Feminine type of stomach; normal for both sexes. The "fish-hook" stomach.
C = Slight cardioptosis; extremely common, more so in women.
C′ = Moderate or slight gastroptosis. Extremely common.
D = Centrally placed "drop" heart. In the author's opinion possibly expressing very slight enlargement of the "drop" heart.
E = Laterally placed "drop" heart. Right border indeterminate. Both D and E very common, especially in women.
D′ and E′ = Corresponding grades of gastroptosis.

The curious parallelism or concurrence of *cardioptosis* ("drop" heart) and *gastroptosis* (dropped-stomach). So generally does the author find this concurrence in his routine radiography as to make him feel that the presence of the one is almost proof positive of the presence of the other stigma of chronic congenital asthenia. Within variable limits the grade of the gastroptosis and that of cardioptosis correspond. Apparently both are affected by transient or prolonged states of toxemia or subnutrition as regards outline, though gastric atony is also readily induced. The absence of proper support from below as a factor in the intensification of "drop" heart peculiarities is strongly suggested. Atony, actual or relative, is common to both, but obviously must be symptomatically expressed more frequently and decidedly by the heart. The fluoroscopic appearance of a heart of the D and E types is that of a pulsating bag. A, B, C, D and E are drawn direct from *x*-ray negatives. A′, B′, C′, D′ and E′ represent the concurrent types of stomach determined by the radiogram. Heart A measured 13 cm. total transverse diameter; heart B, 11.5 cm.; heart C, 10 cm.; heart D, 8.3 cm.; and heart E, 7.5 cm., allowing half the width of the sternum as representing the concealed right border. All heart radiographs were taken in the inspiratory phase, as this best determines variations in form and tonus according to the author's experience. Even the expiratory phase of quiet breathing markedly obscures the characteristics of the plastic heart and especially of its modifications. The difference is comparable to that observable in gastroptosis as between the erect posture and recumbency. In the latter posture the ptosis may be almost or quite inappreciable.

In *forced expiration* the heart outline is so greatly modified as to increase its transverse diameter by two or more cm. and wholly conceal defects of conformation and tonus.

Hydrochloric acid values are normal in about 50 per cent. of the cases, the remainder being about equally divided between hyper- and hypochlorhydria.

These figures do not include those of primary or secondary achylias with or without stenosis.

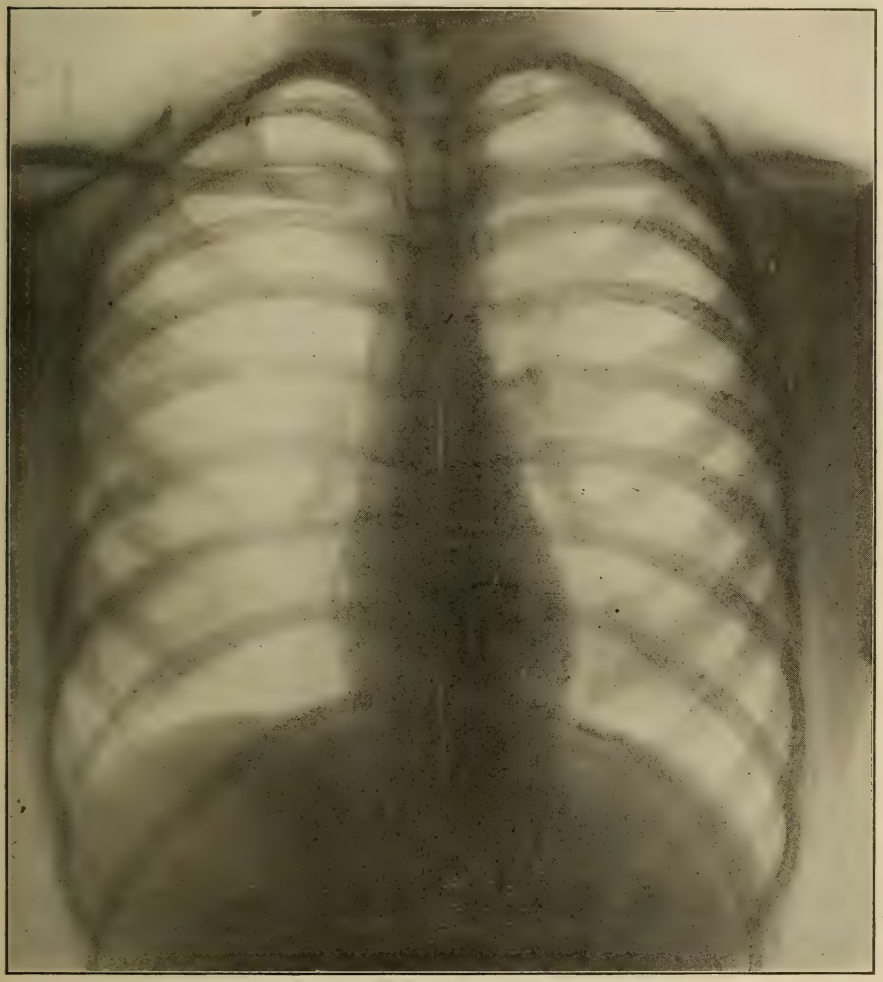


FIG. 456.—Cardioptosis.—Slightly modified "drop" heart, centrally placed. Minor symptoms of cardiac inadequacy present. Total transverse diameter 8.5 cm. The ptotic but non-atonic stomach is shown in Fig. 388.

The stomach contents may show a relatively slight degree of stasis or none at all as indicated by fermentation, though the stomach findings vary greatly and are not in themselves distinctive.

ASTHENIC DYSPEPSIA.—"Nervous Dyspepsia" (*Combined Gastric Neuroses"*).—*These terms cover certain forms of gastric disturbance prolific in subjective symptoms and barren in objective signs other than those related to an*

associated visceroptosis which is manifest in varying degrees in a large majority of the cases.

It is commonly associated with congenital or acquired asthenia, a history of nervous overstrain, or cardiac shock, grief or worry, or it may be due to reflex causes such as chronic appendicitis and pelvic disease. Frank psychasthenia or hysteria may be present.

FIG. 457.—Stomach of the patient whose heart is shown in Fig. 387. Decided gastroptosis without marked atony.

In actual practice it represents in a considerable number of instances the digestive or pseudo-dyspeptic manifestations of minor cardiac insufficiencies, the earlier toxemic expressions of chronic interstitial nephritis or one of the many obscure effects of chronic focal infection of the cryptogenetic type.

There are but few organic diseases that lack dyspeptic subjective symptoms and up to the present time nervous dyspepsia has been nothing more than a more or less kaleidoscopic grouping of symptom fragments.

Fundamental points.

It is essentially a mere syndrome and in itself proves no serious derangement of gastric function or actual disease of the stomach.

Chronic Congenital Asthenia.—*By far the most common underlying condition is that described by Berthold Stiller under this name.*

Such patients are delicately built, have a narrow, attenuated, relatively

FIG. 458. Heart corresponding to sub-type between "B" and "C" of Figure 384. Total transverse measurement 11.3 cm.

This man's heart is distinctly of the feminine type and possesses certain characteristics of the true ptotic or "drop" heart, yet it is perfectly competent and represents a normal in the same sense that we apply that term to the high-lying fishhook stomach.

It is certainly the commoner type of adult heart as seen in healthy, but slenderly-built males and in seventy to eighty per cent. of females.

In this instance gastric atony and ptosis were present because of a sharp nutritional deficit, but the only evidence of heart participation lay in the primary complaint of excessive fatigue following accustomed exercise, this being followed by the development of decided gastrointestinal discomfort.

On the mother's side both male and female members are of the visceroptotic type. The hilus and lung shadows are interesting and suggestive.

or actually atonic, readily displaceable, low-lying heart and an unstable functional and nutritional balance. They tend more or less constantly toward subnutrition; as a group, bear acute ailments badly, recover from them slowly, and are possessed of a highly irritable and labile nervous system.

Their visceroptoses are manifest chiefly in the low position, and often the atonicity of the the stomach, and the instability of muscle tonus and functional potency on the part of both heart and stomach, during periods of

FIG. 459.—Stomach of patient (Fig. 458) showing marked ptosis and atony. All symptoms have disappeared under a gain of weight of fifteen pounds. No medicinal treatment required.

nutritional depression. As might be expected, a large proportion reacts positively to a tuberculin test even though for the greater part wholly free from *active* lesions.

The stomach in such cases may show descent in any one of three grades below the "ideal" and the heart and stomach for the most part run measurably parallel in their degree of departure from the normal in *outline.*

The Tongue.—In asthenic or nervous dyspepsia the tongue is usually clean or lightly furred and may show some flabby enlargement and lateral indentations from the teeth in the long standing inveterate cases.

Hunger Pain.—When the stomach is empty the patient may complain of faintness, a "gone" sensation in the epigastrium, actual epigastric pain, headache, or a sense of cerebral pressure or dulness. Food usually brings marked relief though some patients experience marked subjective weakness and palpitation after taking it.

Protean Symptomatology.—In the kaleidoscopic symptomatic pictures encountered in this disease everything and anything in the way of subjective dyspeptic symptoms may be encountered. There may be troublesome distention, persistent eructation and, oftentimes, peristaltic unrest associated with pyrosis and irritating borborygmi. Dizziness, epigastric pulsation and very rarely the so-called "*globus hystericus*" may be encountered. The subjective sensation last named is, too often, actually a misinterpreted substernal expression of an "insufficient" heart.

Suggestive symptoms.

Globus hystericus.

The gastric contents are usually normal but may show hyperacidity, hypoacidity or a temporary or persistent anacidity. Heterochylia is very common in these cases and malnutrition may or may not be marked.

Heterochylia.

Motility may be excessive or diminished but is usually but slightly impaired though, as stated, gastroptosis of varying degree is almost invariably encountered and may be present in a most extreme form.

Persistence of symptoms is due usually to chronic fatigue and a state of relative starvation chargeable to unduly restricted or monotonous diet, lack of needed rest (mental and physical), and proper recreation and diversion. A pernicious and persistent form of introspection is commonly encountered.

Introspection.

Many of these cases require absolute rest, isolation, a gradually increased and ultimately generous diet, absolute control by a specially trained nurse, the oversight of a competent physician, a modicum of common sense, and renewed will power as a leaven.

ACUTE ATONIC DILATATION.—Terrifying epigastric oppression, violent dry retching, or more rarely, slightly productive vomiting, actual pain, and belching followed by persistent gas distention, cold extremities and a weak and rapid pulse, mark an ailment less often recognized than it should be as a cause of sudden death, yet overfrequently assumed to be responsible for an abrupt exitus due to unsuspected cardiovascular disease.

A cause of sudden death.

*The mysterious sudden death at home, on the street or in hotels, and on railways, which is so often attributed to acute atonic dilatation, is doubtless in nearly every instance due to previously unrecognized disease of the heart, arteries or kidneys.**

The stomach may so dilate as to entirely fill the abdominal cavity and the duodenum may or may not participate.

Etiology.—*The chief cause probably is sepsis, and by far the greater number follow abdominal operations.*

Various causes.

* Few years pass without such a death in some patient of the author's, whose condition is unknown to those about him at the time of his abrupt *exitus*. The primary diagnosis on such occasions is almost invariably acute indigestion or acute dilatation, supported by the statement that the patient had shortly before complained of epigastric distress, vertigo, etc.

It is doubtless true that any acute infection with profound toxemia may cause it in rare instances, and this is especially true of pneumonia.

The assumption that intestinal kinks or mesenteric constriction cause it, seems to be unsupported by adequate evidence.

Mechanical pressure, such as is exercised by a tight plaster cast which produces dorsal counter-pressure as well as anterior pressure, is said to have produced it in a few instances.

Toxemic paralytic dilatation plus abdominal traumatism, and such direct mechanical dilatation as is seen in certain foolish individuals who are eager to back by physical performance, bets on their gastric capacity and tolerance would seem to cover the etiologic factors.

As a cause of death in ambulant patients, or apart from the causes given, it is probably very rare.

Usually frank.

Symptoms of Acute Dilatation.—*The chief and only symptoms of importance are: (a) decided enlargement of the stomach; (b) the repeated and persistently recurring vomiting of bile-stained fluid; (c) the large quantities of such fluid* Stasis signs. *recoverable by the introduction of the stomach tube, and the relief afforded by this measure reinforced by gastric lavage and maintained drainage.*

Distention.

It is often evident that the vomiting represents little more than overflow and indeed the vomitus may come into the mouth without any visible effort in some instances. Decided "straining" is the exception rather than the rule. Some patients do not vomit at all and cessation of vomiting may be ominous rather than encouraging, unless the stomach tube no longer siphons fluid and the patient is manifestly improved in other respects.

The mortality was formerly over 70 per cent., but is much less under modern tube drainage and postural procedure.

POST-STENOTIC MOTOR INSUFFICIENCY.—*In this condition actual obstruction, not mere loss of muscle tone, is the primary factor; indeed, actual muscle hypertrophy may occur in a dilated stomach, as in the case of the heart with an obstructed outlet.*

Etiology.—Whatever produces obstruction of the pylorus or duodenum may cause ultimate gastric dilatation and insufficiency. It may be due to adhesions following operation, pyloric tumors, tubercular or luetic infiltration, congenital stenosis,* or the scars of healed ulcers.

The chief and most frequent causes are malignant growths, pyloric spasm and obstructive scars or deformities. Even excessively tight corsets or plaster jackets may produce chronic obstruction and dilatation through pressure effects.

SYMPTOMS.—The symptoms of simple nervous dyspepsia or myasthenia predominate, but pain may be present and be severe, paroxysmal and attended by violent visible peristalsis or palpable contractions or, on the other hand, be wholly absent or replaced by, or associated with, constant or remittent extreme epigastric oppression with or without tenderness.

* Congenital stenosis is far commoner than has been supposed, three cases (Drs. J. U. Goodrich, P. A. Hoff and W. Ramsey) having been brought to the author's attention within a period of ninety days, all confirmed by autopsy.

Objective Symptoms are Readily Elicited.—*The stomach is often visibly distended or, if inflated, appears in characteristic but enlarged outline with or without displacement* (*gastroptosis*) and is then readily palpated. Increased resistance may be noted as well as "rhythmically intermittent rigidity" ("magensteifung"). Peristalsis may or may not be visible but is marked during painful crises, if such are present, or shortly after a meal.

As in the case of heart, long persistence of stenosis may cause loss of muscle tonus and diminution of the palpable contractions or visible peristalsis. The degree of dilatation may be determined roughly by inflation combined with auscultatory percussion and palpation of the stomach tube *in situ*,

FIG. 460.—Post-stenotic ectasia due to scar of old gastric ulcer. (*After von Bergman.*)

fluoroscopy, the gyromele, etc. *Changes of note corresponding to changes of posture may be evident as in pulmonary cavities if large quantities of fluid are present. Succussion is of course constant in marked ectasia.*

Residual contents in marked quantity, varying with the degree of stenosis and ectasia, are found in the fasting stomach even though the "retention" dinner be taken the preceding evening after lavage, and the contents are foul-smelling. The odor may be that of fermenting cabbage or garlic, or that of hydrogen sulphide, perhaps aromatic like fermenting wine, or inexpressibly vile, according to the nature and cause of the stasis and its accompanying stagnation and fermentation. (See "Retention Meals" and "Vomitus.")

Vomiting is one of the most constant signs of obstructive ectasia in contradistinction to mere atony.

The vomitus is, as above described, frothy, of excessive quantity and contains remnants of several previous meals, especially seeds and the like.

It frequently separates into three layers on standing, and is bitter and foul to the taste.

The total acidity is increased by fermentation acids and the microscope shows in benign cases the yeasts and sarcinæ; in cancer (90 per cent. of obstructive cases) the Boas-Oppler bacilli. The fatty acids may be present by chemical and microscopic tests, the urine is scant, concentrated if vomiting be profuse, and if gastric acidity be high is often alkaline in reaction.

The persistent presence of bile *in large quantity* invariably points to duodenal stenosis.

In stenotic cases the symptoms may come and go over long periods because of the changes in calibration due to temporary local congestion of the mucous membrane.

Such remissions or intermissions are more frequently seen in benign than in malignant cases, but may occur in either.

Complications.—Acute auto-intoxication occasionally occurs and even coma may result. Extreme distention may not only produce dyspepsia,

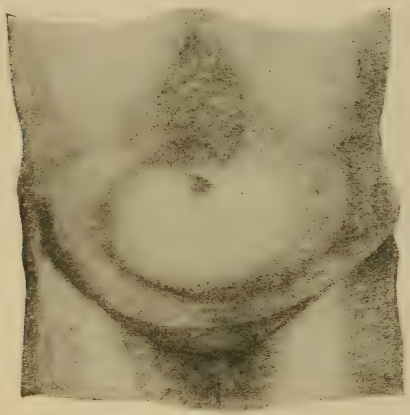


FIG. 461.—Dilated stomach. Post-stenotic ectasia. Slightly modified to conform to personal observations. (*After Riegel.*)

FIG. 462.—Dilated stomach (inflated). Combined displacement and dilatation of lesser degree. (*After Riegel.*)

bradycardia, or palpitation, but may seriously endanger a weak heart. *Gastro-succorrhea* is common and, finally, that rare and interesting condition known as *gastric tetany* may supervene, during the passage of a stomach tube, vomiting, or even percussion.

Differentiation.—The clinical distinction between these various forms rests primarily upon the evidence of delayed emptying of the stomach and the presence and degree of stasis, as indicated by persistent retention of food residue and fermentative changes.

Furthermore, in actual mechanical obstruction, the recurrent vomiting of strikingly excessive amounts of food and liquid showing persisting and often *ancient* food residue, marked visible or palpable peristalsis and usually the presence of a pyloric tumor assist the diagnosis.

In stenotic insufficiency, moreover, we are ordinarily dealing with carcinoma, more rarely with pyloric ulcer or its scars, and the gastric contents will vary somewhat characteristically with the causative factors.

As between the benign and malignant cases great difficulties may arise in diagnosis, but in general it may be said that, while marked stasis characterizes both, the presence of large numbers of the Boas-Oppler bacilli, the cachexia, profound anemia and the entire absence of hydrochloric acid, free and combined, so nearly constant in cancer, are absent or present in less degree in benign stenosis, in which, moreover, the sarcinæ are usually present in large numbers. (See "Roentgenography.")

HOUR-GLASS CONTRACTION (*Bilocular Stomach*).—This deformity, if organic and not congenital, results usually from a penetrating ulcer, the healing of a large annular ulcer, more rarely from adhesions or scirrhus. The stomach is divided into two lateral segments and the communicating opening may be relatively large or very small. The symptoms vary with the cause and may closely simulate pyloric stenosis if the opening be greatly contracted.

Diagnosis.—*Inflation alone will rarely make the diagnosis, which is easily established by fluoroscopy. The following signs are of some value:*

1. *The return of only the major portion of a liter of water introduced by the tube.*

2. *In distention by air and liquid, if the opening is narrow, (a) the bulging or the dull percussion note may at first be only in the cardial pouch and later be apparent in the pyloric portion. (b) The water or gas may be heard or felt to pass through the narrow orifice, especially if pressure be applied. (d) If all recoverable water be withdrawn succussion sometimes may be efficient and only over the pyloric loculus.* (See "Roentgenography.")

Occasionally adhesions may produce a false hour-glass stomach when the patient is erect which disappears in recumbency.

DISEASES OF THE ESOPHAGUS

THE CHIEF ORGANIC ESOPHAGEAL LESIONS.—Varices.—Some very serious, dramatic and even lethal hemorrhages may arise from the rupture of varices of the esophageal veins, usually due to an obligatory collateral circulation associated with portal or inferior vena caval obstruction.

The usual point of rupture is at the inferior segment of the esophagus and hepatic cirrhosis of the Laennec, Banti, or luetic type is the most common basic factor.

Acute Esophagitis.—This occasionally occurs as a result of the irritation of foreign bodies, the action of corrosive poisons or hot liquids, or in connection with diphtheria or other acute infections. It may also be secondary to malignant disease or an extension of a catarrhal process in the throat. Its symptoms are chiefly dull substernal pain and dysphagia with or without spasm and are described further on.

ESOPHAGEAL STRICTURES.—The chief points of normal esophageal narrowing are: *First.—The segment opposite the cricoid cartilage*, normally about 14–15 cm. from the incisor teeth.

Second.—The point opposite the bifurcation of the trachea and at the level of the *crossing of the aorta*, normally 24 to 26 cm. from the incisor teeth.

Third.—At the "hiatus" where the esophagus passes through the diaphragm, about 40 cm. from the incisor teeth.

These are the points at which pathologic constrictions most frequently occur, whether due to the contracting scar tissue following corrosive acid burns, ulcerations, or malignant disease.

Furthermore they represent the points of election for the lodgment of foreign bodies, for instrumental injury and, in the case of the median constriction point, for compression by *aortic aneurysms* involving the transverse portion of the arch. These occasionally constitute a genuine source of danger in the use of dilating esophageal bougies, though if the condition is kept in mind as a possibility, error should seldom occur.

In the absence of paralysis, inability to swallow solid or liquid food demands immediate recourse to the esophagoscope or esophageal bougie. True stricture is usually due to cicatricial contraction following the ingestion of corrosive poisons, syphilis, or *malignant growth* and is rarely congenital. Obstruction from the pressure of *aneurysm* or other tumors, or even, it is said, of massive pericardial effusion may occur.

A bougie should never be passed without a careful preliminary examination for possible aneurysm or necrotic processes and it should be remembered that nine out of ten strictures in adults are due to malignant growths.

Strictures may be single or multiple, sharply defined or involving the whole tube, but are usually either near the cardia or just below the pharynx. No definite information is obtainable without the use of the esophagoscope, X-ray, or bougie, but as a matter of interest the esophageal bruit may be auscultated at the left of the spine. It should be heard at the left of the seventh dorsal vertebra within seven seconds of the time that a mouthful of water is swallowed, and a delay suggests obstruction. The most convenient bougies are those with detachable heads of varying size and an elastic steel or whalebone stem. As in the passage of a urethral sound and for the same reasons, the larger sizes should be first tried and *forcible maneuvers avoided.* The patient should be asked to breathe deeply with the head thrown far back and the spine straight, and, *without introducing the finger,* the bougie is passed gently, firmly and steadily along down the pharyngeal wall disregarding the slight obstruction encountered at the cricoid level. Perforation during the procedure is a rare but deplorable accident.

Symptoms.—Food regurgitation and dysphagia, the latter usually of a gradually but persistently progressive type and preceded by a period of hyperesthesia or recurring spasm are the basic and dominant symptoms, whether the lesion be due to past traumatism, or to malignant growth (70-80 per cent.), syphilis (only in the rarest instances), diverticula, or, the pressure of aneurysm, enlarged bronchial glands or the cold abscess of "Pott's disease."

The use of the fractional repeated ingestion of bismuth subcarbonate or the oxychloride suspended in gum acacia is proving of great assistance in the diagnosis of esophageal cases and the esophagoscope is invaluable.

ESOPHAGEAL DILATATIONS.—These may be due to the effect of recurrent spastic constriction upon the segment just above it, or, to extreme

atony of the esophagus. A form of diffuse dilatation of the lower segment, wholly due to recurrent spasm, is occasionally encountered and may reach enormous proportions.

It is obvious that aside from diverticula more or less symmetrical dilatation may occur above any points of pathologic constriction.

DIVERTICULUM.—This is a pouch-like dilatation on the order of saccular aneurysm, involving one or more of the coats of the esophageal wall.

Etiology.—Aside from the congenital diverticula, this ailment is rare in children, unusual in women and most frequent in men, a triad of facts relating to occurrence that usually strongly suggests a luetic origin. As a matter of fact, esophageal syphilis is an extreme rarity, according to statistics at present available.

Two distinct forms are encountered, viz., pressure diverticula and those due to traction.

The former are caused apparently by a congenital muscular weakness of the posterior wall of the superior esophageal segment or a yielding of the fibers of the anterior or lateral wall of the inferior segment.

"*Traction* diverticula" result from cicatricial contraction or the pull of adhesions formed between the esophagus and contiguous structures.

They are located usually on the anterior wall at or near the junction of the upper and mid-divisions.

For the greater part diverticula are unrecognized during life, though if large enough to retain food and produce stagnation or pressure a diagnosis may be made.

Symptoms.—The leading symptoms in the severer forms are: Substernal burning and pain, foul breath, sense of persistent esophageal irritation or distention, vomiting if the sac be large and, in extreme cases, pressure symptoms similar to those of aneurysm.

Obviously, evidences of abscess may exist in some instances not alone as a result of a purulent esophagitis, but, in the traction forms, from fistulous communication with the primary source of abscess formation.

Such pouches readily escape the sound, but may be detected both by the esophagoscope and the roentgen technic.

Bassler directs attention to the ease with which perforation may result, even from the passage of a stomach tube.

ACUTE ESOPHAGITIS.—Participation of the esophagus in the exanthems of acute infectious disease (scarlet fever, variola and even measles), the pharyngeal inflammation of diphtheria, or the acute toxemic states (typhoid and sepsis) are among the etiologic possibilities.

Extension of any existing inflammation in junctional or neighboring tissues and (most commonly) direct irritation from hot drinks or (less often) corrosive acids or alkalis (lye) and foreign bodies are adequate causes.

Symptoms.—In severe cases, *pain* persistent or only on swallowing, thirst and oftentimes the sensation of a foreign body in the tube, are present.

In milder cases the signs may be merely those of slight substernal distress and pain or decided discomfort on swallowing.

The latter group of symptoms also serve as the sole manifestations of *simple chronic* esophagitis, aside from any apparent or genuine neurosis such as may appear in any esophageal case and often precedes the full development of a serious organic lesion.

SYPHILIS.—This disease is so rare as to render its occasional occurrences clinical curiosities.

It is possible that a routine resort to the luetin and Wassermann tests might affect accepted etiology much as it has that of gastric syphilis, which is known now to be much more common, or, less rare, than previously was believed.

ESOPHAGEAL NEUROSES.—Hyperesthesia, paresthesia, esophagospasm and cardio-spasm are extremely common occurrences, but doubtless their frequency as supposedly idiopathic manifestations will diminish as esophagoscopy becomes more generally employed.

Lerche states that "a local neurosis is present in every case of anatomic lesion of the gullet at some time."

ESOPHAGEAL SPASM.—This condition is usually associated with functional nervous disease, such as hysteria, hypochondria, neurasthenia or, more rarely, epilepsy and chorea. It is also a symptom of hydrophobia and the diagnosis rests upon the recognition of the causative factors named and the passage of an esophageal bougie of full caliber which meets with only temporary obstruction.

CARCINOMA OF THE ESOPHAGUS.—This, the commonest cause of organic obstruction of the esophageal tube, differs in no essential respect from carcinoma elsewhere in its nature, mode of onset, constitutional symptoms, course, or termination.

Primary carcinoma is rare, most cases of obstruction being due to malignant disease extending upward from the cardia. The symptoms are those of obstruction associated with rapid and extreme emaciation and cachexia. Perforation into the mediastinum, aorta, pericardium or pulmonary and pleural structures may occur or the spine may be involved. In some of these cases the X-ray gives evidence of positive value.

It should always be kept in mind when dealing with organic stenoses or even motor or sensory disturbances apparently functional.

Symptoms.—Those symptoms specially appertaining to esophageal malignant involvement are the following:

(*a*) Persistent, steadily but slowly increasing dysphagia and demonstrable stenosis, the latter tending to become absolute.

(*b*) Regurgitation of food.

(*c*) Evidences of malignant ulceration as the disease advances.

(*d*) Substernal pressure or actual pain, seldom wholly absent. In late cases a lancinating type of pain may supervene and present nocturnal exacerbation.

(*e*) Significant enlargement of the supra- and infraclavicular glands may be demonstrable.

(*f*) Thirst is often a prominent symptom in advanced cases.

(*g*) Pressure symptoms from crowding of the many important mediastinal structures.

(*h*) Hemorrhage, often one of the earlier symptoms in the form of oozing.,

ORGANIC DISEASES OF THE STOMACH

ACUTE GASTRITIS (*Gastritis Glandularis, Acute Gastric Catarrh Phlegmonous Gastritis*).

Definition.—An acute (simple toxic, or phlegmonous) inflammation of the gastric mucosa.

Etiology.—Indiscretion in diet, nervous shock, toxic irritants such as ptomaines, arsenic, alcohol, mercuric chloride and potassium cyanide, or, if phlegmonous, pyemia, septicemia, the exanthemata, and rarely direct infection.

It is most frequent at the extremes of life and in those debilitated and dyspeptic. Excessively hot or cold drinks, overeating and imperfect mastication are important factors.

Morbid Anatomy.—The lesions vary from a simple catarrhal inflammation to necrosis and single or multiple abscesses.

Symptoms.—Mere subjective weight or fullness, anorexia, and a coated tongue with or without nausea, slight pain and general dyspeptic manifestations may be the only symptoms. Varying severity.

In the severe forms one finds headache, epigastric pain; nausea, vomiting and constipation or diarrhea, complete anorexia, excessive thirst, and a burning sensation which may amount to agonizing pain in the traumatic cases. In the toxic forms, prostration is extreme, the pulse feeble, the surface cold, the case tending to coma and death. Such cases may occur in myasthenic stenotic ectasia. Toxic form.

Stomach Contents.—Mucus is present in the vomitus, associated with blood and shreds of mucous membrane in the toxic forms and perhaps pus in the phlegmonous forms. Free hydrochloric acid is usually absent.

Prognosis.—Recovery is prompt save in exceptional severe toxic or phlegmonous cases.

CHRONIC GASTRIC CATARRH (*Chronic Gastritis*).—A chronic catarrhal inflammation of the gastric mucosa.

Etiology.—The disease may follow an acute gastritis, but is more frequently associated with a monotonous or improper dietary, chronic disease of the heart, liver, kidneys and lungs, gout, diabetes and the anemias, excessive smoking and drinking, the habitual use of highly spiced dishes, rapid and excessive eating and imperfect mastication.

It follows certain infectious fevers, as typhoid, cholera, yellow fever, and the exanthemata, ulcer, cancer or dilatation of the stomach itself or may be superimposed upon long-standing asthenic dyspepsia. It follows that certain cases complicating other conditions are hardly distinguishable. Obscure cases.

Symptoms.—Among the commoner symptoms, are: flatulence, belching,

morning-malaise, nausea and perhaps vomiting, pyrosis, foul taste in the mouth, vertigo, palpitation, dyspnea, cough and a poor or variable appetite often associated with a sense of repletion after even a light meal. Morning vomiting occurs especially in the chronic gastritis associated with hepatic cirrhosis ("drunkards' catarrh"), and morning regurgitations before or after breakfast are even more common.

Constipation, in some cases alternating with diarrhea, is the rule; there are usually evidences of a poor circulation, the tongue is coated and marginally indented by the teeth, and nervous irritability is common. *Marked loss of weight is seldom seen save in distinctly secondary cases, where it is usually due to the primary cause.*

The disease usually develops insidiously and progresses very slowly.

Mucus

Stomach Contents.—*In advanced cases* free HCl is much diminished or absent, the total acidity is below normal, the total stomach content usually increased, *mucus is abundant even in the empty stomach, and the digestive ferments are diminished.*

Hypertrophic Chronic Gastritis.—This is secondary to the simple form and represents a sclerotic process which results in marked destruction of the secretory glands.

Misleading signs.

This may terminate in a general infiltration which greatly reduces the size and capacity of the stomach. It may so narrow the pyloric outlet as to simulate malignant stenosis, especially as an achylia or decided hypochlorhydria may be evident. Retention is marked in duration, but the quantity of content is small. Even after the test meal, it rarely exceeds 30 c.c.

A dubious condition.

Acid Gastritis.—This shows a remarkable conjunction of hyperchlorhydria and excessive amounts of mucus in the removed test meal.

We do not yet know its true place or the justice of its claim to be considered a clinical entity, and a primary surgical source should be sought always.

Differential Diagnosis.—All differential diagnosis in disease of the stomach is subject to limitations and exceptions, it being often impossible to draw hard and fast lines, and chronic gastritis must, like all others, be encountered in its early and its complicating forms, lacking in the one case its most characteristic symptoms and in the other blending with or overshadowed by the primary disease.

A chronic gastritis lacks the severe pain, extreme localized tenderness, hematemesis and high HCl values of typical ulcer and usually the marked emaciation, excessive lactic acid formation, abundant Boas-Oppler bacilli, and tumor, so generally present in cancer.

Achylia gastrica lacks the mucus so characteristic of the usual form of gastritis, and in the neuroses, the asthenic psychasthenic or hysteric symptoms, asthenic conformation, more trifling or inconstant disturbances of gastric secretion and the absence of an excess of mucus usually differentiate it.

Prognosis.—This varies according to the cause. Many cases may be entirely cured; others, never.

GASTRIC AND DUODENAL (POST-PYLORIC) ULCER

GASTRIC ULCER.—Definition.—An ulcer of the gastric mucous membrane, usually single but often multiple and tending either to an acute course followed by complete or partial healing, extreme chronicity, or, to perforation, either prompt or long delayed.

As to ulcers in close proximity to the pyloric ring, whether proximal or distal, no distinct line can or need be drawn. The so-called duodenal ulcer is actually a post-pyloric gastric lesion, for in 95 per cent. of all such cases it occupies the "first portion of the duodenum," embryologically and anatomically an integral part of the stomach.

Etiology.—Aside from direct irritation or traumatism, toxemia, hyperacidity with deficient mucous secretion and devitalizing anemia, appear to be the most important associated conditions. The true cause is not known,* Still unknown.

FIG. 463.—Prepyloric acute ulcer with total spasm of the pyloric antrum and decided retention. Such a case would show high HCl values. (*After von Bergmann.*)

but the organisms contained in cryptogenetic septic foci may prove the important antecedent factors or even the determining causes. All of the older theories are imperfect and unconvincing.

Both anemia and toxemia are factors in delayed healing.

Sex.—About 60 per cent. of acute gastric ulcers occur in females. The chronic forms on the other hand show decided male predominance.

Age.—It occurs most commonly between the ages of twenty to forty, least often, yet occasionally, in babies,† young children, and elderly persons, and there is probably little or no hereditary element.

Acute ulceration is encountered chiefly between the ages of 20 and 30. About 70 per cent. of all ulcers are recognized between the ages of 30 and 50. As between the age periods of 50 to 60 and 60 to 70 there is little difference in the ratio of incidence, which is about one fifth of that of the fifth decade.

* Recent investigations indicate the existence in the normal stomach of protective anti-enzymes, but under what conditions their activity fails is as yet unknown.

† Doubtless the post-pyloric ulcer is a cause of "*melena neonatorum.*"

Statistics faulty.

Certain Predisposing Factors.—Direct injury, as from blows, tight lacing, pressure, the ingestion of corrosive drugs and such occupations as lead to anemia, irregular or hasty eating and the use of improper food, are held to be important factors, and at least figure prominently in the statistical reports. No statistics are available so far as the author knows, which would bear actuarial criticism.

In one clinic, located in the midst of an agrarian population, the farmer may be found to furnish the highest percentage of ulcer cases; in another, the weaver; in yet another, domestics; the results depending chiefly upon the location of the clinic and the occupational trend of its patients.

FIG. 464.—Typical ulcer ("penetrating") with spasm of the segments. Note small projection toward spine on left side of constriction. (*After von Bergmann.*)

Varieties.—(*a*) *The "Acute" or acutely relapsing chronic ulcer of pronounced and definite symptomatology termed by Welch "gastralgic-dyspeptic."* (*b*) *The latent acute and chronic forms. Single or multiple ulcers may exist for years and ultimately perforate or spontaneously heal without localized symptoms: probably more than one-half of the cases are unrecognized clinically.* (*c*) *The acute perforating form. In this there is a rapid advance and perforation.* (*d*) *The acute hemorrhagic form. In this the symptoms are usually vague, of brief duration or may be entirely absent until profuse hemorrhage occurs. Other types occasionally described are relatively unimportant.*

PATHOLOGY AND MORBID ANATOMY.—The ulcers vary in size from mere erosion to necrotic areas measuring from 4 to 6 inches in diameter. The acute ulcers are ordinarily small (½ to ¾ inch) round and clean-cut, with

little or no induration of the base or the margins, though if chronic they may be irregular or sinuous with terraced sides and will show always marked infiltration or induration.

Acute Ulcer may heal and leave no scar or one so slight as easily to escape observation even at autopsy. Its depth may be merely that of erosion or extend to or through the serous coat. In most instances they are not palpable even when the stomach is exposed at operation and may readily be missed even though its inner surface is under the direct vision of the operator. On the other hand, it is the form most likely to present the typical symptoms of ulcer as set forth in our older text-books.

Chronic Ulcer is, as a rule, productive of quite opposite conditions. There are periods of activity, partial healing, sluggishness or latency and the tendency to infiltration is decided. They are usually palpable or even visible at operation, but most inconstant and variable in their clinical symptoms.

They are ordinarily single, but not infrequently (20 *per cent.*) *there are two, very rarely a score or more.* Usually single.

Location.—Acute ulcers occur with about the same frequency in the cardia, midzone and pyloric region. Chronic ulcer "straddles" the lesser curvature in from 80 to 85 per cent. of all of the cases not definitely post-pyloric (duodenal). They are relatively rare at the cardia and on the greater curvature. Rare at cardia.

Gastric ulcers of severity show a marked tendency to recur, usually within the old scar of a healed ulcer. In rare instances, true carcinoma may develop *its own primary ulcer** on an old simple ulcer base and many ulcers doubtless undergo during years, recurrent partial healing only to break down again *and may thus renew repeatedly the active symptoms of the disease.* Self limitation.

Thus is developed the significant but not distinctive history of recurrent, distinctly painful, dyspepsia, of greater or less degree and duration, with longer or shorter intervals of immunity.

This is a common feature of the histories of certain chronic types of gastric ulcer, but not at all uncommon in cases other than ulcer.

Healing.—*They heal by granulation and in cases of extensive ulcer may produce deformity, such as the hour-glass contraction, but more commonly a pyloric stenosis, two of the three conditions always justifying operation.*

As stated, toxemia, anemia, and, in some cases, hyperchlorhydria and hypersecretion are factors of some importance in the prevention of healing. Far more important is intermittent spasm or increased peristalsis. In this respect, as in relation to pain, the nearer the ulcer (gastric or duodenal) to the pyloric ring, the worse are the conditions.

Perforation.—Perforation may result in general peritonitis demanding surgical interference and tending to a fatal termination, or, adhesions may form, limiting the spread of the infection and saving life. Many such cases of self-limited perforation go unrecognized. The omentum, liver, gallbladder or pancreas may be involved, and adhesions thus formed complicate the Often overlooked.

* All primary carcinomata of the stomach begin as ulcers (Aschoff).

Misinterpretation.

clinical picture and lead to mistaken diagnosis. The author has observed in cases in which the gall-bladder was involved, symptoms exactly simulating gall-stones and leading to operation for their removal.

Obscure abscess.

The perforations of acute ulcer are usually direct and involve the general peritoneal cavity. Obscure abscesses may result in the chronic forms, subphrenic abscess and pyopneumothorax being sometimes observed and fistulous communication may involve tracts far distant from the original seat of trouble.

Fallacious figures.

The frequency of perforation as usually stated (13 to 18 per cent.) is certainly far too high, being necessarily based wholly upon cases yielding positive symptoms or coming to operation or autopsy, and, of course, omitting the clinically doubtful or larval cases which no doubt outnumber the others.*

In private cases amongst the "well-to-do" it is relatively rare. In public cases its frequency depends largely upon the type of hospital population, and the efficiency of the medical staff organization and personnel.

Frank cases.

ACUTE ULCER.—*The positive diagnosis of acute or subacute gastric ulcer still largely depends upon the concurrence of the old tetrad of symptoms, pain, localized tenderness, vomiting and hemorrhage, yet in most instances we meet cases which lack these franker symptoms wholly or in part.*†

Obscure cases.

A large proportion demand the careful balancing and analysis of a large number of factors which may be strongly suggestive individually, or only collectively significant, and fatal perforation or hemorrhage may occur in cases which have previously lacked the symptoms necessary to even a tentative diagnosis.

Placing the determining diagnostic factors of acute ulcer somewhat in the order of their clinical importance, we have: (*a*) *Hemorrhage.* (*b*) *Pain.* (*c*) *Localiced tenderness.* (*d*) *Vomiting.* (*e*) *Normal or but slightly changed hydrochloric acid values usually with lessened mucous secretion.* (*f*) *General dyspeptic symptoms.* (*g*) *Anemia.* (*h*) *Age.* (*i*) *Sex.* (*j*) *Occupation.*

Those fortunate enough to have at hand an expert roentgenologist may obtain much assistance. (See Roentgenology.)

Characteristics.

(a) **Pain.**—*The characteristics of the pain of gastric ulcer are:* (1st) *its intermittent and paroxysmal nature;* (2*d*) *its relation to the taking of special kinds of food;* (3*d*) *its time of occurrence*; (4*th*) *its periodicity in the individual.*

Other factors are of some importance in differential diagnosis and amongst these are: The tendency of the pain or distress to increase its intensity as the attack progresses, the immediate relief, complete or decided, afforded by involuntary or, more frequently, by induced vomiting. Any actual abdominal pain which occurs day after day during several weeks in the same region is almost proof positive of an organic lesion of the stomach, duodenum, appendix or gallbladder, though relaxed hernial openings or umbilical hernias must not be forgotten. If this pain observes a certain schedule with relation to its appearance in the particular individual under observation, varying only with the size of the meal taken and coming on more rapidly after light than after heavy meals, it is most

* Correctly stated the figures represent the frequency of perforation in cases clinically or pathologically demonstrated.

† The disease is often one of extremely insidious development.

suggestive of ulcer of the stomach or duodenum. If the appetite is well preserved the significance is yet greater, although the same elements may be present in certain functional disorders. Mere discomfort, a "faint feeling," a "gone sensation" a "sense of fulness" and the like must not be construed as ulcer symptoms, save in the presence of strong corroborative evidence. Flatulent distention, often due to air-swallowing, may accompany ulcer pain as well as pure asthenic dyspepsia or purely temporary and trivial digestive disturbances. As stated previously one must carefully analyze the statements of nervous patients and allow for their almost invariable tendency to exaggerate their symptoms.

Duration.

It is not as a rule immediate, but usually becomes severe and even cramp-like in from half an hour to two to three hours after eating the ordinary meal and continues until the stomach is emptied by vomiting, stomach washing,* or by the natural mechanisms. *Delayed pain (one, two or three hours) is far more common than was formerly believed and immediate pain the exception.* Immediate pain usually means a lesion of the esophagus rather than the cardia.

Causes.

The onset of the pain is in large measure dependent upon the location of the ulcer, and its intensity upon one or more of several factors, *i.e.*, peristalsis, spasm, acidity, character of the ingesta, irritability, congestion, inflammation and pyloric patency.

Referred pain.

The pain itself varies greatly in character and degree. *In its typical form* it is a boring, deep-seated pain, but is also described as burning, stabbing, cutting or cramp-like, quite frequently felt at the back, sometimes radiating upward to the region of the shoulder-blades.

This typical pain is often replaced by that of a duller character and a suggestive feature when present, is the relief or intensification of pain accompanying changes in attitude both in the presence and absence of adhesions.†

Ulcer pain may be increased in standing or walking, and lessened in the *dorsal recumbent position, but often intensified when the left lateral decubitus is assumed.* If markedly intensified by standing, walking, or deep breathing, it suggests peritoneal involvement with perigastritis or adhesions and, sometimes, an impending perforation.

The writer believes with Bourget, that whether an ulcer be gastric or duodenal, the nearer it is to the pyloric ring the more intense is the pain.‡

Both the character of the pain and the area of tenderness are relatively fixed and show little variation even when the attacks recur over long periods.

Helpful sign.

In some instances distress almost immediately following the ingestion of very hot, cold or acid substances is significant.

Persistent pain.

In certain cases, especially those of chronic ulcer, with hypersecretion, we find in addition, persistent pain of a duller character between meals and

* Hazardous in acute ulcer.

† This is by no means distinctive if the relief of pain follows the assumption of *dorsal or right lateral* recumbency *for these postures are peculiarly adapted to the relief of pain associated with visceroptotic dyspepsias.* Actual severe pain is relatively rare, however, in the ordinary asthenic dyspepsias.

‡ The same general rule may be applied to the occurrence of hyperacidity, though other factors are present in both instances.

especially at night, which is relieved by taking food (hunger pain). Much stress has been laid upon the occurrence of such pain in ulcer cases, but in a mild form it is one of the common symptoms observed in various forms of even functional dyspepsia and particularly that associated with visceroptosis.

Important points.

The location of the pain necessarily varies somewhat with the position of the ulcer and of the stomach, being as a rule, lower in tightly laced women and in cases of ectasia or gastroptosis than under ordinary conditions.

As five-sixths of the stomach lies to the left of the linea alba, and most ulcers are upon the lesser curvature, one usually finds the pain at, or more commonly just to the left of, the median line, between the ensiform and the umbilicus, usually near, or nearer, the former.

Should it be found that the seat of pain and tenderness shifts upward when the patient retracts the abdomen, assumes the recumbent posture (physiologic postural ascent of stomach) or downward in forced inspiration (respiratory movement) the evidence of an organic gastric lesion is thereby strengthened. Absence of such a shift is of little importance in negation.

Usually present.

Localized Tenderness.—*The importance of this sign lies in the fact that a distinctly circumscribed superficial or, less often, deep tenderness and defensive rigidity, often extreme, is sometimes present and usually corresponds closely to the area of maximal pain.*

Site.

It is commonly epigastric, but is a trifle to the left of the median line and when localized constrasts sharply with the tenderness of gastric hyperesthesia which is usually much more diffused. About one-third of the active cases show a sharply localized area of superficial or deep dorsal tenderness, often extreme, slightly to the left of the lower dorsal spines (3 to 4 cm.).

Spinal areas.

Rarely this is bilateral or on the right side alone and a second zone may be found occasionally at the level of the fourth and fifth dorsal spines. These areas seem to be associated with lesions at or near the pylorus, but they are far from constant or pathognomonic.

Hemorrhage.—*Visible hemorrhage* occurs in about one-half of recognized acute ulcers and, *if massive*, is always associated with a vomiting attack, usually subsequent to the pain crisis if this be present. It commonly occurs several hours after eating and often at night and is easily recognized as of gastric origin; but the smaller hemorrhages rapidly undergo such digestive changes as to closely simulate the small dark clots or coffee-ground particles of carcinoma. Furthermore, severe or even fatal hematemesis may be due to gastric erosion or hepatic cirrhosis as well as ulcer.

Visible blood.

In many instances of acute ulcer (15 to 20 per cent.) it constitutes the first *decided* symptom.

Occult blood.

The stools as well as the stomach contents should be carefully investigated for both visible and occult blood before declaring hemorrhage absent, and concurrent symptoms be carefully weighed before a frank hemorrhage is ascribed to ulceration. Under the restrictions and precautions previously laid down, the examination of the stool is far more important and productive of results than is that of the stomach contents. Blood may and does quickly pass from the latter viscus and furthermore both vomiting and the stomach

Examine the stools.

tube may cause slight hemorrhage. Enormous hemorrhage may be recovered from and but a small number die directly from it.

Bassler has suggested the use of an extremely simple device for aiding in the diagnosis of ulcer or erosion by the swallowing of a silk string carrying an ordinary shoe button at one extremity. This is swallowed by the patient; he is given sufficient "rope" and the free end is looped over his ear throughout one night. It is merely a simple and convenient application of the "string and bucket" method of Einhorn and, if the string be blood stained when recovered, serves to prove the presence of blood, but little more. A simple device.

Vomiting.—Though often coincident with a painful crisis, spontaneous vomiting with or without hemorrhage is far from constant and self-induced vomiting is more often seen. If present, vomiting usually occurs at the acme of the pain and relieves or greatly modifies it.

Persistently recurrent vomiting of a *definitely periodic* type is strongly suggestive of ulcer, either acute or chronic, but will usually be associated with other corroborative symptoms, viz., localized tenderness, visible hemorrhage in the acute, and less frequently in the chronic form, and in the latter especially with hypersecretion or hyperacidity. It frequently is the "gush" vomiting of hypersecretion. When less definitely periodic and persistent it suggests disease of the appendix or gallbladder. In a few instances the author has seen most misleading vomiting of this type in the crises of locomotor ataxia. The fact that occult or even visible blood may be present in this type of case without ulcer must be borne in mind.

Most of the cases of anorexia nervosa or mere cyclic vomiting observed by the writer have been quite without the usual delay of ulcer appendicitis or gallbladder disease with respect to food ingestion—the return usually being more like regurgitation.

In ulcer the delay may vary, but is likely to run between one and three hours with a regularity which is disturbed chiefly by the size and character of the meal taken. As possible causes, the crises of locomotor ataxia and the possible presence of incomplete hernia must not be forgotten.

Acidity.—In *acute* ulcer normal or high-normal values probably are the *rule;* hyperacidity uncommon, subacidity the exception except after hematemesis.

Hyperchlorhydria is a common condition without ulcer, so that even in relation to chronic lesions the symptom, while important, is only corroborative; nevertheless *decided hypo*-acidity goes far to exclude ulcer, acute or chronic, or, if constantly diminishing and accompanied by a clear antecedent ulcer history, suggests that rare development, *a carcinoma upon an old ulcer base.* (Decided subacidity is usually associated with complicating catarrhal gastritis or ectasia or an antecedent hemorrhage of considerable amount.)

The stomach tube must not be used if violent retching is thereby induced nor shortly after a hemorrhage. If vomiting is present in such cases a proper test meal sometimes may be taken and thus recovered, the interval being often sufficiently well observed.

The duodenal tube is well adapted to the recovery of stomach contents in acute ulcer cases in many instances.

Marked or excessively high HCl values associated with clean-cut ulcer symptoms undoubtedly represent usually an active, or relatively recent ulceration at or near the pylorus with prolonged retention of ingesta.

The relief of pain following the administration of warm solutions of sodium bicarbonate or milk of magnesia will often assist diagnosis slightly, though in gastric neuroses with pylorospasm and hyperacidity the same result may be obtained.

CHRONIC GASTRIC AND POST-PYLORIC (DUODENAL) ULCER.—*Active or extremely irritable* chronic ulcers may present the same fundamental symptoms as the classic acute or subacute ulcer, but show many and wide variations from the typical picture in the great majority of instances.

"Hunger-pain."—As stated under "acute ulcer," when a distinct and more or less sharply localized *pain or painful discomfort regularly* appears when the stomach should be empty or nearly so, two, three, or four to six hours after a meal, or, on a fasting stomach, it constitutes a symptom of some importance and significance, somewhat intensified if the pain is relieved by the taking of food and if the duration of such amelioration or relief is more marked with a large meal than a small one.

As stated elsewhere "*hunger-discomfort*," amounting to actual "hunger-pain" if much flatulent distention is present, is one of the commonest symptoms of asthenic ("nervous") dyspepsia, and in such instances also the taking of food may give immediate and marked relief. In such instances, as stated, the element of exaggeration must be dealt with and may be most misleading. In general, however, the distress experienced is more diffused than in ulcer and the *large* meal may or may not prove more disconcerting than comforting.

As one of several mutually corroborative symptoms "hunger-pain" is of importance. Existing alone it can merely excite suspicion, save when sharply localized and severe, and, in the presence of an anamnesis suggestive of preëxisting ulcer.

In any event it is evident that such a pain is not born of mere food contact, but distinctly suggests a direct relation to heightened peristalsis or spasm, with hypersecretion and heightened acidity or difficult emptying from any cause.

The greater the part played by direct irritation, the more likely is a definite localization of the pain; *i.e.*, the more active or irritable the ulcer and the nearer it lies to the pyloric ring. In certain cases of duodenal ulcer, unassociated with any hindrance, organic or spasmodic, to free emptying, hunger-pain may be present two hours after eating or even earlier.

Hemorrhage in Chronic Ulcer.—Hemorrhage in chronic ulcer is less frequently visible (10 per cent.) than in acute ulcer and less constant and *persistent* as occult blood in the stools than is the case in carcinoma.

Occult blood is nevertheless quite frequent, and, as in acute ulcer, but much less often, may be not only visible, but massive and even fatal.

It should be remembered that a large hemorrhage may occur with pallor, faintness and even syncope, without vomiting, and be revealed later only by an examination of the stools.

Repeated small hemorrhages may induce gradually and *progressively* a decided, though seldom extreme, anemia.

Fatal hemorrhage occurs in only a small proportion of gastric ulcers, oftener in public than in private cases. The precautions to be taken in the examination for occult blood have been mentioned, but one properly may repeat the warning against accepting positives without an investigation of such common sources of oozing or active hemorrhage as the naso-pharynx, gums and rectum.

Bright blood in the stools is, of course, never of gastric or duodenal origin.

Persistence of bleeding in a case of supposed ulcer under proper conditions of rest and diet strongly suggests malignant growth and usually demands an exploratory incision.

Hyperacidity in Chronic Ulcers.—*In the predominating type of chronic iuxtapyloric ulcers (prepyloric or post-pyloric) high acid values are present in between* 80 *and* 85 *per cent., and in the remainder the values are actually or approximately normal in nearly all instances.*

The importance of these facts is very great with relation to the early diagnosis of carcinoma or the rare instances of its development upon an old ulcer base. So important in fact that if decided symptoms and signs of chronic juxtapyloric ulceration are present and free HCl values are very low or progressively falling, an exploratory incision is imperatively demanded.

In the ulcers outside of or distant from the pyloric segment normal acid values predominate (⅔–¾) but *very* low acidity is present in only 10 to 12 per cent.

It must be remembered always that in early gastric carcinoma the gastric contents may show for a time merely low HCl values and lack both lactic acid and Boas-Oppler bacilli, though these last are present in nearly all cases.

According to the author's experience, however, these HCl values *steadily diminish.*

Hypersecretion.—Persistent or periodic hypersecretion is peculiarly frequent in association with chronic ulcer of the juxtapyloric type whether gastric or duodenal. The HCl values are usually high in the hypersecretion of chronic ulcer and more or less delay in emptying results from the tendency to pyloric spasm. The quantity of fluid obtained from the fasting stomach is above the normal though not necessarily extreme.

After a test meal also the recovered gastric content shows clearly the predominance of the liquid element on standing and sedimentation, 60 to 90 or 95 per cent. of liquid being often manifest. In ulcers of other portions of the stomach the viscus may empty readily and HCl is likely to be normal or but slightly high or low.

In these cases two or more of the classic symptoms, i.e., pain, circumscribed tenderness, vomiting and hemorrhage are particularly likely to be present.

Hypersecretion occurs frequently in simple atony, but is usually slight

and not as likely to show in the recovered test meal so great a predominance of liquid over solids. As stated previously, food retention, and even hypersecretion alone, in a fasting stomach, associated with true hunger pain, strongly *suggests* a juxtapyloric ulcer. In duodenal ulcer with an unobstructed pylorus, the emptying may be more rapid than normal but is not as rapid as in simple achylia. *Obviously*, if one finds rapid emptying with achylia he need not ordinarily consider ulcer, for in the latter some grade of acidity is present unless a malignant transformation is developing.

Tenderness and Defensive Rigidity in Chronic Ulcer.—Both symptoms are variable and inconstant, but very important when present, and both suggest an active process in the area involved, or, marked irritability. As in the case of acute ulcer the location may vary quite widely, but *diffuse tenderness* is of no importance.

As stated in connection with acute ulcer, if the position of any tender or painful point shifts upward, in changing from the erect to the recumbent posture or when the abdomen is strongly indrawn or shifts downward in deep inspiration, the suggestiveness of the symptom is intensified.

Even a post-pyloric ulcer may reveal tenderness to the left of the median line and even in the left hypochondrium. In either gastric or duodenal ulcer it may be low as is readily understood by a reference to the illustrations of the gastroptotic and ectasic stomachs.

Save in direct connection with roentgenologic studies the actual *localizing* value of tenderness is not great. On the other hand its mere persistent presence and strict adherence to site, under like conditions of posture and respiration, is valuable.

RADIOGRAPHY IN GASTRIC AND DUODENAL ULCER.—The essential points are clearly set forth by Dr. Bissell in another section, but the topic may be touched upon here in direct connection with the symptomatology and the use of the stomach tube.

It should be understood clearly that roentgenography and roentgenoscopy are invaluable aids in diagnosis, but that of and by itself roentgenology does not and cannot achieve a correct diagnosis in more than a large minority of the cases.

Furthermore, in any hands save those of the trained expert they are likely to lead to most serious blunders and many futile and unnecessary operations.

The all too prevalent hasty resort to exploratory operations and the overconfidence in roentgenologic findings so commonly observed at the present time have resulted in displacing to an unwarranted degree the careful, exact, and discriminating anamnesis and the results of test meals and investigations of the gastric contents as determined by the stomach tube or more safely in ulcer, by the partially introduced duodenal tube.

One encounters in the arbitrary diagnostic "formulæ" offered the physician by certain noted authorities an extreme example of an excessive and unwise dependence upon roentgenographic findings.

As an adjunct, roentgenography is extremely valuable and oftentimes

indispensable. As a sole dependence it is a broken reed and none are quicker to proclaim this fact than are the best of our modern roentgenologists.*

Proper Field in Gastric and Duodenal Ulcer.—To one so fortunate as to be able to combine roentgenology with careful and intelligent case-taking and modern clinical methods of a less objective type, the clarifying value of roentgenoscopy and serial roentgenography is clearly evident and the accuracy of localization frequently obtainable is most impressive. The following facts should be held clearly in mind:

(*a*) Simple acute or subacute ulcer yields, as a rule, slight distinctive roentgenographic signs, or, none at all, unless it is located so near the pyloric ring as to cause spasm and retention, or, perforates the wall and forms a diverticulum modifying the profile or capable of retaining a portion of the bismuth after the remainder has passed on. In rare instances the ulcer crater may hold a fleck of bismuth which is recognizable apart from the bismuth shadow within the stomach cavity.

(*b*) Chronic infiltrating ulcers create the greatest disturbance in peristalsis and those of the callous perforating type are recognizable in many instances. The fact that, in the standing position, the lesser curvature is shown in profile, and that 80 per cent. at least of chronic ulcers appear in this region makes possible the recognition of the projecting sac or pouch, an air bubble sometimes surmounting the surface of the bismuth.

(*c*) Hour-glass contraction, spasmodic or true, is a readily recognizable roentgenologic phenomenon, not restricted to ulcer, but of decided importance as a corroborative sign. It may be true (permanent) or spasmodic and repeated exposures or the administration of atropin may be required to demonstrate its temporary nature.

Either the false or true form may represent the result of gastric ulcer, and as between that due to malignant growth two chief differences exist. In ulcer the sulcus is band-like and smooth and the isthmus is not central, lying usually toward the lesser curvature by which, in fact, it is formed in most instances. In carcinoma the sulcus is usually broader and less regular, and the isthmus is centrally placed.

In the very rare false hour-glass stomach which results from adhesions the appearance may be deceptive and in certain instances the hour-glass form is present only in certain positions of the body.

SECRETION.—By the use of a "floating" and a "sinking" capsule the degree of hypersecretion or retention may be determinable roughly without the removal of the stomach contents or the administration of a bismuth meal, a measure of some special value in continuous or periodic hypersecretion from whatever cause.

* The heavy expense involved in the maintenance and *expert operation* of a modern roentgenologic outfit places it beyond the reach of the average practitioner and the necessity and propriety of charging adequate fees for such work is as yet so little appreciated by the layman as to constitute a decided limitation to its much needed wider development.

In such a book as this it is necessary therefore to emphasize those non-roentgenologic signs and symptoms of value to the general practitioner.

Schwarz and others have devised opaque capsules of "gold-beaters' skin" which when introduced with the contained excess of pepsin and 4 grams of bismuth are thought to measure the degree of acidity through the time required for their dissolution, compared with a standard table determined experimentally by digestion with fixed amounts of HCl and an excess of pepsin *in vitro*. Neither method is as yet used widely or fully accepted.

Form and Position of Stomach.—Gastroptosis, pyloroptosis, atony, and ectasia are beautifully and directly shown, and may markedly influence our judgment as to the meaning and importance of "retention" and "hypersecretion" alike in cases of suspected ulcer lacking more definite symptoms.

The gastroptotic atonic stomachs and their latent capacity as symptom producers in the presence of coexisting subnutrition and circulatory inadequacy have led to a vast amount of unnecessary and futile surgery for the relief of purely suppositious ulcers or "relapsing appendicitis." Their possessors constitute the least fit and most "operated" clinical type.

Areas of Induration.—It is induration chiefly which makes the roentgenologic recognition of chronic ulcer possible in so considerable a number of cases. It produces a rigidity of the wall which *persistently* interrupts the peristaltic waves without of necessity producing a "filling defect" (alteration of the outline of the contained opaque meal) of the kind and degree encountered in new-growths of the stomach. At all events when persistent it indicates an infiltrating lesion of some sort involving the stomach wall, and is to be considered and evaluated in connection with the anamnesis and the other symptoms and signs.

In such instances the lesion is almost invariably chronic ulcer and one seldom lacking other corroborative evidence. Even though an actual "filling defect" is demonstrable it is rarely of such a character as to deceive the expert and lead to a roentgenographic diagnosis of carcinoma.

Spasm and Incisuræ.—These roentgenologic signs afford only indirect or presumptive evidence of gastric or duodenal ulcer and may be merely reflex manifestations of disease in other portions of the gastrointestinal tract, simple hyperacidity, hypersecretion, erosions, or purely nervous conditions. They are nevertheless of considerable importance if sustained by other corroborative signs and symptoms or a very suggestive anamnesis.

Spasm of the Pyloric Antrum.—The lumen of the pyloric antrum may be so reduced as to contrast strikingly with the *pars media* and *cardiaca* and serial roentgenograms may reveal a resumption of normal proportions during periods of relaxation or a persisting diminution of caliber in long-standing cases or those of persistent and extreme irritability.

Spasm not only extends the period of emptying *but delays the beginning* of expulsion which should commence immediately following the ingestion of the bismuth meal. This results in a six, eight, ten or even twelve hour residue, usually markedly exceeding in amount the residue of mere atony which is seldom greater than one-fourth of the total meal. In rare instances, however, it may account for even a twelve-hour retention.

The incisura is merely a slight spasmodic retraction of the circular fibers,

usually most marked at a point opposite the seat of irritation if due to ulcer. It may be fleeting or persistent, is often a mere indentation, but its importance is in proportion to its persistence.

Actual Pyloric Obstruction.—The benign type of pyloric obstruction is usually due to chronic ulcer (infiltration, scar formation and contraction) and is of excessively gradual production in most instances. This fact is of diagnostic value within or without the field of roentgenology, for with a decided stasis and ectasia, *i.e.*, a huge stomach with actual stasis, one is usually (not always) dealing with obstruction due to chronic ulcer, or adhesions. In new growth obstruction the stomach is usually relatively small, often of the steerhorn type. Furthermore in benign stenosis hypertrophy has taken place and peristalsis is likely to be very marked. The rule is only a general one, but is useful and usually borne out by the nature of the stasis as determined chemically and microscopically.

Post-pyloric (Duodenal) Ulcer.—When one speaks of "duodenal ulcer" to all intent and purpose he must mean ulcer of its first portion, which compose over 95 per cent. of the total and this tissue, as stated previously, is anatomically, embryologically, and, to a large degree, clinically, inseparable from the pyloric portion of the stomach.

Roentgenologically the brilliant work of Lewis Gregory Cole has demonstrated the possibility of proving by serial roentgenography this portion of the gut "normal" or "diseased" in a large proportion of cases. By painstaking work under his technic one may also demonstrate ulcer directly in many instances. Persistent failure of the stomach "cap" (Cole) to attain a normal outline means disease, if many confusing factors can be eliminated.

It will suffice to say here that the demonstration of a wholly normal cap absolutely rules out any ulcer of the duodenum which represents anything more than erosion.

Otherwise, aside from certain cases showing not only hyperperistalsis, which occurs in a considerable proportion of duodenal ulcers and in many other conditions, one must consider them merely as a variety of the juxtapyloric ulcer already fully discussed.

General Dyspeptic Symptoms.—These are more commonly associated with varying degrees of *actual pain* than is usual in other ailments. Because of normal or high acidity the digestion is normal or often more rapid than normal, the tongue relatively clean and the appetite usually good. Indeed true anorexia is an exceptional symptom in gastric ulcer, insufficient nutrition being ordinarily dependent upon the patient's dread of the pain and distress following the ingestion of food.

Fever.—*In cases of insidious obscure or wholly silent perforation which does not result in general peritonitis, a sluggish chronic fistulous abscess results in most instances. Any case combining suggestive symptoms with persistent low-grade fever should suggest ulcer as a possible cause, and such ulcers must be held in mind as possibilities in any obscure febrile case.*

Anemia.—The author feels that too little stress is laid by writers upon the frequency of *obstinate or relatively intractable anemia* and regards it as one of

the more important of the subordinate factors in diagnosis. It is probable that the anemia of gastric ulcer in some cases antedates the lesion, probably invites, and tends to perpetuate it. Whether antecedent or consequent, as the result of persistent or recurrent hemorrhage of varying degree, it assumes an intractable though not extreme type.

Marked intractable anemia of the secondary type, in young persons, associated with gastric disturbance and normal or high acidity is suggestive, and when accompanied by even minor degrees of persistent local pain and tenderness will justify the institution of medical treatment under a tentative diagnosis.

The presence of an active irritable or infiltrating ulcer involving the pylorus or first portion of the duodenum tends to produce hyperacidity, hypersecretion, painful peristalsis, pyloro-spasm or true obstruction at the outlet.

Such conditions yield signs and symptoms more or less complete, but usually sufficient to justify at least a tentative diagnosis of juxtapyloric ulcer and the institution of medical treatment.

Simple hyperacidity is commonly temporary, intermittent, dependent upon known etiologic factors and usually lacks severe pain, localized tenderness and hemorrhage.

CERTAIN PRINCIPLES BASIC IN DIAGNOSIS.—From a study of the diagnostic factors as set forth in the preceding paragraphs and under Roentgenology it becomes obvious that certain facts of great value are fundamental in the diagnosis of gastric and duodenal ulcer.

Primarily one must appreciate the fact that, in general practice, of 1000 cases complaining of dyspeptic symptoms not more than 50 would show a gastric ulcer. On the other hand 700 or more would prove to be victims of asthenic ("nervous") dyspepsia.

Hemorrhage constitutes a corroborative sign of value when present, greater when gross, massive, or merely visible, but very important even when manifested only by a positive thread test or the presence of occult blood in the stools or the gastric recovered content. Occurring initially in a case lacking a history of chronic intermittent dyspepsia it often constitutes the first sign of acute ulcer. Occurring unexpectedly in a case yielding a history of chronic painful "dyspepsia" intermitting or remitting periodically, it strongly suggests renewed activity in an old latent or sluggish ulcer.

Pain.—The clinical significance of hemorrhage is greatly intensified if sharply circumscribed local tenderness or pain co-exists and, still more, if such pain bears a definite time relation to the taking of food, relatively constant for the individual affected, varying only with the amount and character of ingestæ or the nature and degree of bodily activity following the meal.

It is evident that the pain of ulcer may be that of direct irritation, attrition, food contact, stretching and contraction or compression of the affected area, extension of the necrotic process to sensitive tissues, or, the drag of adhesions. That in all save the wholly silent cases each of these factors, save the last, must play a part, and that in many all of them are involved is obvious. It is true, nevertheless, that in the case of the chronic infiltrated

ulcers the most dominant factor is peristaltic activity. With this is combined that of hyperacidity and attrition in the case of all juxtapyloric ulcers, but to what degree these enter into the pain complex is difficult to determine.

Anyone familiar with the phenomena of chronic sluggish or relapsing ulceration can readily understand the wide variations in the degree of pain or discomfort encountered which ranges from zero to that which may be termed fitly "atrocious" and it is equally easy to comprehend that in many instances direct surface irritation of any sort may be a trivial factor in pain production. In the case of acute ulcer this factor is of great importance at certain stages as is readily shown by the immediate painful effect so often produced by hot drinks, acids, and the like.

The degree of surface irritability is no doubt largely determined by the particular stage of ulceration and the presence or absence of surrounding intense congestion or actual inflammation and a parallel doubtless is to be found in the varying pain response of an ulcerative stomatitis.

Here also, however, we must deal with the question of peristalsis and this is true especially of acute juxtapyloric ulcerations. Acidity is less often excessive in these acute cases, but again recalling the varying but often exquisite sensitiveness of involved areas in stomatitis one can readily believe that *excess* of acid is not always necessary to a painful reaction.

The administration of alkalis and the temporary partial or complete relief thereby afforded in most instances is only in part the direct effect of neutralization of acidity upon pain inasmuch as it secures also to a greater or lesser degree a diminution of peristaltic activity. On the other hand, it is well known that anesthesin and orthoform administered after an alkali will often reduce or abolish residual pain, whereas if given primarily and alone to the same patient little or no effect is evident in many instances. Their effect is largely that of a local anesthetic.

Oil checks peristalsis and probably induces a periodic regurgitation of the alkaline duodenal contents.

Circumscribed Pressure Tenderness.—Obviously the presence of this important though inconstant and occasionally misleading sign is dependent chiefly upon the acuteness of the ulcerative process, the depth of ulceration, the factor of irritability, *per se*, the presence or absence of adhesions, and the location of the lesion. It must be evident also that in many instances it may be wholly lacking or substituted by or associated with a diffuse tenderness clinically indistinguishable from that associated with asthenic dyspepsia, simple hyperacidity and hypersecretion, atony and other common gastro-intestinal disorders. When so diffused it loses all importance as an isolated symptom. When circumscribed it shows a peculiar fixity in location from day to day varied only by changes of posture or the phases of forced respiration in some instances.

A point too little appreciated and emphasized is the fact that it may be present only at a specific interval after meals, corresponding to the period of maximal pain if this be a feature of the case, but manifesting sometimes the same periodicity in the absence of decided pain.

Retention and Hypersecretion.—In juxtapyloric ulcers the factors of retention and hypersecretion are of cardinal importance and one readily understands that either pylorospasm, actual scar contraction or inflammatory tumidity may so interfere with the emptying of the stomach as to produce a condition of retention which in the case of organic obstruction may attain such proportion as to create ultimately a high grade of motor insufficiency and actual stasis of the benign type.

Shortened Emptying Time.—Complete emptying of the stomach within two hours or less suggests achylia gastrica, duodenal ulcer and less often gall-bladder disease. According to the author's experience the stomach empties most rapidly in cases of achylia gastrica and in cases of duodenal ulcer well removed from the pyloric ring and associated with relatively low acid values. Hyperperistalsis is frequent in duodenal ulcer.

FIG. 465.—Normal "cap." *A*, "cap;" *B*, sphincter; *C*, lumen of sphincter; *D*, congenital fold; *E*, terminal wave. (*After Lewis Gregory Cole.*)

The hypertonic, "cowhorn" (steerhorn) stomach normally should discharge the standard bismuth meal completely in three hours. An orthotonic stomach of the "fish-hook" type carrying its lower pole at, or just above the navel usually is empty after four hours. If the pole lies below the navel the emptying time is usually increased to six or eight hours, or, by a period varying somewhat inconstantly with the degree of gastroptosis and latent or demonstrable atony or ectasia. In cases of demonstrable atony or atony and ectasia without actual pyloric stenosis the six-hour period may not be extended decidedly, even in the presence of marked sagging and atony. An emptying time of twelve hours or more strongly suggests, and one of twenty-four hours or more, proves the existence of an *organic* obstruction at the pyloric outlet. With respect to ulcer it is obvious that the question of induced pylorospasm and prolonged retention must largely

depend upon the proximity of the lesion to the pyloric ring and the activity or irritability of the involved area.

A normal emptying time does not prove that ulcer is absent nor would one at this time accept Haudek's dictum that a retention period exceeding six hours is proof positive of an organic lesion of the stomach wall.

The patient should not lie down during any tests for retention, unless for purposes of comparison. It should be remembered that a barium sulphate meal requires much less time for complete expulsion than does that containing bismuth.

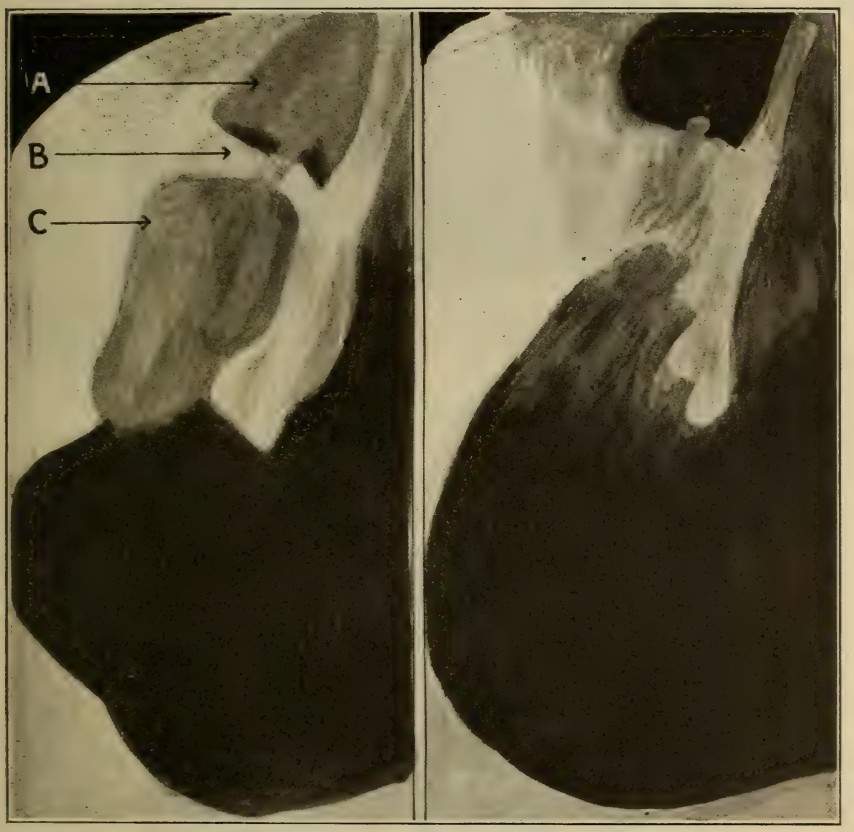


FIG. 466. FIG. 467.

FIG. 466.—Systole. *A*, "cap;" *B*, phincter; *C*, terminal wave (*After Lewis Gregory Cole.*)

FIG. 467.—Diastole. (*After Lewis Gregory Cole.*)

Antiperistalsis.—The presence of antiperistaltic waves is a relatively common clinical phenomenon in stomachs distended by air or gas in the presence of organic obstruction at or near the pylorus and in connection with ulcer in the same region. Roentgenologically it is less often demonstrable under the use of the bismuth or barium meal, but its significance is the same.

Ulcer with Adhesions.—The diagnosis of ulcer with adhesions depends

largely upon the general symptoms of ulcer as stated, combined with those referred to adjacent organs and, sometimes, a sensation of dragging or pull-

FIG. 468. FIG. 469.

FIG. 468.—Post-pyloric ("duodenal") ulcer. *A*, deformed cap; *B* and *D*, everted edges of ulcer; *C*, crater of ulcer. (*After Lewis Gregory Cole.*)

FIG. 469.—Fleck of bismuth in crater of ulcer. (*After Lewis Gregory Cole.*)

FIG. 470.—Ulcer on anterior wall of "cap." *A*, deformed "cap;" *B*, ulcer, seen full face. (*After Lewis Gregory Cole.*)

ing experienced in certain movements or attitudes. Very often a tumor may be palpated, but many cases offer none but indefinite or misleading signs.

PERFORATION.—*The symptoms of acute perforation into the free peritoneal cavity are those of sudden severe pain, and abrupt profound surgical shock,*

FIG. 471.—Post-pyloric ("duodenal") ulcer, non-obstructive, involving the sphincter. *A*, "cap," deformed; *B*, ulcer; *C*, terminal wave (antrum). (*After Lewis Gregory Cole.*)

FIG. 472.—Complete destruction of "cap" (first portion of duodenum). *A*, shows destruction of cap; *B*, terminal wave (antrum). (*After Lewis Gregory Cole.*)

succeeded by symptoms of general peritonitis, and often, but by no means invariably, occur in cases giving a clear history of ulcer.

Classification.—Complete perforation occurs in three forms, viz.,

1. *The acute*, as described above.

2. *The subacute*, in which there may be less violent pain and a delay in the onset of peritonitis owing to primary limitation and secondary leakage into the peritoneal cavity.

3. *The Chronic Cases.*—In perforating but contained cases not only may the symptoms of perforation be slight and ephemeral, but they may be wholly absent save in the form of a persistent or recurrent low fever.

On the other hand, the violent symptoms of pain, shock and collapse in perforation into the general peritoneal cavity may be the first which suggest a chronic silent, slowly perforating ulcer, of years' standing.

It is evident that gastric ulcer is not a single-symptom disease, but one that usually demands a careful grouping of probabilities and a thorough knowledge of all of the diagnostic factors involved. No doubt an immense number are overlooked and go on to spontaneous healing, certainly many exhibit hematemesis or die of perforation without previous recognition.

Having these facts in mind, the author believes that wherever any reasonable probability exists that a given case is one of ulcer, the physician should not wait for more light but should at once institute appropriate medical treatment.

DIFFERENTIAL DIAGNOSIS.—Gastric erosions cause great difficulty, but are in general to be distinguished by a more diffuse burning sensation upon the taking of food, actual pain being as a rule, slight or absent.

Pressure tenderness is usually lacking and hyperacidity is the exception. The wash-water of the fasting stomach contains shreds of mucous membrane, but this may occur in other conditions.

Finally, inasmuch as, aside from surgical intervention, which is distinctly contraindicated, a case of erosion demands much the same treatment as ulcer, it is not necessary to weigh too carefully those differential points which, the writer believes, must be taken cum grano salis, the points of distinction being less definite and absolute than are formally stated above.

Gastric Hyperesthesia not only involves the whole stomach outline, but is associated with distinctive neurotic symptoms.

Hyperacidity.—*It is an extremely common condition*, and in the absence of definite signs of ulcer, differentiation often proves impossible, but it must be remembered that "hyperacidity" may accompany "asthenic" ("nervous") dyspepsia, with or without marked atony, chronic constipation, hyperacid chronic gastritis, certain lesions of the appendix and gallbladder, rare instances of benign stenosis or early malignant growth, gastric syphilis, or mere lack of exercise and overeating. Fully one-third and, perhaps, one-half are associated with the stigmata of "congenital asthenia" and a nutritional deficit.

In not more than one case in five of hyperacidity do we find ulcer, prepyloric or postpyloric acute or chronic. In cancer it is rare, progressively diminishing, and usually represents cases developing upon a preëxisting ulcer. About one case in ten may be chargeable to the gallbladder or appendix. In one out

of a hundred cases it may accompany benign pyloric stenosis. If, therefore, one were to make a diagnosis of ulcer upon the basis of existing hyperacidity he would be taking extremely "long odds." It is convenient and proper to consider a free HCl content exceeding 40, and a total of over 70, as representing "hyperacidity," although this amount of free acid or much higher values may produce no *symptoms* whatever, whereas lower values may excite them in other cases.

The presence or absence of "hypersecretion" should be determined whenever possible.

Hypersecretion.—The greater number of cases of hypersecretion represent *motor defects* of some degree and, indeed, this statement doubtless applies in a large degree to "hyperacidity."

The sources and character of such defects are varied and the occurrence of either hyperacidity or actual hypersecretion is conditioned upon functional adequacy of the secreting glands. In congenital asthenia the structural conformation and functional and nutritional instability are ideally adapted to produce the two symptoms under consideration. In the gastric crises of locomotor ataxia and in ulcer upon the lesser curvature or just beyond the pylorus the element of obstructive spasm is clearly a factor quite apart from actual scar contraction or obstructive tumefaction in the ulcer cases.

The element of spasm is undoubtedly the important one in reflex hyperacidity or hypersecretion of disease of the appendix or gallbladder and in all the conditions mentioned heightened stimuli to secretion may be assumed. The occurrence of hyperacidity as a purely temporary and fleeting phenomenon in association with violent emotion or profound mental depression is well known. To interpret recurrent periods of burning pain or discomfort, after eating, followed by intervals of entire immunity, as, of itself, constituting strong evidence of actual gastric ulcer is to raise to an exaggerated level the commonest phenomena of functional gastric disorders. Such attacks coming on very late after meals or persisting over an interval of more than three or four hours, does at least suggest hypersecretion rather than mere hyperchlorhydria. This assumption is strengthened by the concurrence of late, and free, acid eructations ("water brash"), usually also a purely trivial and transient though troublesome phenomenon.

Acute hypersecretion is due usually to spasmodic or organic narrowing of the pyloric outlet and copious vomiting of acid secretion in gushes and with little persisting nausea and in quantities wholly out of consonance to the stage of digestion and quantity of liquid intake is characteristic.* Such attacks may be transient, trivial and reflex but must never be accepted as such until an organic lesion of the nervous system or the gastrointestinal tract has been sought for and excluded.

Chronic hypersecretion is evident if the fasting stomach contains fluid *persistently* exceeding in amount 25 or 30 c.c. and its significance depends largely upon its volume, the presence of free HCl, and the ratio of liquid to

* The acid taste is important only as indicating the presence of acid. All *normal* gastric content is acid to the taste.

solids if the latter are present. The presence of *visible* food remnants proves retention from atony, spasm, or some form of actual pyloric obstruction.

In other words, persistent hypersecretion represents chiefly *motor defect* and usually if not always a definite *obstruction to outflow* either organic or spasmodic. Furthermore the amount of fluid in the fasting stomach and the degree of hyperacidity increases in proportion to the grade and persistence of the obstruction and the amount of *secondary* atony and ectasia in all save the stasis induced by malignant growths in which acidity (free HCl) falls as obstruction, hypersecretion and retention increase. Continuous hypersecretion, *i.e.*, the presence of an excess of gastric content in a *fasting stomach* is almost positive proof of ulcer, gall-stones, chronic appendicitis or disease of the intestines. In itself it strongly suggests but does not prove chronic ulcer. One of its most characteristic symptoms is epigastric distress upon arising in the morning. Hunger-pain or discomfort is of course present, but has been discussed sufficiently hitherto.

Alimentary Hypersecretion.—This condition, a mere excessive amount of secretion attending the digestive process, is of importance chiefly with relation to asthenic dyspepsias and associated ptosis and atony of the stomach. The fasting stomach contains no excess of liquid and free HCl is not of necessity present in excess. The relation of liquid to solid in a recovered test meal has been dealt with in another paragraph. The chief interest in this condition arises from the fact that the attacks of pain or discomfort resemble extraordinarily those of ulcer. Many cases are symptomless if acidity is low, nutrition well preserved, and no marked symptoms of nervous instability present. Severe attacks are so exactly like ulcer as to make differentiation impossible save in the presence of other distinctive signs and in many instances the patient's welfare may demand the institution of ulcer treatment. The condition is not a common one.

Carcinoma of the stomach presents little difficulty save in the relatively rare achlorhydric "ulcer" cases which almost always prove later to be early cases of malignancy. Those rare cases in which a carcinoma develops upon an old ulcer base may reveal more or less clearly and frequently the antecedent history of simple ulcer.

The obstructive cases of gastric ulcer present the picture of benign stenosis elsewhere fully described, in striking contrast to that of the malignant form.

With respect to **early carcinoma,** *one of the chief factors in diagnosis is to be found in progressive persistent impairment of health and loss of weight together with steadily diminishing or wholly absent HCl values or actual achylia.*

The absence or presence of pain or even its character if existent is not distinctive, though the dull relatively persistent character of the pain of cancer is, in general, suggestive.

The pain of cancer is ordinarily less sharp and paroxysmal, and more continuous, and the superficial or deep tenderness is *usually* slight or wholly absent.

The roentgenologic evidence is conclusive in many instances. In many cases tentative treatment must be undertaken and the case judged by the

relief of symptoms, improvement in nutrition and cessation of any bleeding which may have existed primarily.

The infrequency of ulcer with very low HCl values becomes a differential factor of considerable importance, and the age of the patient may aid the exclusion of malignant growth in young patients, though this factor must not be permitted to outweigh other important signs and symptoms.

A palpable tumor makes its appearance in a large majority of the cancer cases and is usually solid and firm, whereas in ulcer, save in pyloric *obstructive* cases or where adhesion and perforation have occurred, if any tumor is felt on the wall it is usually thin and disc-like, representing the inflamed ulcer base.

The nutrition suffers greatly in carcinoma and is associated with a striking persistence of symptoms, a progressive failure of strength and if not operated early the gradual development of the well-known cachectic appearance and morose facies. Hemorrhage is more consistently of the oozing type, appearing as "coffee-ground" detritus in the vomitus and stomach washings, and finally and most conclusively an analysis of the stomach contents reveals in most instances a total absence of free hydrochloric acid. In 60 per cent. or more lactic acid is present and in at least 90 per cent. the Boas-Oppler bacillus is demonstrable in large numbers in the stomach content or the stools.

OPERATIVE CASES OF GASTRIC ULCER.—*The tendency to perform radical surgical operations of the major type as a primary measure in gastric ulcer is to be deplored, and many such cases are undoubtedly those of simple erosion. Nearly every case even of true ulcer should first receive prolonged medical treatment and the knife be reserved for the obstinate, recurrent, persistently hemorrhagic, or perforative cases, for those associated with adhesions which tend to impair the health or persistently annoy the patient, or for actual pyloric stenosis and true hour-glass stomach.**

Closing Comment.—Surgical reports are valuable as showing the immediate mortality of operative procedure, but give only slight information as to the ultimate state of health of those operated. Medical reports are but little better.

Autopsy records show so strong a tendency to multiple ulcerations (20 per cent. plus) as to make doubtful the entire adequacy of surgical excision.

The general practitioner, especially, sees a host of "successfully" operated cases whose last state, months or years after, is infinitely worse than their first.

The surgeon sees many cases of missed diagnosis on the part of the physician, but with ready and confident recourse to the exploratory incision must still miss a considerable number and perform many futile and unnecessary operations.

To balance fairly the present sins of medical omission against those of surgical commission is absolutely impossible.

* One of the most eminent of American surgeons has stated recently to the author that he now operates primarily only upon perforated cases, "hour-glass stomach" and actual pyloric obstruction.

To outline anything purporting to be an **infallible** *formula for the diagnosis of gastric ulcer, with or without the knife, would be a futile and ridiculous procedure.*

We may only consider the typical picture which is reasonably clean-cut and definite save as to exact anatomic localization and set forth as clearly as may be the evidence which must be taken and sifted with respect to the many atypical forms.

GASTRIC EROSIONS.—Under various conditions and influences, both local and remote, the gastric mucous membrane may show a surprising vulnerability, and erosions are easily produced varying in size from that of a pin head to that of a lentil. Various names have been given to this condition and Einhorn has described it as a separate disease; but in view of the fact that it may occur in the asthenic dyspepsias, in chronic gastritis, achylia gastrica, and, *probably, as an early stage, in certain gastric ulcers*, it should hardly as yet be dignified to that extent.

Symptoms.—*They may closely simulate the classic gastric ulcer with considerable or small hemorrhages, be entirely symptomless, or blend with the symptom-complex of the various diseases above mentioned.*

In those cases simulating clinical ulcer the pain is usually less intense than in typical ulcer, and comes on earlier, but may continue until the stomach is empty.

Stomach washings may show tiny shreds of membrane, local tenderness is not marked and mucus is always present in excess in the vomitus or removed contents if a chronic gastritis is present. **Diminution or even entire absence of hydrochloric acid is the rule.** *Occult blood in the feces and slight staining of the stomach washings are common findings. Under proper treatment recovery is usually prompt though recurrences are common.*

Occult blood.

Comment.—Differential diagnosis is important only as preventing unnecessary operations, as the medical treatment of the two conditions is almost identical.

GASTRIC CARCINOMA

Causes unknown.

Etiology.—Frequency and Predisposing Causes.—The stomach is more commonly the seat of cancer than any other part of the human body and 50 per cent. of all cases of carcinoma in the male are gastric.

Age markedly affects the ratio of incidence and the disease is largely one of the fifth (18 per cent.), sixth (28 per cent.) and seventh (28 per cent.) decades. About 8 per cent. occur during the fourth decade and 14 per cent. in the eighth. Such growths are rare between the ages of twenty and thirty (1.5 per cent.) but are always possible.*

Sex.—It is slightly more frequent in males than in females and heredity is a factor of some slight importance.

* The author recalls a serious and humiliating early blunder due to the fact that the extreme youth of the patient (fifteen) made him loath to accept what should have been regarded as positive evidence of malignant growth.

At this age it is one of the rarest of diseases.

It is wholly probable that the scar of preëxisting gastric ulcer may, in rare instances, undergo malignant change.*

The frequency of such a transformation as stated by modern writers varies from a fraction of 1 per cent. to 71 per cent.† The higher figures have gained only slight credence and the present consensus of opinion places its incidence at about 2.5 per cent.

Usually primary.

Traumatism seems in rare instances to be a factor and some authorities lay much stress on mental worry or strain and even upon the habitual use of hot drinks, but it is probable that these conditions exercise little influence.

Types.

Varieties and Preferential Sites.—Cancer of the stomach is almost always primary though, rarely, secondary to cancer of the pancreas, esophagus, and, yet more rarely, the breast, uterus or other organs. The most common form is the cylindrical-celled adeno-carcinoma. Encephaloid is next in frequency, then scirrhus and colloid.

Sites.

Malignant growths of the stomach are frequently the source of *metastases* in their advanced stages. In nearly 80 per cent. the tumors develop between the orifices on the lesser curvature and at the pylorus. In the latter situation they tend to cause early obstruction and, sometimes, dilatation. Growths at the cardia are next in frequency, are often associated with gastric atrophy and occasionally with marked esophageal distention and obstruction. The stomach may be greatly displaced in the case of pyloric growths, and adhesions may obscure and complicate the clinical picture. Such tumors are ordinarily, however, freely movable and in rare instances may be found in almost any part of the right half of the abdomen.

Dislocation and adhesion.

Symptoms.—*Cases may reach an advanced stage of development without producing any recognizable symptoms save those made evident by examination of the stomach contents, but, ordinarily one finds:*

(*a*) *Dyspepsia.* Eructations, pressure, heavily coated tongue, and foul taste in the mouth.

Seldom severe.

(*b*) *Progressive loss of weight unrelieved or but temporarily improved by a period of absolute rest and careful feeding.*

(*c*) *Marked progressive impairment of strength and endurance.*

(*d*) *Pain.*—Usually dull, boring, and somewhat *persistent.* This symptom is present in 90 per cent., but is sometimes wholly absent in all stages (12 to 20 per cent). Usually epigastric, it may be referred, especially if adhesions exist or if the stomach is greatly displaced, and in rare instances it may be severe and spasmodic.

Pain in the left upper or even in the lower quadrant is encountered occasionally.‡

* This was asserted by Cruveilhier, who first described gastric ulcer, and by Rokitansky in 1835. Later it was discussed by Brinton, Leube, Lebert, and especially worked out by Hauser in 1883.

† L. B. Wilson and McCarthy, Amer. Jour. Med. Sci., Dec., 1909.

‡ As in the case of the perigastritis of ulcer, the increase of pain on walking, deep breathing, strongly indrawing the abdomen or changing from the erect to the recumbent posture suggests peritoneal involvement.

One must not forget that accession of pain following the ingestion of food and amelioration of such distress by alkalis may be present in 25 or 30 per cent. of cancer cases.

Immediate pain upon taking food usually points to a cancer at the lower extremity of the esophagus.

Persistence of pain under a short course of ulcer treatment is most suggestive of cancer.

Usually moderate.

Associated *tenderness* may or may not be present and is usually not sharply delimited or extreme. It is most marked usually in the right epigastrium and may be accompanied by sensitiveness in the lower dorsal region.

May be extreme.

(*e*) *Anemia.*—At first moderate but *progressive*, this may later become extreme, though usually preserving a definite secondary type (see "Anemia"), but advanced ulcerative cases show at times extreme grades closely simulating the anemia of atrophic gastritis or pernicious anemia itself and it is more or less characteristically resistant to treatment. *Leucocytosis* is usually present in metastasis, or rapid ulceration.

Resists treatment.

(*f*) *Fever.*—Fifty per cent. of the advanced cases are accompanied by a moderate fever of the hectic type, in rare instances associated with "chill and fever," due probably to septic absorption from an ulcerating surface.

Indican and diazo.

(*g*) *Urinary Signs.*—The only important urinary findings are indicanuria which is usually present to a marked degree, and the diazo-reaction, as reported by the author, in certain *advanced* cases.

A terminal symptom.

(*h*) *Cardiac Weakness.*—Marked cardiac weakness and edema of the lower extremities are usually terminal symptoms.

Common symptoms.

(*i*) *Anorexia.*—This is usually marked and often associated with a somewhat significant repugnance to red meat.

It may be an early and very important symptom.

(*j*) *Nausea.*—This is present in a great majority of the cases and may be extremely persistent and troublesome.

Seldom lacking.

(*k*) *Vomiting.*—A symptom present in 90 per cent. of the pyloric cases and occurring usually one or more hours after meals. It is ordinarily occasional, early and irregular, but may be frequent or very rarely almost continuous, tending to rapid exhaustion and death.

Only in rare instances does it show the individual periodicity of ulcer.

The relief afforded by vomiting is seldom complete and may even increase any preexisting pain or initiate the symptom.

Evidence of the first statement is found in the relative rarity of self-induced vomiting in cancer cases, as compared with those of ulcer, simple hyperacidity or hypersecretion.

In a surprising number of the author's cases the disease has been initiated by vomiting attacks resembling those of acute hypersecretion or gastro-enteritis.

Macroscopic.

(*l*) *Hemorrhage.*—In from 20 to 30 per cent. there is recognizable hemorrhage and it undoubtedly occurs in a much larger portion. Oozing is the more frequent form and in all cases of suspected cancer of the stomach, the stools should be examined as well as the vomitus and stomach contents.

Bright red arterial blood in the vomitus is unusual, it being ordinarily in small dark grumous clots or "coffee-ground particles." "Coffee grounds."

Occult blood is a peculiarly constant finding in gastric carcinoma (90 per cent. on repeated examinations).

(*m*) *Cachexia.*—In advanced cases the skin is pale, yellow, inelastic and ill-fitting, because of anemia, malnutrition and loss of weight, and often shows areas of pigmentation or pigment atrophy. *A curious earthy pallor is often present* and is more or less characteristic, and, quite as striking, is the peculiar *facial expression* often encountered in these cases, the patient appearing saturnine, anxious and depressed. Wasting. Pallor.

(*n*) *Tumor.*—A palpable tumor may be found ultimately in about 80 per cent. of the cases, may occupy almost any part of the right upper quadrant and either be freely movable or attached by adhesions. Tumors of the cardia or pylorus, if so fixed, may be entirely obscured by the ribs and liver, respectively; tumors of the fundus are less movable in respiration than are those of the pylorus. Examination both when the stomach is full and when it is empty should be the rule and immersion in the warm bath, or even narcosis, may be necessary. Tumors of the cardia with rare exceptions produce a stenosis readily recognized by the tube or esophageal bougie. The liver surface and margin should in all cases be carefully examined for the presence of metastatic nodules. Location. Source of error.

Stomach Contents.—To obtain the stomach contents one should thoroughly wash out the stomach in the evening and give a retention meal, followed by a test breakfast if desired the following morning, and should use a Boas test meal, or the shredded wheat breakfast. The preliminary washing may show an excessive quantity of fluid or give evidence of imperfect digestion, impaired motility and pyloric obstruction with stasis and fermentation.* On the other hand, pyloric incontinence or duodenal stenosis may be indicated by the persistence of excessive quantities of bile. General findings.

Chemical Findings.—Hydrochloric acid is rarely present in any considerable quantity. It is probable that most of the rare cases in which high percentages of free hydrochloric acid are found have their origin in an old ulcer base or scar and in such cases a progressive diminution is of diagnostic significance.† HCl absent.

The ferments vary *pari passu* with the HCl but usually are entirely absent or greatly reduced. Lactic acid is usually present in quantity if stenosis exists.‡ Lactic acid.

Microscopic Findings.—The microscope may reveal fragments of growths and evidences of hemorrhage, the Boas-Oppler bacillus in great numbers if Boas-Oppler bacillus.

* Substances are sometimes retained for many days as was the case with a segment of orange brought in by a patient under the impression that it was a cyst, its residence in the stomach having completely changed the appearance of the undigested food.

† B. Moore, Lancet, p. 1121, 1905, reports gastric subacidity or anacidity in two-thirds of all cases of cancer affecting *other* organs, confirming that of the elder Fenwick.

‡ Estimation of the albumin contained in the normal saline solution used for washing the previously emptied and washed stomach involves too much handling, as it is present only in ulcerating *carcinomata or occasionally in gastric ulcer.*

stasis and achlorhydria co-exist and the significant absence or sparseness of yeast fungi or sarcinæ.

Early Diagnosis.—This is of cardinal importance in the light of modern surgery and the following points are helpful:

1. *Anorexia* amounting to loathing may be an early symptom, but is common also in simple atony and certain asthenic functional dyspepsias.

2. *A persistent subjective or visible distention* unaffected by dietetic restriction.

3. *Vomiting* when present procures but little relief to any existing pain.

4. *Failure to regain weight under rest and feeding*, an extremely important symptom.

5. *Progressive loss of strength* is another symptom of great importance.

6. *Onset of symptoms*, usually supervening upon a good, or fairly good, digestive record. This is said to be the exception by some surgical authorities, but the author's experience agrees with that of most internists in support of the general statement.

An abrupt but persistent gastric upset has been the first definite disturbance recognized by the patient in some instances.

7. Persistence of pain under rest and a bland diet.

8. Persistence of occult blood under same conditions.

9. Peculiar persistence of any existing epigastric discomfort.

Differential Diagnosis.—*The following facts should be borne in mind, viz.,*

Chronic gastritis.

that in chronic gastritis, the hydrochloric acid may be diminished or absent and the ferments more or less inactive, but in this disease lactic acid is not present, pain is absent or slight, hemorrhage does not occur except in connection with simple erosions or cirrhosis of the liver, emaciation is seldom as early, progressive or extreme, and a tumor is not to be found.

Achylia gastrica.

In achylia gastrica, hydrochloric acid, lactic acid and the ferments are absent, there is usually no pain or hemorrhage and never tumor. Emaciation is usually not marked or progressive until the condition has lasted for long periods and symptoms usually are readily relieved, though occasional exceptions occur.

In gastric ulcer, hydrochloric acid is usually in excess, less often normal, rarely low, very rarely absent, lactic acid is usually absent, ferments are normal, emaciation is ordinarily less extreme and decidedly less persistent than in cancer, and a tumor is absent save in the rarer instances when one may feel the infiltrated base of an ulcer, a thickened pylorus or a mass of adhesions.

In such cases one may encounter both obstruction and stasis but the benign character of the latter is established readily.

Ulcer has only pain and hemorrhage in common with carcinoma. Its hemorrhage is usually arterial, its pain more paroxysmal, directly related to the taking of food, and usually marked or completely relieved by emptying the stomach or by the ingestion of bicarbonate of soda. Cancer occurs somewhat more frequently in the old, ulcer more frequently in the young.

Finally.—The diagnosis of cancer must rest chiefly upon the presence and

group value of some or all of the following symptoms, viz.: persistent anorexia; intractable, progressive, dyspeptic symptoms, especially in middle-aged and in elderly people; the presence of lactic acid in excess after a shredded wheat test meal preceded by lavage; stasis and fermentation with achylia; evidence of hemorrhage; Boas-Oppler bacilli in quantity; persistent anorexia, early and progressive loss of strength; emaciation, and tumor.

Vomiting is an inconstant symptom and is usually late and the same statement applies to the presence of lactic acid, the Boas-Oppler bacillus, and in less degree, to the achlorhydria.

Pain.—Actual pain is likely to be a late symptom, but is seldom wholly lacking and a **persistent distress** *is both usual and significant.*

It bears a much less constant relation to food ingestion and ejection or removal of the stomach contents, than is the case in ulcer.

The evidence of the X-ray is often either conclusive or extremely suggestive and it should be remembered that actual stenotic stasis and fermentation with absent hydrochloric acid, Boas-Oppler bacilli, constitute a practically pathognomonic group, subject to only rare exceptions. (See also "Roentgenology.")

As compared with benign stenosis the stomach is often small and seldom attains either the degree of ectasia or the vigor of peristalsis seen in simple chronic obstruction.

Every case of suspected carcinoma should if possible undergo a short course of antiluetic treatment; all, a Wassermann and a luetin test.

Syphilis of the stomach is more frequent than had been supposed prior to the introduction of these specific tests.

Consistently early, absolute, diagnosis *is impossible save by some purely fortuitous circumstance.* **Timely diagnosis,** *from the present surgical point of view, is less exacting but will probably prove disappointing in end results of operations. In all cases one must seek to find and combine as many symptoms as are ascertainable.*

The author depends chiefly upon the X-ray findings, anorexia, persistence of pain and dyspeptic symptoms despite treatment, weight loss and weakness resisting rest and dietetic management, diminishing or absent HCl and retention of food remnants, all these occurring at the cancer age.

Coexistence of anorexia, persisting pain or painful distress, blood, and low HCl values is a combination of importance.

One cannot exact even this complex in its entirety and make early diagnoses—a mere strong probability often justifies exploratory incision.

The Abderhalden test has been proven worthless whether as a test for cancer or for pregnancy by the complete and exhaustive work of D. D. Van Slyke and associates at the Rockefeller Institute. The many other tests assumed to be relatively or actually specific, also have fallen by the wayside.

GASTRIC CRISES.—Lightning-like attacks of agonizing cramping epigastric pain, associated violent vomiting, at first of but a few minutes duration, later persisting for hours, or days, separated at first by long intervals, but progressively increasing until for a period of weeks they may occur

Tabes.

Variations.

daily, are characteristic of tabes dorsalis in most instances* and may constitute the first symptom.

The recession is usually as abrupt as the onset, but unusual cases are encountered in which the pain is absent from the complex or the critical termination may be replaced by slow tedious improvement.

Intestinal and rectal crises of much the same type also occur. As an early symptom it is rare but important and would be extremely difficult to place in the absence of tabetic symptoms because of its resemblance to many other conditions. Proof of syphilis and the increasing frequency and duration of the apparently causeless attacks combined with their dramatic suddenness of onset and recession are valuable suggestive factors in differential diagnosis.

The Deep Reflexes.—The loss or marked disturbance of tendo achillis response and the knee-jerks will almost invariably precede the true crises and with rare exceptions no error is possible if the examination includes these tests, as every examination of a patient should.

SPASTIC SPLANCHNIC ABDOMINAL CRISES.—Frequent attacks of severe paroxysmal pain of brief duration (fifteen to thirty minutes) induced by exertion or emotion and frequently by the recumbent position, if occurring in men past forty, suggests spasm of the splanchnic vessels, hypertension or *cardiac angina with predominant epigastric localization.* The aortic region is usually tender and another type is actually combined with true angina pectoris. The attacks may show the same tendency to progress over a period of many years as is the case in tabetic crises, but are more frequent, less sudden in onset and recession and lack the gastric disturbance. The age, the presence of arteriosclerosis and hypertension, the lack of relation to meals or constant gastric findings should suffice to exclude ulcer. Max Buch claims that the relief afforded by diuretin, 3 to 4 grams and tincture of strophanthus, 15 to 24 minims daily, is of diagnostic importance.†

SYPHILIS OF THE STOMACH.—At the period of general infection dyspeptic symptoms are not uncommon, though usually indeterminate or taking the form of hemorrhagic erosions due to specific endarteritis. Gastric syphilis is by no means as uncommon as formerly supposed and, clinically, may be represented by diffuse gastritis, ulcer, gumma, diffuse pyloric infiltrations, or by gastric adhesions, and hence may resemble simple chronic gastric catarrh, ectasia, erosions, ulcer or carcinoma.

Three cases.

Three particularly interesting examples may be quoted from those which have come under the author's observation. In one, an extremely puritanic old gentleman, of seventy, there was a palpable pyloric tumor, stenosis with marked dilatation, extreme gastric intolerance, tenderness and emaciation. The cause was suggested by Hutchinsonian teeth in a daughter.

* Similar crises may be encountered in developed spinal paralysis, multiple sclerosis, morphinism, lead colic, etc.

† These attacks may be entitled to separate and specific description, but as stated previously, the personal experience of the author has not convinced him that they differ often in significance from ordinary angina pectoris with epigastric pain maximum.

Another was a steeple climber, formerly a sailor, of thirty-five, who presented a flat, definite and exquisitely tender area in the left epigastrium and atrocious paroxysmal pain. The third was a young man of twenty-seven, who presented the clinical symptoms of malignant pyloric stenosis. In all three specific medication promptly relieved the pain and resulted in complete disappearance of all symptoms.

Excessive pain and tenderness with signs of carcinoma were the prominent features and in all the evidence of past syphilis was directly or indirectly adequate to justify a diagnosis. Coffee-ground vomitus was present in the first and third. The first and second cases were remarkable because of the palpable tumors.

Whenever possible, a brief intensive course of antisyphilitic treatment should be instituted in all doubtful cases of the ulcer or carcinoma-like types, as a part of the diagnostic procedure.

A Wassermann or luetin test also should constitute a part of the procedure in appropriate cases, but negative results should not halt the tentative therapy, which, moreover, will often enable one to avoid the raising of the question of lues in sensitive patients.

TUBERCULOSIS OF THE STOMACH is so rare as a primary disease as to be a clinical curiosity. Secondary lesions are common in pulmonary tuberculosis. The signs of ulcer, the presence of the tubercle bacillus and the preëxisting disease in secondary cases make the diagnosis.

CONGENITAL STENOSIS OF THE STOMACH.—This interesting condition has been reported sufficiently often by reliable observers to entitle it to a definite place in clinical medicine. Its symptoms are vomiting, constipation, dilatation, increased peristalsis and, in most instances, palpable tumor representing the thickened pylorus. In some instances a tumor has not been felt and in a recent case (McCaw and Campbell) the obstruction was apparently due to redundancy of the mucous membrane with obstructing folds, a condition encountered, very rarely, in adults.

THE FECES

Lines of Investigation.—Careful inquiry should be made as to *antecedent diseases*, especially dysentery, typhoid fever and appendicitis, and in the case of the latter often necessitates a careful cross-examination covering the actual symptoms present in what may have been *misnamed* attacks.

As in disease of the stomach, the *habits* of the patient as regards smoking, drinking, meal hours, the *time consumed at meals*, *character of the diet*, and the *condition of the teeth* are important.

The character of the stools demands attention. Their consistence, size, number, form, quantity, color and the admixture of mucus or blood are the chief features.

The presence of constipation, diarrhea or pain and the relation of the two latter to meals or a fasting period should be thoroughly gone into. In making inquiry concerning constipation it is best to ask whether the bowels move

daily without medicine. In the case of diarrhea one should know whether it is nocturnal or diurnal and induced or increased by mental excitement or emotion. In certain forms the loose stool invariably follows a meal.

Finally it should never be forgotten that the stomach may be at the bottom of the intestinal disturbance. (Late achylia gastrica and chronic gastric catarrh, hyperchlorhydria, etc.)

Clinical Significance of Abnormal Findings.—*Absence of HCl and the gastric ferments* is suggested by defective conversion of gluten and connective tissue.

Pancreatic inadequacy leads to the imperfect conversion of both proteins and fats. *Absence of bile* will result in faulty digestion of the fats without of itself affecting protein conversions.

Normal Content.—Normally the stools consist of food remnants digested or undigested, fatty acids, epithelium, salts, mucus, bacteria, which constitute nearly one-third of the weight of the normal stool and represent 48 species, and traces of the digestive fluids.

The color depends upon the diet and is normally light brown or brownish yellow, because of the presence of hydrobilirubin, being darker on meat diet, light yellow on milk diet or in conditions where the usual bacterial decomposition processes are temporarily absent and bilirubin is present unchanged. Certain drugs, as iron and bismuth, produce a black stool; senna, rhubarb, santonin and calomel a greenish-yellow tint; certain fruits a reddish or black color. When bile is deficient a putty or clay-colored stool results as in obstruction of the common duct in gall-stones or catarrhal jaundice. A very similar stool due to a failure of fat absorption may be encountered in pancreatic disease or a tubercular peritonitis.

Form.—In prolonged constipation and in certain forms of chronic obstruction or atony, balls or other scybala may be encountered of various forms and sometimes grooved by intestinal folds. The "goat droppings" aggregations are encountered especially in spastic constipation. The pipe-stem or ribbon-like stools when persistent, suggest but do not prove organic obstruction in the terminal portion of the lower bowel and may also be present in spastic constipation or cases of channeled fecal impaction.

Suggestive types.

On the other hand, all grades of liquid or semi-liquid stools may be met with in the diarrheas; such as the so-called "pea-soup" stool of early typhoid or the brilliant ochre liquid of the later stage and the peculiar rice-water stools of true cholera and cholera nostras.

Food remnants consist chiefly of undigested vegetable matter; never, macroscopically, of meat, save in the serious intestinal diseases.

Blood from the lower bowel lies usually on the surface of the stool. It is usually red, if from the rectum, and even if darker and of higher origin in the bowel, is not usually coagulated.

In such cases "piles" or fissure should be looked for and, if absent, attention is at once demanded by the segments higher up; carcinoma, polypi, or ulcer, being suggested.

Blood from the stomach or upper bowel is incorporated with the stool

and usually presents a "*tarry*" appearance but it should be remembered that in diarrhea these distinctions of color may in part be lost.

Mucus may appear as a membrane (mucous colitis or enteritis); as an envelope, covering the stools; in long strings or spawn-like masses; or intimately mixed with the feces (catarrhal enteritis).

Pus if present proves a lesion of the lower bowel and fragments of tumor may yield information of value. Intestinal parasites are considered elsewhere.

Collecting the Specimen.—The simplest method consists in the use of one of the covered receptacles which can be placed within and rest upon the basin of a water-closet or directly under the patient lying in bed. These can be procured with a removable wire screen which is useful in washing the stools for concretions. As an emergency measure any receptacle may be used and for a sieve one may employ ordinary mosquito screen fastened to an extemporized hoop. Sieves.

Microscopic Examination.—The portion to be examined is prepared by adding a small amount of physiologic salt solution and a few drops of 1 per cent. formalin solution.

If one has excluded or completed the examination of living organisms turpentine may be used to cover the specimen under the microscope and thus avoid odor.

Normally the appearance varies with the diet. In ordinary mixed diet there are various vegetable cells, usually characteristic; free starch granules are, normally, absent, but if present are readily demonstrated by a drop of Lugol's solution. Their presence is pathologic for, normally, only starch enveloped in cellulose should be present.

Meat fibers in small numbers may be recognized, as may elastic and connective-tissue fibers, and indicate impaired or absent gastric digestive agents. Oxalate of lime and fatty acid, calcium carbonate and calcium and ammonio-magnesium phosphate may be present in characteristic crystals.

The Charcot-Leyden crystals occur in pulmonary tuberculosis, typhoid fever, dysentery, and with intestinal parasites. Ruby-red or orange-yellow rhombic plates of hematoidin, or its amorphous forms, may be present in hemorrhage or severe catarrhs.

Fat may also be present as characteristic highly refracting globules, and both fat and fatty acid crystals are increased in diseases of the liver or pancreas, and in acute enteritis.

Blood cells may be present if the bleeding originates in the lower bowel, and pus corpuscles if there be an ulcerative process or discharging abscess.

Tumor fragments, mucin threads, and the large variety of bacteria may be found, most important of which pathologically are the Boas-Oppler bacillus, the colon bacillus, and the tubercle bacillus.

The Boas-Oppler bacilli are extremely plentiful in the stools of most patients suffering from established stenotic gastric carcinoma, a fact of value when a patient is not fit for the use of the stomach tube.*

* Bassler also points out the value of a smear taken from the back of the tongue in such cases as have been vomiting.

SCHMIDT'S METHOD.—A formal and extended examination of the feces is ordinarily too exacting and time-consuming for the general practitioner, but one of the simplest methods is here given for the sake of completeness.

Test Diet.—This, as given by Schmidt, outlines the following procedure:

In the morning: 0.5 liter milk [or, if milk does not agree, 0.5 liter cocoa (prepared from 20 Gm. cocoa powder, 10 Gm. sugar, 400 Gm. water, and 100 Gm. milk)], to this 50 Gm. zwieback.

In the forenoon: 0.5 liter oatmeal gruel [made from 40 Gm. oatmeal, 10 Gm. butter, 200 Gm. milk, 300 Gm. water, 1 egg (strained)].

At noon: 125 Gm. chopped beef (raw weight) broiled rare, with 20 Gm. of butter, so that the interior will still remain raw, to this 250 Gm. potato broth (made of 190 Gm. mashed potatoes, 100 Gm. milk, and 10 Gm. butter).

In the afternoon: as in the morning.

In the evening: as in the forenoon.

This diet thus consists of: 1.5 liters milk, 100 Gm. zwieback, 2 eggs, 50 Gm. butter, 125 Gm. beef, 190 Gm. potatoes, and, as gruel, 80 Gm. oatmeal. It contains about: 102 Gm. albumin, 111 Gm. fat, 191 Gm. carbohydrates, or a total of 2234 calories (raw calories). The test is generally given for three days, until a stool is obtained which represents the test diet.

Steele's Method.—*Breakfast.*—Two eggs, one-third of the amount of toast and butter, two glasses of milk, oatmeal with milk and sugar. *Dinner.* The steak and potatoes, one-third of the amount of toast and butter, one and one-half glasses milk. *Supper.* Two glasses of milk, remainder of toast and butter, one or two eggs if desired.

Initiation of Tests.—These test meals need be given for only two or three days, a capsule (0.3) carmin being given with the first meal. The appearance of the dye in the stool determines the beginning of the examination and serves to measure the time of passage.

One then proceeds to determine consistence, color, and odor and reduces the feces to a liquid form in a mortar with distilled water. The ordinary blacked plate or a Petri dish placed over a black background then facilitates the examination.

Macroscopic Findings.—*Normally* there should be nothing except the indigestible oatmeal hulls and sago-like particles of potato.

Pathologically one may find: (*a*) *Mucus* appearing as glassy flakes sometimes stained yellow. (*b*) *Remnants of potato* the resemblance of which to mucus may require the microscope for differentiation. (*c*) *Remnants of muscle fiber* indicating impaired intestinal ferments, absence of enzyme (enterokinase) or abnormal motility. (*d*) *Crystals of magnesium and ammonium phosphates indicating fermentation.* (*e*) *Connective-tissue remnants* differentiated from mucus by their yellowish-white color and their toughness. (*f*) *Pus or blood.* (*g*) *Parasites.* (*h*) *Concretions.* (*i*) *Foreign bodies.*

Microscopic Examination.—*The microscopic examination merely confirms and somewhat amplifies the findings mentioned in the foregoing paragraph.* It is recommended that three special mounts be made: (1) A drop of the

prepared material. (2) Same + acetic acid. (3) Same as No. 1 + a drop of dilute Lugol's sol. (iodin 1, potas. iodide 2, water 50).

No. 2 is heated to boiling before covering and upon cooling will show fatty acid crystals set free by the acetic acid; if reheated these will form droplets. No. 3 shows the violet-blue reaction of potato cells and certain spores.

Chemical Examination.—The chemical examination consists of three routine tests: (1) *The reaction*, obtained by dropping the prepared material into a few cubic centimeters of dilute aqueous litmus solution.

(2) *The sublimate test.* (Equal parts of the prepared material and a sol. of mercuric chloride (25 per cent.) should strike a pinkish-red color indicating hydrobilirubin.) Unchanged bile pigment yields a green color and is pathologic.

(3) *The fermentation test.* Five cubic centimeters of formed feces, or its equivalent if liquid, are prepared with sterile water and poured into the bottle of Strasburger's instrument or the simple and easily cleaned modification of Steele which can be made by anyone.*

The tube surmounting the bottle is filled with water, the parallel tube remaining empty. If kept at blood heat for twenty-four hours, gas will rise and displace the water in the first tube, forcing it into the outer parallel tube whose air finds an outlet through the longer inner tube; thus the amount of water displaced is a measure of the amount of gas formed. One-third displacement is distinctly pathological. Albuminous putrefaction is indicated by a foul smell and alkaline reaction, carbohydrate fermentation by acidity.

Inferences from the Tests.—*Color.*—A pink color reaction in the case of the sublimate test (test 2) is normal; the green indicates increased motility; absence of any color, a fat stool or absence of bile.

Meat Remnants.—Whether of muscle or connective tissue and even if microscopic, an excess of either is pathologic. In the case of connective tissue, deficient gastric digestion is indicated or excessive gastric motility. In the case of the muscle the digestive trouble is probably intestinal.

An excess of fat merely indicates deficient digestion of that material, but suggests hepatic, pancreatic or intestinal disease; fermentation means either poor starch digestion or intestinal disturbance.

Albumin fermentation, indicates either deficient gastric or intestinal digestion or increased motility.

Value of Results Obtained.—*As a matter of fact it may be stated, with regret, that up to the present time the amount of information gained is too slight to compensate the busy practitioner for even the small amount of time necessarily consumed.* Nothing but constant practice will suffice to establish proper

* The materials needed are a large-mouthed medium-sized bottle carrying a rubber cork, perforated by a glass tube. Two test-tubes are prepared each having a rubber cork with two perforations. Both are inverted and connected by a U-tube the extremities of which project slightly above the inner surface of the cork, one is then pressed down over the vertically projecting tube of the bottle and holds the other tube parallel with it by virtue of the connecting U-tube. Test-tube No. 2 is fitted with a small glass tube which goes nearly to the top and projects below.

standards representing the normal and the abnormal and, finally, proteid fermentation is more easily estimated by testing for indoxyl.

Concretions.—*Enteroliths.*—Enteroliths originate usually in the small intestines, are small, light in color and ordinarily of no importance, consisting chiefly of magnesia and lime.

Coproliths.—These hard sausage-shaped fecal concretions are most frequent in the cecum, sacculations and rectum, rarely they attain considerable size and may cause obstruction.

Pancreatic Calculi.—These may be faceted, but are usually rough and friable; they contain no bile pigment and cholesterin, and are soluble in chloroform.

Biliary Calculi.—These occur as well-known faceted bodies, or, if in the form of sand, may require chemical tests for their identification.*

The cases of "intestinal sand" (supposedly associated with neurotic states and mucous colitis) which have been observed by the author have almost invariably proven to be associated with gall-stones.

HEMORRHOIDS (*Piles*).—This common condition, rare in children, most frequent in men, consists of a diffuse or circumscribed varicosity of the hemorrhoidal veins, either in the lower portion of the rectum (submucous) or at the anal margin (subcutaneous).

Site.

Etiology.

The condition is induced by constipation, continuous standing or sitting (*i.e.*, sedentary occupations), the habitual use of cathartics, pregnancy, the pressure of tumors, or, obstructed portal circulation from whatever cause, and is favored by the lack of valves in the hemorrhoidal veins.

Source of error.

They are frequently a source of recurrent hemorrhage which in turn may be the actual cause of an obscure anemia, often of a most severe type. Furthermore, the frequency of the lesion may lead to the too ready acceptance of the patient's own diagnosis and a failure to search for and detect the existence of rectal fissure, foreign body, malignant growth, tuberculous or syphilitic ulceration or fistula.

The rectal examination is too often neglected, though its disagreeable features are in part removed by the use of the rubber glove or finger shield.

Appearance.

Hemorrhoids are easily recognized if superficial, the patient being asked to strain and bear down, when the reddish-blue nodules will become evident. Skin tags and condylomata are common but easily differentiated. Internal hemorrhoids may require the use of the speculum, though usually recognized by digital examination. Retained fecal matter has a characteristic feel and

Fecal masses.

malignant growths are peculiarly indurated and firm.

In any rectal examination the tonus, *i.e.*, the resistance and grasp of the

* *Test.*—Powder the stone or gravel, add 20 c.c. of ether, mix thoroughly, filter, and evaporate filtrate, divide residue in three portions. No. 1 is dissolved in hot alcohol, allowed to evaporate without heat and the residue examined for the rhomboid crystals of cholesterin. No. 2 is treated with HCl and a trace of ferric chloride; a blue color on evaporation indicating cholesterin. No. 3 is placed on a slide, treated with a drop of concentrated sulphuric acid and covered. The cholesterin crystals show carmine margins. Another portion may be treated with dilute HCl, heated, cooled and treated with chloroform; to the chloroform extract Gmelin's test for bile pigment is applied.

anal sphincter, should be noted. It is markedly relaxed in certain organic nervous diseases and in obstruction of the rectum and sigmoid flexure. Tonus.

MECKEL'S DIVERTICULUM.—Persistence of the omphalomesenteric duct may be of slight consequence unless its extremity is fixed by adhesions or remains attached at the navel.

Either acute or chronic intestinal obstruction may result in the latter instances. Rarely it may be involved in or imitate an intussusception or volvulus or become the seat of abscess and perforation.

ENTERITIS

Primary Divisions.—*In the diarrheal diseases one must distinguish those affecting the small intestine alone from those involving the colon exclusively or in part.*

The latter, more severe and likely to be attended by serious complications, are described under "dysentery" and "enterocolitis; nearly all forms of *enteritis* are trivial and temporary in the case of the adults, but may assume considerable importance in the case of infants and young children, occurring usually under conditions of improper sanitation or still more frequently in *artificially or badly* fed infants. Enteritis vs. Colitis. Variable severity.

Bowel disturbances in breast-fed infants are rare and usually temporary and trivial unless secondary to some grave primary condition. All varieties are prone to appear during hot weather, particularly when extreme variations in temperature and humidity are present, and reach their maximum in prevalence and mortality in July and August. A common ailment.

Milk and water are probably the chief vehicles of contagion, but the demonstration of a specific bacillus for the ordinary diarrheal diseases must await further investigation, inasmuch as a large variety of organisms are found, many of which are quite competent in themselves to produce decided symptoms or to act with and intensify the virulence of others.*

CLINICAL VARIETIES.—We recognize: (1) **Acute Intestinal Indigestion.** (2) **Acute Fermentative Diarrhea.** (3) **Cholera Infantum.**

Morbid Anatomy.—The pathologic changes in enteritis vary from a mere congestion or catarrhal inflammation of the mucosa, with slight infiltration of the submucosa and enlargement of the lymph follicles, to actual ulceration. Ulcerated areas are ordinarily limited, originating at the follicle but extending by the coalescence of contiguous ulcers. In combined lesions of the upper and lower bowel one may of course have all the changes described under "dysentery." In the more severe types, particularly the combined form, complicating broncho-pneumonia is common and the other viscera present evidence of severe toxemia.

* Much interest attaches to a bacillus recently discovered by Duval and Bassett which closely resembles Shiga's bacillus of dysentery and has been found in a large number of cases of the summer diarrheas of children. Certain normally harmless bacteria seem to be capable of assuming virulent characteristics. Bacillus dysenteriæ, the colon bacillus and, in the small intestine especially, the bacterium lactis aërogenes, may be active.

Acute Intestinal Indigestion.—Colicky pain, tympanites, and diarrhea may come on suddenly, immediately or from several hours to a day after some dietetic indiscretion or, without apparent cause;* the fever is usually moderate and of brief duration, the pulse rate is increased and in the child may be extremely rapid. The stools are at first fecal, then watery and contain mucus and food particles imperfectly digested. Such an attack is ordinarily cut short by the administration of some appropriate cathartic, such as castor oil.

Stools.

Severer type.

Acute Fermentative Diarrhea.—This is characterized by *more marked symptoms* throughout and may come on suddenly or succeed a milder attack of acute intestinal indigestion. Vomiting is usually present, the fever may reach 105°F., the pulse is rapid, nervous symptoms are pronounced, exhaustion marked and, in children, convulsions are not uncommon at the onset. The stools are more frequent than in the simple form, rapidly become watery, are green in color and contain much mucus. The disease is ordinarily of brief duration and favorable termination, but may prove the commencement of an ileo-colitis and may terminate fatally by coma, exhaustion or broncho-pneumonia.

Green stools.

Chronic Enteritis.—This hardly justifies a separate description, being characterized by intermittent or persistent symptoms of a mild sort which mirror the acute attacks. If long-continued it impairs strength and nutrition and it may be associated with mild daily diarrhea, mere unformed stools, or obstinate or alternating constipation.

Reflects primary disease.

In every such instance the condition of the *stomach*, duodenum, appendix, and gall bladder should be investigated thoroughly and the stools carefully examined.

CHOLERA INFANTUM.—This ailment, remarkably fatal and prevalent in the large cities among the poor, may affect the children of any age and is both sporadic and endemic. The onset is usually sudden, with high temperature.† Vomiting is usually present, and the stools, at first fetid, may reach 20 or more daily and rapidly become watery, light yellow or greenish, then colorless and odorless. There is marked thirst, the urine is scant and often albuminous and the disease usually terminates fatally, often with delirium, stupor or coma and perhaps convulsions. Marked tympanites is not present nor is the abdomen tender, but there is evidence of profound collapse, the features being pinched, the skin ashy-gray and the surface usually cold even though rectal temperature may be high.

High mortality.

Violent choleraic symptoms.

Ileo-colitis is predominatingly a dysentery and will be described under that heading; the small intestines seem to be primarily involved, but symptoms very soon become distinctly dysenteric.

MEMBRANOUS ENTERITIS (*Membranous Diarrhea, Mucous Colic*).—**Definition.**—An affection characterized by the presence of mucus, in quantity, in the feces.

Etiology.—More common in women than in men, this affection long has

* These attacks are frequently of *gastric origin.*

† This must be taken by rectum or vagina because of low surface temperature.

been regarded as largely dependent upon the elements of hysteria and functional nervous states. It may nevertheless be associated with chronic appendicitis, gastric ulcer, and gallbladder disease, but is a relatively rare condition, most frequent in connection with an enteroptosis or achylia gastrica.

According to the author's experience this condition is associated with two conditions aside from the causes mentioned, which may or may not co-exist. *These conditions are impaired general nutrition and spastic constipation.*

As spastic constipation demands an entirely different line of treatment from that usually adopted in simple constipation and is very often quite overlooked, the condition usually goes unrelieved by medical means.

Symptoms.—Following an attack of obstinate constipation, violent, colicky pains occur associated oftentimes with diarrhea, perhaps of a dysenteric type, and more or less decided dyspeptic symptoms. Mucous masses are discharged which are usually grayish-white, ribbon-like or membranous, rarely forming complete molds of the intestinal canal (tubular form) or occurring in masses like frog spawn. Their nature may be proven by Pariser's method.*

DYSENTERY

Definition.—A term applied to a group of diseases characterized by acute or chronic inflammation and ulceration of the lower bowel, diarrheal stools, containing blood and mucus, and associated with colicky pain and tenesmus.

Etiology.—The disease may be primary or secondary, acute or chronic. The secondary cases depend upon a primary cause, such as tuberculosis, syphilis or Bright's disease. The primary cases fall under three heads: (1) *Amebic dysentery.* (2) *Acute specific dysentery* (*tropical dysentery*). (3) *Acute catarrhal dysentery* (*acute ileo-colitis*).

The Cause of Amebic Dysentery.—The ameba dysenteriæ now known to be the *Entameba tetragena*, representing actually, merely the end stage of *Entameba histolytica*, was described first by Lösch in 1875 and first identified in hepatic abscess by Kartulis (1887), but to Shaudinn's description (1903) and careful work we owe most of our knowledge of the characteristics of the specific organism. It is from 15 to 20 μ in diameter, *i.e.*, about twice the diameter of the red blood cell. See Fig. 404.

Entameba histolytica (tetragena) causes from one-third to one-half of all cases of tropical dysentery, and if a warm slide is used for examination the amebæ may be readily identified by their structure and movement and are usually contained in the flakes of mucus or pus of the fecal discharges or may be obtained from abscess or stained *in situ* in tissues.† Amebæ readily recognized.

The description of the methods of "laboratory diagnosis," immediately following is taken from Stitt's admirable little book on "Tropical Diseases."

Laboratory Diagnosis.—In the fresh specimen of the milky mucopurulent mass of bacillary dysentery one observes large numbers of pus cells and par-

* They are treated with sublimate alcohol and then yield a green color with Ehrlich's triacid stain, in contrast to fibrin which stains red.

† Immunity to the pathogenic entamebae is pronounced in many individuals, only the lesser proportion of individuals carrying them being seriously affected.

ticularly very large phagocytic cells which greatly resemble amebæ. Upon staining with Gram's stain one may find numerous Gram negative bacilli in the cystoplasm of the cell.

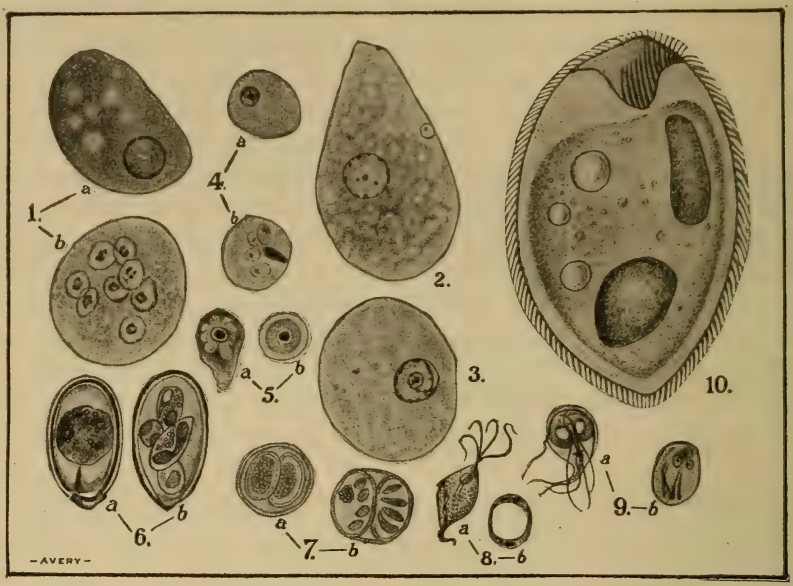


FIG. 473.—Important pathogenic protozoa of the intestinal tract. (1a) Motile *E. coli*. Note large amount and peripheral arrangement of chromatin in nucleus. (1b) Encysted *E. coli*. Note larger size than *E. histolytica* cyst, its 8-ring form nuclei and absence of chromidial bodies. (2) Motile *E. histolytica* from acute dysenteric stool. Note histolytica nucleus with scanty chromatin. (3) Tetragena type of *E. histolytica* from case of chronic dysentery. Note greater amount of chromatin and central karyosome with centriole. (4a) Preëncysted *E. histolytica* from carrier. Note small size and heavy peripheral ring of chromatin in nucleus making this feature of chromatin in nucleus similar to the larger *E. coli*. (4b) Encysted *E. histolytica* from dysentery convalescent. Note small size, 4-ring nuclei and a dark chromatin staining mass, "chromidial body." (5a and 5b) Motile and encysted cultural amebæ from Manila water supply. (6a and 6b) Oocyst and sporozoite production in 4 spores of *Eimeria stiedæ*. (7a and 7b) Oöcyst with 2 sporoblasts and oöcyst with 2 spores containing 4 sporozoites of *Isospora bigemina*. (8a and 8b) Vegetative and encysted *Trichomonas intestinalis*. (9a and 9b) Vegetative and encysted *Lamblia intestinalis*. (10) *Balantidium coli*. Illustrations of amebæ from Walker—others from Doflein. (*Stitt.*)

"The large cells which resemble amebæ are often vacuolated, thus intensifying the similarity. They are nonmotile, however, and do not show the small ring nucleus which is so characteristic of the vegetative human amebæ. The nucleus of the confusing cells is also larger, approximating one fourth the size of the cell.

"For bringing out the nuclear characteristics of human amebæ Walker recommends fixation of thin moist smears in sublimate alcohol (absolute alcohol 1 part, sat. aq.sol. bichloride 2 parts) for 10 to 15 minutes. These smears are then well washed with water and stained with alum hæmatoxylin for five minutes. The nuclear characteristics are noted under etiology. With vegetative amebæ I have obtained beautiful results with vital staining which can best be done by tinging the feces emulsion with a 1 per cent. aqueous solution of neutral red. I have also had good results by emulsifying the feces in a drop of 1 or 2 per cent. formalin and then adding a drop of 2 per cent. acetic acid. The mixture is then tinged with either neutral red or methyl green.

"For distinguishing the encysted form of *Entamoeba coli* one can obtain beautiful results by emulsifying the feces in Gram's iodin solution. Owing to the glycogenic reaction given by *E. coli*, the round amebæ, with its 8 nuclei, stands out very distinctly."

"For diagnosing the four-nucleated cyst of the pathogenic ameba one gets better results with hæmatoxylin as this brings out not only the four nuclei but the chromidial bodies as well. It was formerly customary to recommend the administration of salts prior to examining for amebæ. Walker warns that such a procedure gives us amebæ which are difficult to differentiate, the nuclear characteristics of *E. coli* and the tetragena nucleus of *E. histolytica* being much alike as they both contain much chromatin. In a dysenteric stool the histolytica type of nucleus, containing but little chromatin,does not resemble the nucleus of *E. coli*."

WALKER'S DIFFERENTIAL TABLE

Motile Stage

A. Entameba histolytica	B. Entameba coli
1. Appearance hyaline.	1. Appearance porcelaneous.
2. Refractiveness more feeble.	2. Refractiveness more pronounced.
3. Movements active in the fresh stool.	3. Movements sluggish.
4. Nucleus more or less indistinct.	4. Nucleus distinct.
5. Chromatin of nucleus scanty.	5. Chromatin of nucleus abundant.

Encysted Stage

A. Entameba histolytica	B. Entameba coli
1. Cyst smaller.	1. Cyst larger.
2. Cyst less refractive.	2. Cyst more refractive.
3. Cyst usually contains elongated refractive bodies known as "chromidial bodies."	3. Cysts do not contain "chromidial bodies."
4. Nuclei never more than 4.	4. Nuclei 8, occasionally more.
5. Cyst wall thinner.	5. Cyst wall thicker.

The recent experiments of E. L. Walker and A. W. Sellards have shown that amebæ cultivated from "water or other non-parasitic sources" or, *Entameba coli*, are non-pathogenic.

The former do not appear in the stools at all subsequent to feeding. The latter may be recoverable from the feces over long periods but produce no symptoms.

Walker's individual experiments with *Entameba hemolytica* from normal stools of *carriers* showed that parasitization was readily produced, 17 of 20 volunteers showing parasites within an average period of nine days.

One of the three remaining was parasitized by three feedings. In the two others no further feedings were administered.

Only four of the eighteen parasitized men developed dysentery, after periods of 20, 57, 87 and 95 days.

It would appear that entamebic dysentery is conveyed chiefly through the encysted forms from the stools of healthy or convalescent carriers. The motile forms of active dysentery are not highly infective, and are but slightly resistant.

Acute Specific Dysentery (*"Acute Tropical Dysentery"*).—Practically all tropical dysenteries not due to *Entameba histolytica* are caused by Shiga's bacillus which produces the disease in animals by inoculation and is agglutinated by the blood serum of affected patients.

Acute Catarrhal Dysentery (*Acute Ileo-colitis, Follicular Dysentery*).—This is the ordinary well-known form of dysentery encountered in the temperate zone. No specific germs have been isolated but a large variety have been described as associated with the process.

Diphtheritic Dysentery.—This is characterized by diphtheritic inflammation with extensive infiltration and formation of sloughs. It is often secondary to exhausting chronic diseases, such as chronic nephritis, heart disease and malaria.

General type.

SYMPTOMATOLOGY OF DYSENTERY.—This is essentially the same for all varieties though varying greatly in degree. Any form may become chronic, tropical forms are both endemic and epidemic, and for their development all forms of primary dysentery elect hot weather. *It will be noted that the symptoms are precisely those that might be expected to follow inflammation of the lower bowel plus widely varying degrees of toxemia.*

The amebic form is essentially chronic and recurrent, although acute cases occasionally are encountered.

The onset *is sudden and violent* in all bacillary tropical forms, being often associated with chill, whereas the milder variety is often preceded by an initial diarrhea. Abdominal distress and fever are likely to be high during the active stage of all forms, and febrile phenomena are marked.

Essential symptoms: *Purging, marked tenesmus, severe colicky pains, constant desire to go to stool, excessive thirst, great prostration, and tenderness of varying degree over the descending colon are symptoms common to all forms.*

The stools: These contain mucus, blood and scybala, and in the severer forms pus and sloughs. The number of stools varies greatly, but may reach 100 or more in the twenty-four hours. They tend to become fetid and sanious in severe and unrelieved cases.

Emaciation and exhaustion.

The exhaustion is great, yet vomiting and gastric irritation are often absent. Emaciation is of course rapid and in severe cases extreme.

Ileo-colitis is usually preceded by a short period of ordinary diarrheal stools and it must be remembered that in children mucus and blood may appear in any severe diarrhea.

Often malarial.

Chronic Dysentery.—Chronic indigestion, recurrent dysenteric attacks of varying degrees of severity and marked emaciation are the chief features in chronic cases. Many of these have been found to be due to the persistence of a malaria in soldiers returning from the tropics, many more to the persistence of the *entameba histolytica* (*tetragena*) but any form occasionally may

Usually amebic.

become chronic and the irritability of the lower intestinal tract may last for a few weeks or months or even for many years.

Twenty per cent. of the amebic form develop abscess of the liver.

Differential Diagnosis.—The peculiar character of the diarrhea with its bloody stools and *marked tenesmus* serves to distinguish the acute form. Entameba should be searched for in all dysenteries and the specific agglutination reaction of the Shiga bacillus, the forms of Flexner and Strong or the "y" strain of Hiss and Russell, sought with known immune sera when such are obtainable.

The serum of an active case possesses but slight agglutinating power until ten or twelve days have elapsed, too late to be of much value in diagnosis.

Typhoid fever, of the rare dysenteric type, is readily differentiated by its gradual onset, diazo-reaction, rose spots, temperature curve and finally and conclusively by the agglutination test with the typhoid germ. As it ordinarily wholly lacks the characteristic dysenteric phenomena, confusion can seldom arise.

Tuberculous or malignant ulcers and *foreign bodies* in the rectum may cause attacks more or less closely simulating chronic dysentery, but the associated lesion and the results of local and microscopic examination will ordinarily distinguish them.

Mortality and General Comment.—The death rate is of course highest in the severe tropical forms but dysentery of the temperate zone is seldom fatal except in children and old people or persons exhausted by disease.

On the other hand, the total mortality in chronic dysentery under conditions of poor food, overcrowding and general insanitation may be very heavy. The United States government is still paying a large number of persons for disability attributed to chronic dysentery of the Civil war, and the *deaths* during that four-year period reached an enormous total. The mortality in Japan for the decade following the first introduction of the disease reached 247,000.

MISCELLANEOUS INTESTINAL NEUROSES

Rectal spasm is almost invariably secondary to inflammatory or ulcerative processes either of the rectum itself, the colon, the anus or neighboring organs. Rarely it is supposed to be primary. Defecation is intensely painful, the anus sensitive and resistant, an anesthetic being required for an examination.

Peristaltic unrest associated with borborygmi may be primary or secondary. The former is a pure neurosis, the latter may be associated with various gastric disturbances or with actual obstruction. The condition is usually accompanied by constipation and in its primary and severe form by hysteria, psychasthenia or hypochondria. It is, as a rule, trivial and unimportant.

Asthenic women chiefly.

Meteorism.—Gaseous distention is a mere symptom too well known to require extensive description and results chiefly from "air swallowing" or the fermentation of carbohydrates or proteids, is usually temporary and purely

Sometimes serious.

neurotic, but is also an important manifestation in true obstruction and peritonitis. It may prove a troublesome and dangerous condition in the typhoid state or any virulent infection, and is particularly dangerous in pneumonia and chronic incompensated disease of the heart.

Enteralgia has more basis in fact than gastralgia, being descriptive of ordinary colicky pain of intestinal origin, whether it be primary or secondary.

Hypogastric Neuralgia.—This term has been applied to a painful condition localized in the epigastrium and lower part of the back and associated with sense of pressure in the rectum, bladder, uterus and vagina. It is purely a symptom, undoubtedly dependent upon uterine, ovarian, hemorrhoidal and other similar conditions.

Hyperesthesia, Paresthesia, Anesthesia.—The intestines share with other portions of the body these symptoms of hysteria or psychasthenia and they not infrequently take the form of aura in epilepsy. They are important only to the possessor of them save in actual disease of the brain and cord when rectal anesthesia may be associated with sphincter paralysis, or in the cases where the anesthesia leads to a serious accumulation of fecal masses.

Intestinal neurasthenia is given a place by most authors, but deserves no such distinction. The term really applies to the asthenic-psychasthenic state with predominance of abdominal symptoms.

Paralysis of the Intestines.—This has already been referred to elsewhere. The condition may be a very serious one and presents many of the symptoms of obstruction.

It is associated with direct abdominal trauma, sepsis, chronic inflammation and ulcerative diseases of the intestinal tract, with profound hysteria, melancholia, hypochondria or actual organic disease of the brain and cord and is frequently observed in prolonged and *massive* fecal impaction.

CHRONIC INTESTINAL OBSTRUCTION.—Acute obstruction has been dealt with already. Chronic obstruction may be due to the same factors and one is chiefly concerned with its recognition.

Often obscure.

Symptoms.—These are extremely variable and often obscure. The history of previous ailments is important as is the history of constipation, diarrhea, or symptoms suggesting possible ulceration or appendicitis. Little stress should be laid upon the pipe-stem or tape-like conformation of the stools as these may be met with in spastic constipation or anal spasm.

The gradual onset of the symptoms is usually characteristic. At first hardly noticeable, they may become severe and troublesome and always depend greatly upon the site of the obstruction.

Regional variations.

Duodenal stenosis produces symptoms strongly resembling pyloric obstruction and is frequently associated with the vomiting of bile in quantity, thus indicating a stricture below the papilla of Vater.

Any marked obstruction in the *small intestine* will be accompanied by a great increase of indoxyl, and the more remote it is from the stomach the more will the colicky pains and constipation predominate over the gastric symptoms, nausea and vomiting.

If in the *lower ileum or colon*, there may be no gastric symptoms. Local

distention of the abdomen or visible peristalsis may give a clue to the site, and gurgling and bubbling sounds may be audible and palpable with or without the pressure of the hand. It may be possible to recognize the large peristaltic waves of the colon as compared with those of the lesser intestinal coils, but it is evident that this sign must often fail.

Chronic intussusception is usually associated with palpable tumor, bloody stools and tenesmus as in the acute form, its most frequent site being at the ileo-cecal valve. Old appendiceal adhesions may produce chronic obstruction in the same region, but lack all distinctive symptoms of intussusception. In this connection it may be said that following appendicitis, adhesions may occur about the cecum or the ascending portion of the colon producing obstinate constipation and frequent attacks of more or less extreme pain, though the appendix is found at operation practically normal and free.

Tumor and bloody stools.

Adhesions after appendicitis.

Lane's Kink.—Sir Arbuthnot Lane lays extraordinary stress upon the presence of accessory peritoneal bands which may produce "kinks" of the colon at any one of the three flexures or similarly affect the lower ileum or even the pylorus.

That they possess anything approximating the importance or frequency of occurrence ascribed to them, or that they often demand or justify the radical surgery advised may well be doubted.*

Doubtless, cases arise in which persistent symptoms of duodeno-jejunal block occur which may demand surgical interference, rather than lying down after meals, proper support to the abdominal viscera, or a few days or weeks devoted to rest and efforts to improve nutrition, but the tendency to turn too readily to extreme surgical measures is unfortunate.

A surprisingly large number of cases presenting both subjective and fluoroscopic evidences of such a condition wholly and completely recover without surgical intervention, or, prove phantom findings (George and Leonard). Furthermore, these cases occur chiefly in the congenitally asthenic visceroptotic cases which, as most surgeons now have decided, are bad subjects for any operative procedure.

CONSTIPATION.—This common and often troublesome symptom may be caused: (*a*) *By the character of the food taken*, being more common in those taking an exclusive meat diet than in vegetarians. (*b*) *Disturbances of innervation*, more frequently than from any other cause. It is seen in hysteria, neurasthenia, brain injuries, simple neglect or lack of proper habit, injuries and disease processes affecting the peritoneum, lead poisoning, nervous dyspepsia, hyperchlorhydria, etc., etc. (*c*) *Mechanical causes*, among which are weak abdominal musculature, strictures, growths and the recumbent

Etiology.

* The extreme views advanced, and the tremendously radical surgical measures undertaken, represent at once the revival and the maximal exaggeration of the most extreme views of Glenard and Bouchard.

The author notes with gratification the reaction against this operative procedure is growing rapidly and, indeed, has been emphatically expressed by one of the most brilliant and ultraradical of American surgeons.

posture. (*d*) *Reflex causes* and direct irritation, as in fissure of the anus, hemorrhoids, rectal ulcer, prostatitis, and displacement of the uterus or ovaries. (*e*) *Fever* and other conditions associated with profound toxemia, diminished secretion or excessive abstraction of fluids. (*f*) *Chronic disease of the stomach, colon or small intestine.*

To an astonishing degree so-called "constipation" depends upon the failure of the individual to go to stool regularly, regardless of the absence of the usual suggestive or impelling "call," such signal being readily diminished or wholly lost save under intensive stimuli in victims of the vicious cathartic habit.

Another factor of primary importance is the position assumed. In every instance of supposed constipation the patient should be instructed to bring the knees together *and lean as far forward as possible when straining.* Actual atony of the bowel is a relatively unusual factor, as compared with a weak and unreënforced abdominal musculature.

Clinical Divisions.—One distinguishes an *atonic and a spastic form*, the former representing either a weak intestinal musculature or impairment of its nervous mechanism. This is seen in chronic venous congestion (chronic heart disease, hepatic cirrhosis, etc.) or in states of profound general debility, asthenia, hysteria or certain organic nervous ailments.

Atonic.

Spastic constipation is characterized by permanent increased tonus of the intestines and the rectal segment. The spasm may be of variable duration and involve one or many intestinal segments in varying degree. If the whole small intestine is affected, a scaphoid abdomen is produced as is seen in spinal meningitis or in certain irritative cerebral lesions. More frequently the colon is involved and yields no symptoms on inspection save that the stools resemble round hard balls, often like goat droppings, or the pipe-stem or tape-like forms. One somewhat characteristic symptom of spastic constipation is intermittent pain in the left lower abdominal segment relieved by stool or enema. This at times is very severe and constitutes a type of colon colic.

Spastic.

Stools.

Colic.

FECAL ACCUMULATION is most common in persons suffering from profound toxemia in the acute infectious diseases, such as typhoid, in the insane, or in profoundly hysteric or neurasthenic individuals. Careless physicians, house officers or nurses encounter them in their patients with especial frequency. They are sometimes associated with profound autointoxication, partial or complete intestinal paralysis and acute gastric dilatation. Such masses frequently produce neuralgic symptoms or even a sciatica and, if the condition be of long duration, the utmost difficulty may be encountered in attempting to relive it by enema or by physic.

Etiology.

Hospital cases.

Various sequelæ

Rectal obstruction is easily diagnosed by the finger and speculum. It may take the form of irregularity, greatly diminished frequency, or a mere diminution in quantity, the fecal passages being chronically insufficient. In fecal impaction and indeed in almost all forms of constipation palpation of the rectum or even the relaxed abdominal wall is sufficient for the detection of fecal masses. If actual obstruction occurs from this cause ileus is closely

Readily detected.

simulated, otherwise the symptoms are too well known to require description. (See also remarks on "Abdominal Tumors.")

THROMBOSIS AND EMBOLISM.—This rare condition results usually from a clot in the left auricle in cases of mitral disease, thrombosis being the common form and the *superior mesenteric* the almost invariable site. The condition can seldom be recognized ante-mortem. Septic emboli carried into the *smaller branches of the intestinal arteries* from an endarteritic or septic focus also may cause infarction and produce the same severe colicky pain, localized tenderness, abdominal distension and bloody diarrhea.

Site.

Obscure.

TUBERCULOSIS OF THE INTESTINES.—This is rarely primary, but a common secondary manifestation, occurring in a large percentage of the advanced pulmonary cases. It affects chiefly the lower portion of the ileum and may extend downward even to the rectum or to a variable extent upward. The ulcers are irregular in shape with unclean bases and bright red margins which are usually undermined or overlapping. They readily form adhesions and hence seldom perforate. Owing to their tendency to encircle the intestine they may in rare instances heal and produce cicatricial stenosis. Diagnosis is evident from the pus, blood and tubercle bacilli in the stools, associated with localized pain and tenderness, and, usually, a preëxistent known tuberculosis.

Ulcers.

SYPHILIS OF THE INTESTINES.—Syphilitic ulcers formed by the softening of gummata occur, congenitally, in the small intestines, but in the acquired form, chiefly in the colon, rectum and anus. They present a bacon-like appearance, with well-defined margins and an indurated base. Ulcers are occasionally observed in secondary syphilis and a form of ulceration of indefinite causation termed "toxic" occurs occasionally in connection with the cachexias of leukemia, scurvy, nephritis and the like.

ENTEROPTOSIS (*Glenard's Disease*).—*This syndrome represents a ptosis involving the stomach, intestines, kidneys, liver and spleen and is, for the greater part, only a major portion of the general visceroptosis of "chronic congenital asthenia" (Stiller) elsewhere described.* (See "*Congenital asthenia.*").

The name "Glenard's disease" might well be applied only to cases of the type associated with *extreme* relaxation of the abdominal wall, either from repeated pregnancies or recurring ascites, which often involves separation of the recti (*diastasis*).

Two types.

APPENDICITIS

The remarkable prevalence of acute appendicitis as a disease is due merely to its separation under correct pathology from the blanket term "peritonitis" which formerly covered appendicitis together with its secondary and most fatal sequence.

Nowadays, aside from traumatism and perforating ulcer of the stomach and duodenum, acute general peritonitis is rare in the male save as a result of appendicitis, while, in the female, appendiceal peritonitis is overwhelmingly predominant, though the pelvic structures share the dubious preference.

Mortality.—Appendicitis causes upward of 2 per cent. of all deaths in the United States, a higher mortality than is shown by the statistics of foreign countries.

Age.

Etiology.—The disease is more prevalent in the young, being most frequent under the age of thirty and comparatively rare as a primary lesion in persons of middle age. It is *slightly* more frequent in the male than in the female and

Sex.

in the latter is frequently combined with inflammation of the pelvic viscera.*

Occupation.

Occupation may be a factor, especially that involving hard muscular work or severe strains, such as lifting.

One cannot but believe that most of the factors usually detailed under etiology are inoperative in the primary attack, though possibly more or less potent in recurrences. As a matter of fact one is dealing with vestigial tissue of low vitality and one peculiarly susceptible to the attacks of micro-organisms.

Its peculiar form and its calibration, choked at its beginning at the cecum, is well adapted to make it an incubator for bacterial flora.

The frequency with which foreign bodies derived from the food are found in its cavity emphasizes its unfortunate construction as a blind pouch with a constricted opening. A large number of microörganisms are competent to produce appendicitis of varying severity and course and, of late, much emphasis has been laid upon the possible importance of the bacterial flora of the mouth, tonsils and nasal-accessory sinuses.

Certainly one properly may assume that any hidden foci of infection in the form of diseased tonsils, sinuses, teeth, gall bladder, and the like are matters of decided or even cardinal importance in etiology.

SYMPTOMS.—Acute appendicitis yields symptoms which vary with the nature and extent of the inflammatory process, but show usually certain well-defined characteristics.

Obscure cases.

The subacute and acute catarrhal (endo-appendiceal) form in which the local changes are slight may yield neither definite symptoms nor signs, or disproportionately and misleadingly severe ones.

Frank cases.

The acute diffuse form is associated with an active inflammation and infiltration with retention of exudate and often with erosions of the mucosa, and produces usually but not always marked symptoms. Purulent and gangrenous forms of appendicitis are advanced forms of the same primary lesions.

Essential symptoms.

Severe abdominal pain, nausea and vomiting, fever, rigidity and localized tenderness, are the essential features of all acute forms. Pain of sudden onset and a tendency to localize itself within a few hours, at a point representing the intersection of the outer edge of the right rectus with a line drawn from the anterior superior iliac spine to the umbilicus (McBurney's point), the associated *localized rigidity, tenderness and fever* really make the diagnosis.

McBurney's point.

Fever.—Without fever a positive diagnosis of acute appendicitis is often difficult. Cases rarely occur in which local and even general peritonitis are present without fever and remembrance of the fact may prevent error.

Afebrile cases.

* Dr. A. MacLaren and other surgeons of wide experience believe this male predominance a myth.

Vomiting, usually delayed for a few hours, and *constipation*, may be marked features, and *children may show diarrhea.*

PHYSICAL SIGNS.—Early in the disease there may be none of importance save the evident pain and distress and fever, although rigidity is seldom long deferred. Lacking early.

Palpation may show a distinct defensive rigidity (*muscular rigidity*) with or without tenderness, localized or maximal, in the region of McBurney's point, *but in some cases this symptom is delayed for twenty-four hours or more.* Doubtful *early* cases justify McMonagle's maneuver, *i.e.*, fixation of the knee in extension and asking the patient to attempt flexion of the thigh on the abdomen against resistance, while the unoccupied hand palpates the abdomen. Cutaneous hyperesthesia of the eleventh dorsal nerve may be present. The relaxation of muscular rigidity under anesthesia in perforative cases is said to occur last over the exact site of the perforation. Defensive rigidity. McMonagle's maneuver.

Rectal or vaginal examination may yield important information in obscure cases and should never be omitted.

Percussion is of no special value and *auscultation* is negative. As the disease advances exquisite local tenderness develops, there is a tendency for the patient to draw up the leg of the affected side, and in certain cases palpation may reveal an actual induration, sometimes in the cecal region, more often just above Poupart's ligament. Tenderness.

Bladder irritability is common and, in the female, involvement of the appendages by inflammatory adhesions is frequent.

It is said that in certain instances the genito-crural nerve may be involved and pain radiating to the testis and associated retraction of that gland has been noted. The author has had no personal experience with such cases.* Misleading signs.

The sudden primary pain is usually merely that of a severe colic though it may be extreme and agonizing and is often epigastric or so widely diffused as to be misleading. Sudden violent pain.

In such extreme forms it often indicates actual perforation in a fulminant case.

Early tenderness is also misleadingly diffuse in many instances.

It should be remembered that defensive rigidity may antedate the pathognomonic localization of both pain and tenderness.

Abnormal Localization of Tenderness.—The appendix varies widely in position and for that reason one may find the tender point some distance removed from the typical circumscribed location.

Leucocytosis is present in most cases and high figures are occasionally noted but little dependence can be placed upon it as a guide to treatment or an aid in prognosis.

On the other hand it is a most valuable and important corroborative sign.

Perforation and General Peritonitis.—In perforation of the stomach, duodenum or appendix the typical, and usual, evidences of rupture of the viscus are often almost identical save for the *location of maximal defensive rigidity or tenderness.* Important points.

*The author has seen a number of cases of renal colic in which a primary diagnosis of appendicitis was made.

Pain usually is atrocious and abrupt, "cutting," "stabbing" or "bursting" with wide areas of lancinating radiation, and "shock" is evident.

Tenderness is usually acute, easily recognized, and associated with a marked and progressively intensifying defensive rigidity tending to extend the area of primary localization and often attaining a board-like hardness.

Atypical cases.

Unfortunately typical cases are not constantly encountered and both extreme pain and shock may be absent.

Rigidity never has been wholly lacking in the author's cases.

Shock and collapse.

In such atypical cases fever may be low or absent, the pulse slow, any antecedent pain may suddenly have disappeared and the patient believe himself better, but soon the facies assumes the Hippocratic cast, the abdomen is distended, motionless and strikingly resistant. Both knees are drawn up, the pulse becomes rapid, wiry, and small, the tongue dry.

It should never be forgotten that in the earlier stages of appendicitis the pain and tenderness may be general, and that in perforation and general peritonitis the patient's sensations and the body temperature may be most misleading.

Suggestive conjunction.

Differential Diagnosis of Acute Appendicitis.—Any sudden attack of acute abdominal pain attended by fever should at once *suggest* appendicitis as the cause, the suddenness of the onset followed by or concurrent with fever being an important though no absolute distinction as regards cases of typhoid, too often operated under a mistaken diagnosis of appendicitis.

Renal colic may prove deceptive in two forms: *first*, the so-called Dietl's crises which usually lack the ultimate localization of appendicitis and are exceedingly rare. *Secondly*, the passage of calculi through the ureter and especially such as are temporarily lodged in its lowest and narrowest portion. The recognition of renal colic should offer no difficulty by reason of the localized original pain over the kidney and its distribution. Furthermore, even the ureteral cases are usually made clear if the urine be examined *as it always should be before any operation is undertaken.* Several times in the author's experience such patients have narrowly escaped the knife.

Examine urine.

Gall-stone colic is usually definitely localized both as regards points of tenderness and referred pain. *Pelvic peritonitis* presents bilateral rigidity, hypogastric pain, and localizing symptoms *per vaginam*, but nevertheless, *acute tubal or ovarian inflammation* or a pelvic situation of an inflamed appendix *may complicate the diagnosis*, and a local examination is always necessary in women.

Pelvic disease.

A source of error.

Pneumonia and pleurisy at the base not infrequently cause temporary confusion if, as occasionally happens, the pain is referred to the abdomen, although the relation of the pain to respiratory movement is usually in itself distinctive.

Strangulation and intussusception differ from appendicitis in the fecal vomiting of the former and bloody stools of the latter.

Avoidable error.

The number of conditions which may confuse diagnosis are many and various, but those which may rightly do so are few in number, if the case is not wholly atypical, if the cardinal symptoms and the mode of onset are remembered, the urine examined and the lungs carefully interrogated.

Of the thirty odd conditions which may be associated with individual symp-

toms of acute appendicitis, not one lesion combines in itself the complete picture represented by the cardinal factors in the diagnosis.

Site often obscure.

Perforated gastric or duodenal ulcer, though usually showing early localized defensive rigidity and decided and easily detected localizing tenderness may in some instances be indistinguishable from appendiceal perforation.

In cases seen only after general peritonitis has developed, the differentiation between primary appendicitis with rupture and that due to perforating gastric or duodenal ulcer is as unimportant as it may be difficult.

Important precaution.

The commonest error associated with the diagnosis of appendicitis occurs in the early, not the later stages, when the "colicky" pain is regarded as a simple colic and castor oil or some similar cathartic is administered.

In all abrupt, severe febrile colics, however simple in appearance, and even if associated with a history of dietetic indiscretion, the question of appendicitis should arise in the physician's mind and be given expression in orders that stop all food and limit purgation for a few hours.

The temperature should always be taken and relief of a simple colic may be secured by repeated hot enemata and the hot water bag pending further investigation.

Wide variations.

Prognosis.—Mild cases may discharge into the cecum and recover without operation after a few days' illness, those going on to suppuration may result in a general peritonitis, gangrene, or perforation attended with extensive purulent inflammation in remote regions, one of which is the peri-renal tissue. Certain of the acute suppurative cases rupture into the bowel, thus draining the abscess cavity, and may show a remarkable freedom from recurrence.

Bowel drainage.

The question of surgical interference in appendicitis is a difficult and important one. An immediate surgical consultation should be the invariable rule; early positive diagnosis usually justifies immediate operation, and surgical judgment should ordinarily be accepted.

*No matter how mild the case, a second attack usually calls for operative interference and in those who live in, or whose duties take them to remote regions, a primary or interval operation should be the invariable rule.**

The frequency of relapse is so great and interval operations so safe under conditions which include expert service, that in general such operations should be advised.

CHRONIC APPENDICITIS.—*As physical signs may be wholly lacking and the symptomatology suggestive of disease of other organs, the diagnosis usually depends upon recurrent localized tenderness or a definite history of antecedent acute, subacute or recurrent appendicitis, which may masquerade under the title of "typhoid fever," "inflammation of the bowels," "constipation," "gall-stones," "renal colic," "gastralgia," "hyperacidity"* or *"ovarian trouble."*

Often misnamed.

One definite syndrome.

Occasionally there is an enlarged and thickened appendix or old inflammatory masses that may be palpated. Quite frequently there are mild but

* In children primary operation is usually indicated if the diagnosis can be made within the first twenty-four hours.

definite localizing symptoms and slight fever (99.5°F) at the time of examination.

Symptoms suggestive.

Suggestive symptoms are: pain over the stomach, gallbladder and umbilicus, increased by appendiceal pressure rather than by pressure at the site of pain; hyperesthesia over the right edge of the rectus, dull or colicky pain on exercise and pain associated with visible peristalsis in the ileocecal region (obstructive adhesions).

Right-sided *dysmenorrheal pain* following an acute illness suggesting appendicitis; *diminished power of endurance* and *persistent malnutrition*, are frequently present and a *dull aching* or even *colicky appendiceal pain* may come on *at the height of digestion.*

Misleading associations.

It should be borne in mind that chronic appendicitis is frequently associated with gallbladder disease, chronic indigestion, obstinate constipation, alternating diarrhea and constipation and mucous or membranous colitis. Further, that symptoms of obstruction may be caused by an old appendicitis with dense adhesions.

The relation of pain to meals may be constant or very variable.

Dyspeptic symptoms which are intensified by physical exertion suggest appendicitis or relaxation of the hernial openings, but may also occur in a wide range of conditions.

The location of the pain is usually epigastric but may radiate downward or lie at a lower level and once started may persist for twenty-four or even seventy-two hours even though the patient is fasting.*

Hunger pain and pyloric spasm may occur, as in prepyloric and postpyloric ulcer, but neither are peculiar to either condition.

Rutkevich's sign.

Several special maneuvers are useful as affording confirmatory evidence of the existence of an inflamed and more or less hypersensitive appendix two of which are worthy of special notice: (1) Severe *localized* pain *or tenderness* induced by pressing the fingers deep down along the inside of the iliac crest outside the cecum and making pressure backward and toward the median line. (2) Sharp *localized* pain attending the full inflation of the colon (Bastedo's sign).†

The very existence of a true "chronic appendicitis" is doubted by many surgeons and pathologists at the present time, and a large proportion of appendices removed fail to show pathologic changes of clinical importance.

Differential Diagnosis of Chronic Appendicitis.—This must depend upon the factors already given. In most instances the past history and the acute or subacute attacks are the determining factors.

Roentgenologic Aids to Diagnosis.—With relation to chronic appendicitis the work of J. T. Case and of George and Leonard is of great interest. The latter have stated recently their belief that using their technic, one may demonstrate the patent appendix in every instance. They believe that the

* Undoubtedly, many of the gastric symptoms attributed to the appendix represent unrecognized acute prepyloric and postpyloric gastric ulcers.

† This is closely related to Rovsing's palpatory method of distending the ascending colon.

use of their contrast meal is indispensable to accuracy and report but slight success with bismuth enemata.

This meal consists of 90 grams of bismuth or barium in a pint of buttermilk. They also lay great stress upon the necessity of careful plate work, stating that in many instances the detection of the appendix involves a study of fine detail. Case's adoption of the horizontal position has been followed, plates being taken both from the front and back and when necessary in the erect posture or by the lateral oblique method in retrocecal cases.

Manipulation may be necessitated in some instances and the use of the screen may be of value in the diagnosis of adhesions. The appendix may lie vertically behind the cecum, horizontally along the pelvic brim, high above the iliac crest or low in the pelvis. It should be freely movable under palpation, varies in length from one to eight or nine inches, and may be "straight, curved, or, obtusely angulated." Appendiceal abnormality is suggested by: (*a*) Absence of the appendix shadow, indicating obstruction of the lumen. (*b*) Concretions. (*c*) Immobility under palpation. (*d*) Dilatation (rare). (*e*) Sharp kinks. (*f*) A definite correspondence of the visualized structure to a point of localized tenderness. (*g*) Prolonged retention of the bismuth.

A retrocecal appendix need not be abnormal but is regarded as suspicious.

The translation of such findings into terms of surgical interference is at present largely a matter of individual judgment, but the coexistence of tenderness with or without a past history of acute or subacute appendicitis would give special meaning and importance to findings which considered alone might by no means justify surgical interference. In the case of the appendix as in other abdominal diseases a most gratifying tendency to surgical conservatism is becoming evident.

The Exploratory Operation.—*In no other field is the exploratory operation better justified or more abused.* When one has exhausted all other rational methods of diagnosis, and only then, is it justifiable, in the presence of suggestive troublesome or threatening symptoms and the absence of patent contraindications. In the recent past the relative ease and safety afforded by modern surgical technic has encouraged both the fit and the unfit to use the knife almost as light heartedly as one would employ percussion or auscultation and with respect to the appendix especially the mere detection of some slight departure from the absolute anatomic normal has been regarded, quite illogically, as a sufficient justification for surgical procedure. This unfortunate attitude has been a cause of infinite damage to one class of patients especially, viz., the chronic congenital asthenic (visceroptotic), and, furthermore, by the encouragement it has given the unskilled operator has resulted in a heavy mortality amongst individuals with clinically normal appendices.

ACUTE INTESTINAL OBSTRUCTION (*see "Pain—Abdominal"*).

ACUTE PERITONITIS.—Acute peritonitis is now essentially a surgical disease and needs but scant notice in this volume. It may be *local, diffuse or general* and its causes are too numerous for detailed description, but resolve the cases into *primary* and *secondary* groups.

The so-called primary or idiopathic peritonitis is a dubious clinical entity. Primary rare.
Secondary peritonitis means an extension of inflammation from any structure

to the adjacent peritoneum. Thus it may arise from traumatism including operation, from extension, in the severer inflammation of the intestinal tract, from perforation due to abscess or ulcer, and from chronic ailments, such as cancer and tuberculosis. Furthermore, the lowering of vitality in certain chronic diseases seems to render the peritoneum less resistant to the action of germs.

Etiology.

Misleading forms.

The evidences of infection of the peritoneum are extremely varied, certain cases associated with profound collapse and death subsequent to abdominal operation showing no lesion save perhaps points of congestion. In some there is little fluid or fibrinous exudate, in others a large amount, the fluid being serofibrinous, purulent, putrid, or in wound infection, carcinoma and some tuberculous cases, hemorrhagic.

The two most common organisms are the colon bacillus and the pyogenic streptococcus, but any one or more of a larger number of bacteria may be active. The infection is usually mixed, but with one organism dominant. In general, the staphyloccocus cases produce little or no pus or at best a seropurulent exudate. The streptococcic form is distinctly dry, but the virulent strains of the colon bacillus produce thick, creamy foul-smelling pus in quantity. Adhesions are extensive and often almost universal and the exudate may be confined in pockets. *Friction should always be sought especially over the liver and spleen.* It is by no means constant but in some instances is an early and helpful sign.

Usual type.

Attitude.

Vomitus.

Wasting and tenderness.

Symptoms.—In general peritonitis of the perforative or septic type the *facies assumes the Hippocratic type*, there is usually *severe initial pain* and evidence of *marked prostration or collapse, fever may be preceded by chills* and rise rapidly to a high point, *or chill, fever and pain be absent. Continuous high fever is seldom seen.* Painful vomiting and frequent micturition are common. The *urine is scant, indicanuria is marked*, the *pulse is small and tense*, and the *position is rigidly dorsal*, with the head high, the knees drawn up and the *abdomen tense, usually distended*, but occasionally flat, the *vomitus is yellow and bile-stained*, or, later pure green or even brownish black and of *fecal odor.* Wasting is rapid, distention tends to increase, *rigid tympany* being marked, and there is *generalized tenderness* of an extreme degree.

A common misconception.

Fluid.

It should be emphatically stated that obliteration of splenic and hepatic dulness is not confined to *perforative* peritonitis but may occur in any extreme case of tympanites. Dulness in the flanks is usual, indicating fluid, and the difficulties of diagnosis in these cases are chiefly related to the identification of the primary lesion.

Various confusing conditions.

"Hysterical peritonitis" may, however, produce a typical clinical picture on inspection, but fever, wiry pulse and indican increase are usually absent. *Obstruction of the bowel* offers great difficulty at times and demands a careful consideration of the factors stated under that head. Ruptured tubal pregnancy, acute hemorrhagic pancreatitis, ruptured appendiceal abscess, perforating gastric or duodenal ulcer and various other lesions may demand the surgeon's knife for specific etiologic differentiation.

LOCALIZED PERITONITIS.—In the male the most frequent cause of localized peritonitis is appendicitis; in the female, tubal or ovarian inflammation. A great number of localized inflammatory conditions of the viscera give rise to limited areas of peritonitis, the degree of inflammatory change and its nature depending upon the primary cause. An extended discussion of these forms belongs to surgical text-books.

Appendiceal or pelvic.

Other causes.

The lesser peritoneum, however, must receive special attention because of its frequent involvement, its importance and the obscurity of its symptoms. The lesser cavity is bounded in front by the gastro-hepatic omentum, the anterior layer of the great omentum and the stomach; below, by the upper layer of the transverse mesocolon, and it extends from the splenic to the hepatic flexure of the colon and from the hilus of the spleen to the foramen of Winslow. Above lies the transverse hepatic fissure and that portion of the diaphragm covered by the lower layer of the right hepatic lateral ligament.

Boundaries of lesser cavity.

In the presence of septic inflammation its communicating opening leading to the greater cavity (foramen of Winslow) may be shut off so that inflammatory exudate may cause a tumor in the upper abdominal quadrant. This is markedly affected by inflation of the stomach which tends to obscure or obliterate the dull note or even the palpation outline of the tumor, and an inflated colon does not cross it, but lies below it.

Stomach inflation.

This constitutes one form of the purulent circumscribed peritonitis known, because of its peculiar anatomic position, as **"subphrenic abscess."**

SUBPHRENIC ABSCESS.—Its most frequent cause is perforating gastric or duodenal ulcer and carcinoma of the stomach. Appendiceal abscess accounts for 50 per cent. of all of the remainder.

Differential Diagnosis.—When obtainable, a history of a preëxisting adequate case, such as gastric or duodenal ulcer, appendicitis or gastric carcinoma is of great assistance. A clear history of shock may also aid diagnosis.

The symptoms are usually abrupt and associated with pain, vomiting, dyspnea and signs of sepsis. A tumor may be palpable in some portion of the upper quadrants, *cyst-like in feel, markedly obscured by stomach inflation, and not crossed by an inflated colon,* and when the accumulation is immediately beneath the diaphragm and intraperitoneal the *symptoms strikingly resemble empyema (or pyopneumothorax if gas is present) and the abscess may rupture through the lung, the pus appearing in quantity in the sputum (see also "Hepatic Abscess").*

Offers great difficulties.

The author believes that such cases may in some instances be differentiated from empyema by the absence of respiratory lateral displacement of the heart. *If gas be present (about one-fourth of cases) pneumothorax is simulated, but usually is easily distinguished upon X-ray examination by the high position of the diaphragm and the characteristic upper luminosity and lower liquid shadow of the underlying areas as well as by the atypical lung compression picture.* The fact that the abscess may appear on either side and point either anteriorly or posteriorly makes this sign extremely variable.

In some instances downward displacement of the liver is very clearly

definable. So also, the lower chest may bulge and widening of the epigastric angle may occur. The mortality in this condition is enormous (80 per cent. plus).

Adhesions.

CHRONIC PERITONITIS.—The plastic lymph associated with all peritoneal inflammations, acute or chronic, tends to form adhesion bands of greater or less extent, density and permanence, and movable viscera often drag agglutinated surfaces apart to form false bands.

Valuable point.

*It should be remembered that in any condition simulating local peritonitis the abdominal wall lacks the persistent involuntary general or localized rigidity characteristic of the true ailment.**

Spreading inflammation.

In both acute and chronic forms, moreover, the ease with which the inflammation may spread from peritoneum to pleura and *vice versa* must be taken into account. As Edmund Owen says, "*the peritoneum acts in inflammation much like an inflamed joint,*" *i.e., it seeks functional rest and is exquisitely tender, whence arises the characteristic rigidity, constipation and localized tenderness and the fact that pressure which relieves ordinary colic is intolerable in peritonitis.*

An apt comparison.

Friction.

Further, as in pleurisy, auscultation may reveal areas of friction particularly in the region of the subdiaphragmatic organs which move in inspiration (chiefly the liver and spleen).

A cause of error.

Chronic adhesions in the form of false bands may cause intestinal obstruction, but in the ordinary form they may be symptomless or give rise to misleading and troublesome symptoms, as in two cases recently observed by the author in which a chronic appendicitis with acute exacerbations was exactly simulated by an extensive adhesion in its neighborhood, the appendix being normal.

PROLIFERATIVE PERITONITIS.—This is characterized by marked thickening without extensive adhesions and *is usually associated with hepatic cirrhosis, more rarely with chronic passive congestion, tumors and similar conditions.* The effusion is usually small, the viscera may be included in the thickened layers and the peritoneum may be divided into several chambers. Shrinkage of organs may be produced and the rolled omentum usually forms a transverse *tumor at the lower border of the stomach. Nodular growths simulating tubercles or malignant nodules may be present.*

An obscure condition.

TUBERCULOUS PERITONITIS.—*This condition varies exactly as do pulmonary tuberculous lesions, there being an acute miliary, chronic ulcerative* and *chronic fibroid form.*

Seek a primary focus.

It may be primary but is usually secondary being frequently associated with intestinal tuberculosis or disease of the Fallopian tubes and almost invariably with antecedent pulmonary lesions.

The prostate or seminal vesicles may be primarily involved; and invariably these structures together with the spermatic cord should be examined.

The incidence as regards *sex* is about equal until the age of puberty, after which female predominance is decided. As regards age it is most

* In chronic tuberculous peritonitis it is a peculiarly "boggy" indeterminate rigidity in most instances.

frequent in children and young adults, diminishing progressively in incidence during the third, fourth and fifth decades; as regards *race*, more common in the colored than in the white. Traumatism, cirrhosis of the liver and hernia are associated conditions in many cases.

Symptoms.—*As with tuberculosis elsewhere, the disease may be symptomless and is usually less pronounced than other forms of peritonitis; more rarely the onset and course are extraordinarily severe. In general, there is tenderness, localized or diffuse, and a temperature sometimes continuous, more often hectic in type, usually of slight range, and, a moderate, often hemorrhagic ascites.* Tenderness.

The "boggy" indeterminate rigidity so generally encountered has been mentioned elsewhere. "Bogginess."

Deep pigmentation of the skin or jaundice is sometimes observed and, especially in the upper abdominal quadrants, *tumors are encountered due to inclusion and contraction of intestinal coils, encysted exudate and adherent omentum, or, in children, to enlarged mesenteric glands.* Peritoneal friction may be audible or palpable in respiration and the tumors may simulate enlargement of the spleen, stomach, or other abdominal viscera to which they may be adherent or in deceptively close relation. Pigmentation. Tumor and exudate. Friction.

Differential Diagnosis.—Cases with acute onset and violent symptoms may be unrecognizable as tuberculous; those with continued fever are differentiated from typhoid by the absence of the Widal reaction and the continued development and predominance of peritoneal symptoms. Usually, however, the disease is strikingly chronic. Obscure cases.

Omental tumors are usually transverse and somewhat characteristic in their shape and knobby "feel." The presence of an exudate greatly assists diagnosis. Transverse tumor.

In most instances a suggestive family and personal history or the existence of tuberculous pulmonary, intestinal or tubal symptoms assist differentiation.

Furthermore, the coincidence of a doughy abdomen, a chronic continuous, intermittent or remittent fever with obstinate constipation or diarrhea, digestive disturbance, ascites (in the exudative form) irregularly localized or general tenderness and progressive emaciation, with or without tumors, is strikingly suggestive.

Pain is usually slight or absent, but may be marked and paroxysmal.

A succinct and definite portrayal of this condition is impossible, because of its variable pathologic manifestations.

CANCER OF THE PERITONEUM.—Secondary cases need no special description, they take the form of growths, generally distributed and varying from the miliary nodule to large umbilicated growths. The omentum is frequently involved and forms a peculiar tumor as in tuberculosis. Primary disease is extremely rare and most often is endothelioma. Varied types.

Diagnosis.—A positive diagnosis is easy in secondary cases if the primary source be manifest, but is always difficult in the rarer primary forms; indeed a positive differentiation is hardly possible without exploration, the symptoms being almost identical with those of tuberculosis or echinococcus infection in most instances. Often impossible.

DISEASES OF THE PANCREAS

Primary necrosis

ACUTE HEMORRHAGIC PANCREATITIS.—Etiology.—Infection through the gallbladder and ducts is the chief source of this disease, though bile itself, if forced back into the pancreas by obstruction to its normal outflow, may produce the *necroses* and hemorrhage by activation of its own ferments. It may also be involved in septicemia, by traumatism, or, secondarily, in the extension of inflammatory processes from neighboring organs.*

Necrosis and hemorrhage.

Symptoms.—*These closely simulate perforation, particularly that of duodenal or gastric ulcer, the onset being sudden with violent colicky pain in the epigastrium, associated* with *vomiting* and *collapse*, with *subsequent epigastric swelling.* It is evident that a positive diagnosis is often impossible. The maximum of tenderness *may* clearly follow the outline of the pancreas and there may be a suggestive history of antecedent disease of the gallbladder.

Gangrene follows.

Diagnosis.—The diagnosis must be based upon the *sudden onset* with pain and vomiting, symptoms of *collapse* and, several days later, the appearance of a fever amd *fixed tumor* corresponding to the position of the affected organ. The diagnosis is usually though not always made by exploratory section. The urine may show leucin and tyrosin after treatment with basic lead acetate, filtration, treatment of the filtrate with hydrogen sulphide and evaporation.

Prognosis.—Recovery is the exception.

ACUTE SUPPURATIVE PANCREATITIS (*Pancreatic Abscess*).—Suppurative pancreatitis is associated with preliminary digestive disturbance, fever, possibly of a septic type and what is more important, by a fixed epigastric tumor.

GANGRENOUS PANCREATITIS may result from hemorrhage or rarely suppurative inflammation. It has no distinguishing symptom aside from those of the primary hemorrhagic or septic pancreatitis, followed by collapse and death in a few weeks or days. The distinction is made *post-mortem.*

CHRONIC PANCREATITIS closely resembles *histologically* in its two forms, atrophic and hypertrophic cirrhosis of the liver, the former condition is frequently associated with diabetes. It is seldom recognizable.

PANCREATIC CYSTS.—The chief characteristics are recurrent colic associated with nausea, vomiting and progressive abdominal distention, glycosuria, emaciation and a fixed median tumor, cystic in feel, and *lying above the inflated colon and below the stomach.* They may attain great size.

CARCINOMA OF THE PANCREAS.—Primary carcinoma involves usually the head of the gland. Metastasis or direct invasion of other organs is frequent; the condition is rare.

Symptoms.—These are chiefly *progressive emaciation, anemia, intractable dyspepsia,* and *dull or severe epigastric pain,* the *stools are often light and*

* The actual causes are not yet well understood.

pasty, muscle fibers of ingested meat may result from faulty pancreatic secretion, and jaundice and ascites are not infrequent. The *fixed* tumor is median unless the tail of the pancreas be the part involved and glycosuria, jaundice and various pressure symptoms may be present.*

Jaundice is present from obstruction and the gallbladder is enlarged in about 75 per cent. of the cases, the latter being usually of delayed and gradual development.

PANCREATIC CALCULI.—The rough round or spiculate stones are, in rare instances, associated with violent epigastric colic and may be found in the stools. Ordinarily they produce an interstitial pancreatitis or lead to abscess.

Such stones are composed chiefly of carbonate and phosphate of calcium and magnesium and sodium phosphate.

The Cammidge Test and Loewi's Test.—The many reports at home and abroad indicate that these two tests are no longer regarded as of diagnostic importance, and with the simpler and more direct methods now available through the use of the duodenal tube any such usefulness as the former may have had has passed. Suggestive merely.

The diseases of the pancreas are essentially surgical and best dealt with in surgical treatises.

DISEASES OF THE LIVER AND THE BILIARY PASSAGES

CONGENITAL ANOMALIES.—An extraordinary variety may be found, including abnormalities in the size, number and position of the lobes, the presence of accessory masses of tissue, complete absence of the organ, abnormal mobility, and even its transfer to the left side, as is seen in complete *situs inversus*. It has been found within the thorax in cases of diaphragmatic hernia, or following injury, and may be markedly displaced downward, or grooved, swollen, and thickened by tight lacing. Multiple depressions of a similar sort may appear upon its superior surface, and are not satisfactorily explained. The gallbladder may be entirely lacking and curious variations occur in the size, number and distribution of its ducts. Corset liver.

INFLAMMATION OF THE LIVER (*Acute Hepatitis*).—**Etiology.**—Inflammation may result directly from injury or follow an infection, diseases of the stomach and intestines or portal veins, and may be either simple or suppurative in character.

It is rare in temperate climates and relatively common in the tropics. It constitutes one form of the "tropical liver" and its chief symptoms are *fever, pain, tenderness, and enlargement, with or without decided jaundice.* Tropical liver.

In hot humid climates the transition from functional hyperactivity and its secondary phase, stasis, tends to invite actual acute hepatitis, and comparatively slight blows, chilling of the body surface and like trivial causes may excite an acute attack.

* See also "The Examination of the Duodenal Contents."

Usually secondary.

ABSCESS OF THE LIVER.—Pyemic abscess, whether single or, as is usual, multiple, may result from infection conveyed, directly or indirectly through arterial, venous, or lymphatic channels. Any form of pyemia or general septic absorption may result in infective embolism of the hepatic artery, many of the suppurative and ulcerative lesions of the abdominal tract result in portal embolism and almost any severe acute infectious disease may produce hepatic abscess. Among the vast number of associated conditions one may mention *appendicitis*, ulcer and cancer of the stomach and duodenum, spinal caries, renal or perirenal abscess, the echinococcus, ascarides and liver flukes, Balantidium coli, inflammation and malignant growths involving the pancreas, gallbladder or its ducts, and any ulcerative and suppurative process in and about the intestines, such as typhoid fever, dysentery, appendicitis, cystitis, prostatitis, and tuberculosis. The so-called *tropical abscess* is almost invariably solitary and associated with amebic dysentery.

Associated conditions.

Tropical abscess.

Usually pronounced.

Symptoms.—These, usually pronounced and localizing, more rarely latent and obscure, are chiefly: *enlargement of the hepatic area or localized swelling, leucocytosis* (save in amebic cases), *pain, and sepsis.*

Pain variable.

The pain may be entirely lacking for long periods, is not severe until the abscess reaches the surface, and is not distinctly localized, but felt both over the liver and in the region of the right shoulder-blade and, in some instances, chiefly at a point to the left of the median line, below the costal margin.

Enlargement of the liver is an almost invariable symptom and when present is usually marked; hence both the upper and lower hepatic borders should be examined and the condition of the lower interspaces investigated. The right lobe is oftenest affected. *A localized swelling* corresponds to the "pointing" of the abscess and, when present, is invariably accompanied by edema of the superficial tissues, and in some instances may show fluctuation.

Localizing signs.

Edema.

Auscultation may reveal *peritoneal friction*, due to capsular involvement, and *adhesions* may limit movement.

Adhesions.

Loss of strength and weight and appetite is marked, *morning vomiting* and *pronounced dyspeptic phenomena* are usually present.

Fever is either definitely intermittent or decidedly irregular and is usually associated with chills, and severe nocturnal sweats and *jaundice* may or may not be present. The decubitus of such a patient is not characteristic, as the right knee may be drawn up and the right shoulder lowered in any disease affecting this region and, moreover, this posture is far from constant.

Sepsis.

A variable sign

Persistent sweats or sweating constitute a symptom of decided importance because of its relative constancy as a symptom.

Roentgenology.—Roentgenography may be of the greatest service in many instances. The tube should be at the back at the level of the sixth and seventh dorsal vertebræ, and so placed as to direct the rays slightly downward.

Complications.—*Perforations.—An hepatic abscess may burrow in many directions and discharge its contents with disastrous results.*

Sequelæ of perforation.

Perforation may occur outward through the skin, after the formation of adhesions, either directly or, more frequently, through a sinuous burrow.

Perforation into the peritoneal cavity is very rare, as is that involving the portal vein, the inferior vena cava and the right kidney.

Pulmonary form.

The discharge of pus through a bronchus is not uncommon, for it may reach the pleura and lung and cause empyema or abscess even on the left side, or perforate the pericardium. It is usually preceded by severe cough and the expectoration of bright-red sputum.* Sometimes, though the physical findings are negative, the patient, during a violent fit of coughing, will raise a considerable quantity of pus intermixed with blood, often brown or bile-stained and not infrequently containing shreds of lung tissue. Perforation upward occurs in more than 50 per cent. of all cases. These cases may go on for long periods with a daily expulsion of quantities of pus, and spontaneously subside, leaving only a certain amount of lower chest deformity and a crippled lung, but the aid of modern surgery is, however, always needed.

Error frequently possible.

Differential Diagnosis.—Too often this surgical condition is misconstrued or entirely overlooked and great stress should be laid upon the *existence of possible causative factors*. Furthermore, *enlargement of the hepatic area is almost invariable*. *Jaundice* when present (20 per cent.) is extremely suggestive; sallowness and decided cachexia are seldom absent in the established case. *Localized swelling*, whether over the free surface of the liver or the lower right interspace, is most important when present, but, unfortunately, is often absent. *A distinctly septic trend of the symptoms, especially sweating, at once suggests abscess. The spleen is enlarged* and there is marked *leucocytosis* which may exceed 50,000. As between hepatic and subdiaphragmatic abscesses, a surgical exploration is often required for positive differentiation.

Blood.

Exploration.

Resort to the roentgenogram or, better, both that and roentgenoscopy, as stated, may be productive of results. Exploratory incision is often required and friction over the hepatic area should always be sought.

Exploratory puncture is still employed, though less frequently than formerly, and is seldom attended by untoward results if the rules of asepsis are observed.

Prognosis.—The prognosis depends upon three factors, *i.e.*, the number of abscesses and the promptness of diagnosis and surgical relief. Cases of multiple abscess are practically hopeless.

HEPATIC HYPEREMIA.—Transient active hyperemias are unimportant save in the tropics where they often constitute the usual first stage of an acute hepatitis.

Secondary.

Chronic hyperemia is invariably due to obstructed flow of blood from liver to vena cava. Among the causes are: chronic heart disease, especially such as results in dilatation of the right heart, asthma, emphysema, bronchiectasis, pulmonary fibrosis and portal thrombosis. Such a congestion results in the production of central lobular congestion, peripheral anemia and fatty degeneration. The liver is large, though in the presence of long-continued con-

* In amebic abscesses the pus is of a peculiar "anchovy sauce" character.

gestion it may undergo a certain amount of contraction attended by the formation of a finely nodular surface, the section showing dark red markings on a light background, *i.e.*, "nutmeg" liver.

'Nutmeg liver.'

Symptoms.—The essential symptom is *increase in size*, as recognized by palpation or percussion, with especial reference to the lower border, or, lacking this, increased density associated with a certain amount of pressure tenderness particularly over the left lobe, the secondary symptoms (such as chronic gastric catarrh and intestinal disturbance) and the existence of a primary cause. Ascites may or may not be present, according to the degree of change produced, and hematemesis is rare.

TUMORS OF THE LIVER.—Primary malignant disease of the liver, whether sarcomatous or carcinomatous, is excessively rare. Secondary carcinomatous growths are common, sarcoma most unusual.

Secondary carcinoma most often follows involvement of the stomach, pancreas, kidney and intestines, and usually first appears in the right lobe. As the process advances, the nodular masses tend to soften and become umbilicated by central absorption, this condition being oftentimes distinctly palpable.

Nodules.

Cystic tumors are rare and usually small. They ordinarily originate in the gall-ducts, and yield a clear or slightly yellow fluid.

PARASITIC INVOLVEMENT.—*Entameba histolytica* (*tetragena*) is the cause of practically all tropical hepatic abscesses, being found sometimes even in the absence of dysenteric symptoms. The echinococcus is the most important of the various other forms of parasites encountered.

Fig. 474.—Tenia Echinococcus. (*Braun.*)

ECHINOCOCCUS CYSTS (*Hydatid Cysts*).—These are due to the activity of the embryo of the "*echinococcus granulosus*" (*tenia echinococcus*), a tape worm, which is rarely encountered save in Ireland and Australia, though found occasionally in Germany, and now and then met with among the Icelanders who have settled in British Columbia. The worm is not more than ¼ inch in length, and belongs properly to the dog. It is composed of three or four segments and the mature terminal portion is but 2 mm. long and 0.6 mm. wide, though it may contain several thousand eggs. The head shows a rostellum, four suckers and two rows of hooklets; the embryo, provided with six hooks and arranged in pairs, makes its way through the intestinal wall and may enter the portal vessel and be carried from the liver and the systemic vessels by which it reaches the brain or other distant organs, or it may remain encysted in the peritoneal cavity or the surrounding muscles.

The parasite.

Habitat.

Points of entrance.

Conveyance.

It is thought that this embryo may find entrance through infected dust as well as through drinking water, and in Australia and Iceland, where the disease is most prevalent, the dogs are very generally infected and the relation between them and their masters is very close.

General Symptoms of Hydatids.—Diagnosis primarily depends upon the presence of a cystic tumor and upon the recovery of hooklets from fluids discharged or obtained by puncture. The presence of hydatid disease in other portions of the body is of course suggestive. In the liver the symptoms are more distinct, owing to the fact that these cysts when superficial yield to the pleximeter finger upon percussion a peculiar echoing vibration known as the "hydatid" thrill. They may be large, and, in this location, produce various pressure symptoms, or pyemic symptoms if they suppurate. Such cysts, like hepatic abscesses, may rupture in almost any direction.

Hooklets.

Tumor.

Percussion thrill.

The complement-fixation test is referred to elsewhere. It would seem probable that it is not specific for *Tenia echinococcus*, but for the group of *Tenia*.

CIRRHOSIS OF THE LIVER

Basic Pathology and Varieties.—The central factor in all forms of cirrhosis of the liver is the increase of connective tissue.

Four varieties

We have to deal clinically with four varieties:

(1) **Laennec's cirrhosis,** the common form of the disease as encountered in every clinic. (2) *Hanot's cirrhosis.* (3) *Syphilitic cirrhosis.* (4) *Chronic perihepatitis or capsular cirrhosis.* (5) *Banti's Disease* (see "Splenomegaly).*

LAENNEC'S CIRRHOSIS.—Etiology.—To an extent etiologically significant it affects the male (75 per cent.), and though encountered in rare instances in young children it is essentially a disease of the fifth and sixth decades of life. Certain of the infections, both acute and chronic, may result in cirrhotic changes. Chronic obstruction of the bile ducts, chronic passive hyperemia and anthracosis are to be numbered among the etiologic factors. *Alcohol being the most constant associated factor in Laennec's cirrhosis, information concerning the extent and duration of alcoholic indulgence is always important.* Gout, lead poisoning and the excessive consumption of highly spiced and over-rich articles of food, though contributive factors, are seldom of primary importance.

Many factors.

Alcohol the chief.

Lacking a history of prolonged use of alcohol, a positive diagnosis, though justifiable, will often prove erroneous, while a knowledge of previous alcoholic indulgence, even though moderate, may explain many obscure gastrointestinal symptoms and justify a tentative diagnosis.

A diagnostic aid.

It is possible that syphilis will be found to play a larger part than was supposed prior to the introduction of the Wassermann and luetin tests.†

Morbid Anatomy.—*True Laennec's cirrhosis*, or "*gin drinkers' liver*," of the classic type is small, hard, resistant to the knife, has about the consistence of "wet leather" and is irregularly lobulated or nodular. It is true, nevertheless, that as encountered in ward work the cirrhotic liver is a large or

"Hobnail liver."

* The cirrhosis due to chronic disease of the heart or lungs (nutmeg liver) is discussed under "Chronic Hyperemia," and a large number of minor forms of minor or trifling pathologic and etiologic interest need not be specially considered in this volume.

† Although the author's cases arousing this suspicion may have been wholly cases of accidental concurrence.

normal-sized liver in at least 50 per cent. of the cases, yet possessing otherwise all of the characteristics of Laennec's cirrhosis. The little nodules may, by the contraction of the fibrous bands, become prominent, and thus form the "hobnail liver," so called from their resemblance in size and general appearance to hobnails in a shoe. The color is yellow, and the cut surface is mottled with color running from yellow to brown.

Microscopically, the changes consist in the formation of new connective tissue around the sheath of the veins, and this, contracting, causes compression of the portal capillaries, portal obstruction, and, therefore, congestion of all the territory normally drained by the portal vein.

Source of symptoms.

It is this chronic passive congestion which produces most of the clinical symptoms of the disease, such as ascites, chronic catarrhal gastritis, enteritis, hematemesis, bleeding hemorrhoids, hemorrhoidal varices and that distention of the superficial abdominal veins known as the Caput Medusæ, this last symptom appearing, usually, if at all, after the oncoming and long persistence of ascites, which is a direct and important contributory factor.

Symptoms of Laennec's Cirrhosis.—As suggested previously, diagnosis of the ordinary form of cirrhosis of the liver depends ordinarily to an unusual degree upon the knowledge of the patient's habits and the proper interpretation of secondary symptoms.

Habits.

Given any case presenting a history of immoderate or even moderate alcoholic indulgence extending over a period of many years and showing symptoms of chronic gastric catarrh or chronic catarrhal enteritis, even though a history of hemorrhoids and hematemesis is lacking and the physical signs are indeterminate, a tentative diagnosis of an existing hepatic cirrhosis is justified.

Congestive symptoms.

The fact that, at present, the disease is seldom recognized or suspected until well advanced, is no doubt due in many instances to a failure to appreciate the significance and value of grouped etiologic and diagnostic factors.

Late recognition.

Physical Signs.—*The physical signs of the advanced disease may or may not be well defined.* By auscultatory and direct percussion and by palpation one may often make out a decided shrinking in the hepatic outline, especially that representing the left lobe, and special stress should be laid upon the fact that the early shrinkage is most marked over the left lobe. Quite as often the liver is distinctly large though hard (*fatty cirrhosis*) or, even if of normal size, may appear large because of the obligatory descent of its inferior border in the event of associated emphysema and resultant low diaphragm. Even though the right lobe appears hypertrophic one may often find relative left lobe retraction of decided degree.

The spleen is moderately enlarged, usually just palpable, or but one or two fingerbreadths below the costal margin.

Its degree of enlargement measurably corresponds to the grade of hepatic cirrhosis and both it and the liver may undergo considerable fluctuations in size under rest, cardiac stimulation, or the free use of saline cathartics.

Furthermore, the spleen may undergo decided shrinkage during the terminal stages of the disease and in some instances weighs no more than the normal spleen at autopsy.

The so-called preatrophic stage of mere *engorgement* is so seldom positively determined that it might as well be discarded as unproven. Another fallacy is that pertaining to the direct recognition by palpation of the hobnail projection on the liver surface. These can sometimes be felt very plainly through a thin wall, but no stress should be laid upon negative findings. *When the atrophic liver can be palpated, the sharp firm edge indicating induration is most suggestive and valuable.* Any co-existent ascites prevents accurate palpation of the liver or spleen until after aspiration of the ascitic fluid. The "dipping" method which consists in burying the fingers by a sharp stroke upon the abdominal wall in order to displace the fluid and meet the descending liver edge in inspiration is often successful.

Preatrophic stage.

"Dipping."

Ascites occurs only in about 60 *per cent. of these cases and is always a relatively late symptom.*

Hematemesis (from esophageal varices) is absent in about 80 *per cent. of cases.*

All definite symptoms relating specifically to the liver itself may be lacking until the later stages.

The Caput Medusæ, so called from its fanciful resemblance to the Medusa head, is formed by the enlargement of the superficial veins of the abdomen and, when present, is a symptom of much interest and importance, but is a comparative rarity, often confounded with the general distention of the superficial abdominal veins met with in various conditions producing pressure and obstruction in the abdominal circuit.

Seldom marked.

Diagnostic Summary.—*To summarize: The diagnosis of atrophic and fatty cirrhosis alike must rest upon mosaic groupings or upon all of the following data: (a) A history of high living, overfeeding, and in nearly every instance alcoholic indulgence. (b) Loss of appetite, morning nausea and vomiting, irritable stomach, excessive fermentation of the non-stenotic type, constipation or intermittent diarrhea, and various other symptoms of chronic gastric or intestinal catarrh. (c) The recognition of a shrunken, large, or normal-sized, hardened liver and a palpable spleen. (d) Loss of weight, strength, activity and color (though a ruddy color may persist until late in the disease). (e) Hemorrhoids, blind or bleeding. (f) Ascites. (g) Hematemesis. (h) Jaundice.*

This last is an unusual symptom in atrophic cirrhosis, though the author has frequently encountered it in a form so extreme and so bronze-like as to produce a color strikingly like that seen in typical Addison's disease, though diabetes was lacking. The combination of "bronzing," cirrhosis of the liver, and glycosuria forms a syndrome often described but rarely seen.

It must never be forgotten that the ascites of atrophic cirrhosis ordinarily precedes any marked general edema or anasarca and remains usually for a long period quite alone or associated with a moderate edema of the lower extremities. These cases furnish some typical examples of the "poached-egg belly."

Edema slight.

Prognosis.—The disease is slowly progressive and beyond curative therapeutics, though the cessation of the use of alcohol or of any other habits of a similar nature, attention given to the heart and stomach and a thoroughly hygienic life will do much to lengthen the patient's term and ameliorate his discomfort.

Incurable.

Unimportant.

Differential Diagnosis.—*Simple atrophy of the liver.* This condition accompanies various cachexias, characterized by profound exhaustion and marasmus, is suggested by such conditions, and is of no great clinical interest or importance.

Nutmeg Liver.—An atrophic nutmeg liver may give rise to much difficulty, but the preëxistence of disease of the lung or heart, and the fact that it does not present a primary ascites, but one secondary to or associated with a general anasarca, usually suffices for its differentiation.

Readily recognized.

Fatty Cirrhotic Liver.—A very considerable number of the cases of cirrhosis that we meet with are cirrhotic fatty livers, a form easily recognized by palpation, the organ being enlarged decidedly and of firm consistence. It presents no essential difference in symptomatology from that of the ordinary atrophic form, and has been included in the description above given.

Portal phlebitis and thrombosis. The diagnosis of this condition depends chiefly upon the existence of a potent cause, such as ulceration, gall-stone and suppurative diseases, and generally the condition is suggested by a *very rapid onset of ascites*, splenic enlargement, and the other symptoms of portal obstruction, together with the rapid recurrence of ascites after paracentesis, though this sometimes occurs in true cirrhosis.

The specific gravity of the clear ascitic fluid is nearly always below 1015 in the case of cirrhosis, whereas in chronic peritonitis it is distinctly turbid, and, in ovarian cyst, of higher specific gravity and containing a greater amount of albumin.

HYPERTROPHIC BILIARY CIRRHOSIS (*Hanot's Disease*).—Called also "hepatogenous" and "biliary," this rare disease is characterized by a general increase in size and less firm resistance to section than is present in the atrophic form. The weight may be double that of the normal liver. Both the outer and the cut surfaces are comparatively smooth, much like the surface of morocco leather and the term unilobular as opposed to multilobular is applied to this condition, because of the involvement of individual acini rather than of groups. The process is practically a pericholangitis, and there is much evidence to support the theory of direct infection as a primary cause.

Probably an infection.

Suggestive course.

Cardinal Symptoms.—In its early stages the disease presents the picture of recurrent attacks of "catarrhal jaundice."

It is extraordinarily and insidiously chronic, affecting the general health but slightly for years.

Marked icterus.

After a time the jaundice and digestive disturbances though mild become persistent. The spleen, which previously may have been palpable only during the attacks, progressively enlarges and very slowly the case reaches a stage justifying the following description.

The liver is decidedly enlarged; the spleen, always greatly increased in size, may attain a weight of even ten times the normal; icterus is intense, progressive and persistent, and the secondary symptoms of portal obstruction are entirely absent, ascites if occurring at all, coming on late as a part of the general cachexia.

The color of the stools wrongly suggests an intermittent bile secretion,* the urine is abundant as contrasted with that of atrophic cirrhosis and contains urobilin, bilirubin, and oftentimes albumin. The disease is one of long duration, lasting on the average from five to ten years. Urine.

SYPHILITIC CIRRHOSIS.—This disease, usually congenital and chiefly affecting young male adults, may assume either an atrophic or hypertrophic type and represents a genuine interstitial syphilitic hepatitis. As seen in the adult it is more commonly atrophic; in children, ordinarily hypertrophic. A positive diagnosis is never possible, but is rendered probable by the history of or by marked external evidence of syphilis and a positive Wassermann or luetin test. Two forms.

Symptoms.—The liver edge is peculiarly sharp and indented and the surface is grooved and sometimes nodular. Such livers are often fixed by adhesions to the abdominal wall or viscera in contact with them.

In many of these cases one detects *the friction of perihepatitis* if ascitic fluid is absent, as is likely to be the case until late in the course of the disease.

The spleen is enlarged, ascites is absent or is late in appearing, icterus is relatively common as compared to the ordinary cirrhosis and the urine is dark and usually albuminous with or without bile pigments.

SYPHILITIC GUMMATA OF THE LIVER.—These may occur at any age as a result either of congenital or, more frequently, acquired syphilis and vary greatly in size. The clinical picture is that of hepatic cirrhosis, but after the removal of any ascitic fluid that may be present, the nodular outline of the liver is often appreciable. Pain may be decided and troublesome as in the case of hepatic carcinoma. Fever may or may not be present. Nodules

Perihepatic friction and fixation by adhesions are commoner in lues than in cancer. The nodules, if palpable, are usually smaller and less progressive in growth than those of carcinoma, but may be of great size in some instances.

Certain cases show a peculiar latency as regards general symptoms, though representing marked and easily recognized tumors. Latent cases.

The most important clinical inference is that when one meets with a nodular liver, with or without symptoms of cirrhosis or history of syphilitic infection, he should apply the therapeutic test of mixed treatment, and indeed this should be applied in practically all cases of solid growths in the abdominal region.† Therapeutic test.

ACUTE PERIHEPATITIS.—This condition, invariably secondary to other diseases of the adjoining viscera, or to direct injury, may take on any of the forms of inflammation common to serous membranes.

Symptoms.—Pain, both in respiration and passive movement of the liver, *friction*, usually both audible and palpable, and fever, are the important symptoms.

CAPSULAR CIRRHOSIS.—This is merely a chronic capsulitis, the liver substance taking little or no part in the process. The same process in the

* A finding unjustified if interpreted as meaning that *no* bile passes from the liver to the gut. It is always present in the duodenal content in biliary cirrhosis in sufficient amount to respond to the appropriate tests.

† As should both Wassermann and luetin tests.

spleen and peritoneum is said to be its almost invariable accompaniment, as is also chronic interstitial nephritis. There are two types, one associated with the ordinary symptoms of cirrhosis, the other with adherent pericardium and chronic mediastinitis (Pick's disease).

Secondary.

AMYLOID LIVER (*Amyloid Degeneration of the Liver*).—This condition is invariably secondary to some severe derangement of metabolism, especially those associated with inveterate syphilis, chronic septic absorption, as in cases of caries of bones and pulmonary tuberculosis of the advanced type. It may also be the result of chronic and neglected syphilitic infection. The

Characteristic picture.

liver is quite firm, insensitive, painless, *extremely hard* with rounded edge, and *smooth. All symptoms of portal obstruction are absent and icterus is seldom present.* The disease almost exclusively affects males (80 to 90 per cent.), and is invariably fatal.

ACUTE YELLOW ATROPHY

Clinical curiosity.

Etiology.—This excessively rare disease chiefly affects women and falls mostly upon the third decade. It is frequently associated with pregnancy, and has followed profound emotion or shock, syphilis, alcoholic excess and various other ill-determined factors.

Morbid Anatomy.—The liver cells are necrotic, and there is a general catarrh of the finer bile ducts associated with hemorrhage between the liver cells. The organ itself is greenish yellow, greatly shrunken, flabby, with a loose capsule, and on section appears mottled with yellowish brown and red. The heart may show fatty degeneration, the spleen is usually enlarged and the kidneys may also be degenerated.

Progressive icterus.

Symptoms.—*The picture is primarily that of a catarrhal jaundice succeeded in a few days or perhaps two or three weeks by vomiting, perhaps hematemesis, subcutaneous and mucous hemorrhage, headache, delirium, muscle tremor and sometimes convulsions.*

Variable fever.

The jaundice deepens with the onset of the graver symptoms. There is usually no fever, rarely a marked pyrexia. The general condition is that of the typhoid state and ordinarily the condition terminates in coma.

Profound exhaustion.

Shrinkage of the liver may greatly reduce or entirely obliterate its normal area of dulness. The urine contains bile, frequently albumin, albumose and

Shrunken liver

casts. Leucin and tyrosin are frequently but not constantly present, the stools are clay colored, urea is diminished but ammonia-nitrogen is increased.

Differential Diagnosis.—The interesting symptom-group differentiates this disease from all others except phosphorus poisoning, in which the three leading symptoms, viz.: hemorrhage, profound jaundice and shrinkage of the liver may occur. The examination of the luminous vomitus and history of the case are the chief distinguishing points. Grave symptoms may occur in connection with other diseases associated with jaundice, but the picture of acute atrophy is seldom present.

WEIL'S DISEASE (*Acute Febrile Jaundice*).—This disease is with difficulty distinguishable from an acute catarrhal duodenitis of the severe epi-

demic type. It is supposed to be caused by a *Leptospira icterohemorrhagica* chiefly among butchers and is ordinarily a disease of warm weather, affects young and middle-aged adults, and is characterized by early jaundice, tenderness and tumefaction of the liver and spleen, severe headache, *pain in the legs, arms and lumbar region, and, oddly enough, in the masseter muscles.* The stools are often light as in actual obstructive jaundice. The disease lasts for a week or two, the temperature is septic in type, may be moderate or high and albuminuria may be present.

Differential Diagnosis.—The peculiar symptom-complex just described offers the only means of differentiation from the ordinary acute *catarrhal duodenitis* which may be but a mild variant of the severer ailment. *Malaria* in its remittent form would show the parasite in the blood, *phosphorus* poisoning by the history of its ingestion, and the odor and phosphorescent character of the vomit. The disease is rare and seldom fatal but may occur in epidemic form.

From the most recent reports of Trembur and Schallert, Huebener and Reiter and certain Japanese investigators it would appear that a specific *spirochete* (*"nodosa"*) is constantly present in this disease.

DISEASES OF THE GALLBLADDER

CATARRHAL JAUNDICE (*Cholangitis catarrhalis*).—This is essentially a part of a catarrhal gastro-duodenitis, and is clinically an incompletely obstructive jaundice; inflammatory products and swelling narrowing the caliber and almost completely blocking the opening of the duct. It is especially common in children and young adults, but may be met with at all ages in connection with acute or subacute indigestion or any of a large number of diseases associated with a tendency to catarrhal inflammation of the upper intestinal tract. It sometimes is epidemic and due to acute infection and in such cases may reach such high grades as to demand and receive special description (see "Weil's Disease").

Epidemic type.

A suppurative form also occurs in connection with the severer infections, affecting the gall-bladder and gastrointestinal tract.

Save for the evidence of jaundice (color, light pasty stools, etc.) it may be wholly symptomless or be accompanied by symptoms of indigestion with vomiting or early and more or less persistent nausea. The loss of appetite is often absolute for several days. The liver is usually slightly enlarged, but the spleen rarely tumefies. *The duration is* from two to twelve weeks.

Jaundice marked.

Chronic catarrhal jaundice is merely an expression of chronic obstruction, due to any one of many causes. It may be complete or incomplete, persistent or intermittent, and will be discussed further under "Cholecystitis" and "Cholelithiasis." (See also "Hypertrophic Biliary Cirrhosis.")

ACUTE CHOLECYSTITIS.—In from 15 to 20 per cent. of the cases a cholecystitis exists without gall-stones. The flora of the disease is extensive

and includes the pyogenic staphylococcus, streptococcus, pneumococcus, bacillus aerogenes capsulatus, colon, and *typhoid bacillus*.

Jaundice usually absent.

Symptoms in the Typical Case.—*It should be stated emphatically that jaundice is usually absent* (90 *per cent.*) *and that the pain maximum may be distinctly localized in the region of the tip of the ninth cartilage over the gallbladder itself, or may first be referred to the epigastrium or even to the region of the appendix. Pain usually radiates to the right shoulder and the back, is paroxysmal in type and may be agonizing in its intensity.*

*There are usually nausea, vomiting, rapid pulse, chill and sweats, fever, prostration, local tenderness, and a defensive rigidity, which in most cases become definitely localized over a more or less enlarged gallbladder.**

In old cases of cholelithiasis associated with colic, defensive rigidity is so inconstant as to suggest, when present, a perforation.

The urine may or may not show a toxic (febrile) albuminuria. The liver is rarely enlarged and the question of splenic enlargement depends upon the nature and virulence of the infectious agent. Recurrence is, of course, the rule in cases of cholelithiasis or those associated with chronic infections of the viscera in circuit.

Acute cholecystitis may initiate, or follow, typhoid fever but in such of these cases as have been seen by the author the pain was not excessive.

Typhoid fever.

Duodenal cultures

It would seem probable that an early diagnosis of typhoid fever in the cases of primary cholecystic localization might be made through the making of cultures from the duodenal contents.

It is obvious that a mild but important inflammation may exist without any exudate sufficient even to cause distention, especially if some drainage possibilities are maintained.

Atypical Cholecystitis.—A large proportion of the cases of acute cholecystitis fail to give the striking clinical picture above outlined.

This is particularly true of cases complicating other diseases, but applies also to the exacerbations of chronic cholecystitis, with or without actual cholelithiasis.

Pain may be slight or represented by mere distress or discomfort in the region of the gallbladder.

Tenderness may be demonstrated only by special maneuvers which combine deep thumb pressure over the gallbladder with deep respiratory movements.

Localized defensive rigidity is present at times when other signs fail.

Fever may be wholly submerged in that of any primary disease present, or, in the absence of such primary ailment, be only slight and evanescent.

Vomiting may be wholly absent and even nausea only slight.

Knowing the peculiar difference in the amount and character of the exudate associated with infection by various pathogenic microörganisms, one cannot doubt that many cases yield no recognizable signs nor produce any overdistention of the gallbladder itself.

Inasmuch as the minor cases may be of the utmost importance in diagnosis, the student should not be led to assume that the striking classic

* Such enlargement is usually associated with the presence of gall stones.

symptoms must exist. In typhoid especially the condition is very generally overlooked though the gallbladder is often distended and palpable.

Distention of the Gallbladder.—This constitutes the most constant of the many expressions of cholecystitis and may be present when the others are slight or undemonstrable, save perhaps tenderness which in some degree is its almost invariable accompaniment.

Distention and tenderness

CHOLELITHIASIS.—Gall-stones may be single or present in hundreds or thousands (usually from 5 to 10 or 12).* They may vary in size from mere grains to stones measuring 6 inches or more in length, are polygonal with smooth facets, when multiple, more rarely single and smooth, ovoid or round. They have a nucleus, usually of bile pigment, and various bacteria are actively concerned in the inflammation or congestions, resulting stasis, decomposition, and cellular disintegration, initiating, and contributing to their formation.

Number.

Size and form.

Section of these stones shows a laminated structure with radiations, and an analysis reveals biliary and fatty acids, magnesium and calcium salts and even traces of iron and copper.

Cholesterin constitutes the bulk of the common type of gall-stones, the color is brown, yellow or even white and when just removed they are somewhat soft, have a greasy feel and occasionally the surface of a solitary stone may be rough and mulberry-like. The mixed bilirubin-calcium stone (containing 25 per cent. cholestrin), the pure bilirubin-calcium stone and various other unusual forms may be found. It is probable that typhoid and colon bacilli are especially potent in relation to gall-stone formation, the streptococcus much less so. Autopsies show an astonishing large number of cases of gall-stones and prove that during life they have in many instances yielded no symptoms save, perhaps, "*flatulent indigestion.*"

Structure and constituents.

Autopsies also demonstrate the frequent concurrence of gall stones and carcinoma of the gallbladder, the latter disease being seldom found without the former. This should not be misinterpreted as indicating that patients with cholelithiasis unoperated, are likely to die of primary carcinoma of the gall-bladder, a relatively rare condition.

Frequency.—Autopsy records show that frequently in women of middle age and to a much less degree in males gall-stones are present. In women over sixty years of age the figures reach 25 per cent.

It is wholly probable that of all cases of cholelithiasis 80 per cent. show gastric dyspeptic symptoms only and wholly lack the classical picture and frank signs which depend almost wholly upon concurrent cholecystitis.

All dyspepsias coming on in middle age after preceding immunity should especially suggest cardiovascular disease, gall-stones or carcinoma.

Usually, their migration into the ducts give rise to biliary colic yet the passage of small stones may cause no pain whatever.†

* 7802 were found by Otto in one case, Archiv für Klinische Chirurgie, ci, 54, 1913.

† Naunyn's statement that 90 per cent. of the women who suffer from lithiasis have borne children would lead one to ask what proportion of the female population from which the cases are derived represents spinsters or barren wives. Unless this is stated the figure is valueless for statistical purposes.

Women are far more susceptible than men to cholelithiasis, three, four, or five to one according to the (inevitably fallible) statistics and this is ascribed in part to tight clothing, deficient breathing and lack of exercise. It has been asserted that 40 per cent. of the women patients show the deeply grooved corset liver, an assertion to be taken *cum grano salis*, yet, doubtless, expressing a general truth.

The ailment is one usually discovered only in middle age and is a very common accidental autopsy or operative finding.

Pain.

Biliary Colic.—*This is characterized by violent and abrupt pain in the right hypochondriac region or epigastrium, radiating to the shoulder-blade, with vomiting, sweating, circulatory depression and usually intermittent fever which may or may not be preceded by a sharp chill.*

Tenderness.

The tenderness and defensive muscular rigidity tend after a time to become localized in the region of the tip of the ninth costal cartilage, the liver and spleen may or may not be enlarged and the urine frequently contains albumin.

Jaundice variable.

If the stone be in the first or second portion of the cystic duct, there is no jaundice, or but a suggestion of it, indicating a complicating cholangitis.

If the stone is in the common duct or the terminal segment of the cystic duct, jaundice may be present, either persistent and severe, or slight and transient.

It is usually more intense and persistent if the terminal segment of the cystic duct is involved (pressure upon common or hepatic duct).

These attacks may or may not be repeated at irregular intervals until the stone passes onward or backward or is removed by operation. Death rarely occurs during an attack, though phlegmon, necrosis, rupture and general peritonitis are dread possibilities.

Chief diagnostic points.

The location of the pain in the upper abdominal and lower thoracic segment with localized tenderness (best elicited by deep pressure maintained both during full inspiration and expiration), and perhaps a history of previous attacks, are the chief distinguishing features. A case recently observed showed no tenderness over the liver or gallbladder even under forcible deep upward pressure in deep inspiration and expiration and the pain was invariably left-sided.

The stools should be carefully examined for calculi during and immediately after a supposed attack.

Misleading conditions.

Gall-stones, cholecystitis or adhesions from any cause may alike give rise to recurrent attacks of more or less severe pain, and an old gastro-duodenal ulcer or even pylorospasm may, through the same cause, be associated with symptoms hardly distinguishable from those of gall-stone colic.

Obstruction of the Common Duct.—Obstruction may be clinically absolute* with profound and persistent jaundice (pale stools, etc.), and be due to one or several stones, or it may be only partial with absent or only remittent jaundice. The so-called ball-valve stone is sometimes present producing paroxysmal chills with fever and sweating, persistent but variable jaundice

* According to the reports of B. B. Cronin and others bile is seldom wholly absent from the duodenal contents in common duct block, unless the cause is carcinoma.

(intensified during the paroxysm), hepatic pain and decided dyspeptic symptoms.*

Septic angiocholitis.

Suppuration may or may not complicate common duct stones, and empyema of the gallbladder and even hepatic abscess or perforation may occur.

Enlarged gallbladder.

Cystic Duct.—Even in complete cystic obstruction jaundice may be slight or absent, the gallbladder is distended, unless the case is one of long standing and atrophy of the gallbladder has occurred, and it may attain an enormous size. Complicating cholecystitis is common and is occasionally of the suppurative type with empyema of the gallbladder. Calcification of the walls and atrophy may occur and in some cases is so complete as to leave hardly a trace upon superficial examination. Fever is less often present than in stone of the common duct.

Atrophy.

Often obscure.

General Comment.—*The utmost diagnostic difficulties may be experienced in both acute and chronic forms of cholelithiasis or cholecystitis, and the greatest stress must be laid upon the location of the pain and the presence of localized tenderness, irrespective of jaundice which of course greatly assists the diagnosis when present.*

Chief symptoms.

A palpable gallbladder usually moves with respiration, has its point of attachment in the region of the ninth costal cartilage and is movable only in a small segment of the circle whose center is its attachment.

Jaundice.

It should be noted that jaundice of marked degree is absent in complete obstruction of the cystic duct, and a distended gallbladder is rare in obstruction of the common duct.

Riedel's lobe.

The linguiform process of Riedel and Cruveilhier (Riedel's lobe) is especially common in chronic disease of the gallbladder, though occasionally present in other abdominal ailments (Moynihan). It may lie to either side of, or overlay, the gallbladder, and is clinically a small freely movable but attached linguiform body, which lacks pendulum motion and has a sharp edge.

Malignant cases.

Stone of long standing in the common duct is usually associated with a contracted gallbladder. The same condition may occur in old cystic duct cases, but the usual stage observed is one associated with distention. In malignant disease blocking the common duct, there is profound and persistent and progressive jaundice, slight or no localized pain, but progressive weakness and emaciation. *Finally, all degrees of pain, fever and tenderness may be absent.*

* The combination of gall stone colic and chill almost always indicates a common duct stone.

Infection and Immunity

A Brief Summary of Some of the Important Principles Underlying

THE "WASSERMANN" REACTION AND CERTAIN OTHER TESTS INVOLVING COMPLEMENT-FIXATION, AGGLUTINATION, PRECIPITATION AND ALLERGY

Present Status of the Doctrine of Immunity.—The question of the mechanism of immunity is in a state of flux. The ingenious and helpful, but complex theory of Ehrlich is somewhat severely shaken by the most recent developments in Serology and the entire theoretical groundwork is likely to undergo many radical changes in the near future.

Its most vital phenomena, however, as embodied in its practical application, are relatively straightforward and lend themselves to simple description. Many permit also their clinical use at the hands of any man who is educated along modern lines, even though not a serologist.

The "Wassermann Test" is not of that group. On the contrary, when it is in any but expert hands, the findings are not only untrustworthy but misleading and most calamitous in results with respect to errors of omission and commission alike.

Nature of the Wassermann Test.—It is a test of the fixation or non-fixation of complement in the blood serum or spinal fluid of an individual suspected of syphilitic infection, past or present, and its intelligent discussion involves a brief review of both infection and immunity.

Infection.—Reduced to its simplest terms "infection" means the invasion of the body by pathogenic parasites, animal or vegetable, under conditions which so favor or permit their lodgment, persistence, and *reproduction*, as to produce direct or indirect injury to their host.

Arbitrary Divisions.—The line drawn between infection by bacteria or fission-fungi and that due to the parasitic worms or insects rests largely upon the greater size of the latter and their greater tendency to cause *direct* damage to their host, either of themselves or through their larvæ.

This line is not a clearly defined one inasmuch as mass-effects such as thrombi and emboli may be produced by microparasites, and most virulent tissue poisons by macroparasites.

Bacterial Toxins.—The chief pathologic effects of pathogenic bacteria result from the action of soluble poisons, specific for the individual bacterial species, and representing either a secretion or excretion of the bacterium involved or a pathogenic element of its cytoplasm liberated at its death or elaborated during its gradual dissolution.

Exotoxins and Endotoxins.—A distinction is drawn between "true" toxins ("Exotoxins") such as those of diphtheria, tetanus, B. botulinus and B. pyocyaneous, all products of cell metabolism, secretion or excretion, and "endotoxins" bound by the cell cytoplasm during its life and released only by its death and disintegration.

"*Exotoxins*" are present in liquid culture media containing such of the pathogenic parasites as produce them, and alone give rise to powerful "*antitoxins*" in infected individuals.

The "*endotoxins*" can be obtained readily, if at all, only after the death of the bacteria which contain them, and their effects are obtained by the use of such dead bacteria or to a less degree by extracts of such parasites.

Leucocytosis and Chemotaxis.—Various substances stimulate the production of leucocytes and determine their localization and accumulation at the point of maximal tissue reaction, *i.e.*, they are "chemotaxis-inducing substances" best exemplified in the case of the pus-producing (pyogenic) cocci but in some degree resident in all bacteria pathogenic or otherwise as well as in the products of cell disintegration whether of the tissues or of the leucocytes themselves.

This phenomenon sometimes wholly fails (negative chemotaxis).

Bacterial Hemolysis.—The growth and multiplication of many species of bacteria within the living tissues is associated with a marked destructive effect (hemolysis) upon the red-blood cells, these yielding up their hemoglobin to the blood plasma.

Ptomains.—These bacterial products, toxic or non-toxic, and resembling alkaloids, are formed without the body, and depend for their production largely upon the nature of the culture medium and for their toxicity chiefly upon the oxygen supply.

Any one of several bacterial species may produce the same ptomain. Their chief importance lies in the clinical domain of food poisoning (meat, milk, etc.).

Selective Affinities and Effects of Toxins.—Most astonishing selective localization and tissue affinities are demonstrable with relation to many bacterial toxins, a notable example being afforded by that of tetanus which promptly seeks a *locus* in, and exerts its chief pathogenic effects upon, the central nervous system.

IMMUNITY.—There are several forms of immunity; *i.e.*, (*a*) that which is inherited, *i.e.*, "*natural*" *immunity;* (*b*) that which results from poisoning or infection and toxemia, by substances or microörganisms capable of conferring immunity upon their host, *i.e.*, "*acquired*" *immunity:* (1) that which is artificially induced by systematic inoculation with graduated doses of bacteria or toxins, *i.e.*, "*active*" *immunity;* (2) that which is produced by graduated systematic doses of immune substances recovered from immunized animals, *i.e.*, "*passive*" *immunity.*

Mechanism of its Production.—The vital factors in immunity are relatively simple; the complexities of their variants, enormous.

The toxicity of specific bacterial poisons varies greatly with respect to the species and even the strain producing the toxin, and in most instances equally so with respect to the individual attacked, whether the bacteria carry endotoxins or exotoxins. "Dosage" and strain-virulence are also important factors, *but even against such poisons as diphtheria-toxin and the virulent culti-*

vable filtrable virus of acute infectious poliomyelitis ("infantile paralysis") many individuals possess a high degree of natural immunity.

Requisites for Immunity.—*Obviously immunity must depend upon (a) some peculiar characteristics of the tissues which make them inimical to bacterial growth; (b) the blocking of channels of entrance, or (c) the possession by the body cells and fluids of properties or qualities necessary to the prompt organization of an effective defensive mechanism.*

Antibodies.—It is wholly rational to assume such a defensive mechanism for body tissues and to assert that not only must bacteria be destroyed or rendered inert, but that their toxins must, in some degree, be neutralized or so "bound" as to be rendered harmless.

We find these requirements met by the production of "*bacteriolysins*," "*immune-opsonins*," "*agglutinins*," "*precipitins*," "*cytolysins*," "*antitoxins*" *and the like.*

When one attempts to formulate *exact processes* he at once enters the realm of speculation. All that we know positively is that certain phenomena occur, which are readily demonstrable and, in great part, easily reproduced in the test-tube ("*in vitro*").

Correlation of these phenomena permit certain general deductions of a most helpful and constructive sort, but of the exact nature and form of the basic reactions we know little or nothing.

Processes Producing Immunity.—Phagocytosis, the envelopment and destruction of bacteria by the leucocytes is a readily demonstrable process, but certainly not the chief and most effective one.

Opsonins.—These will be discussed further on, but the readers will recall that their assumed function is to so affect the invading bacteria as to prepare them for, or to facilitate, their inclusion and digestion by the leucocytes.

How great the part played in immunity by phagocytosis and opsonic substances, we do not know.

In all forms of immunity, natural, acquired or artificial, it is wholly probable that the same factors are dominant in each.

Antigens.—This term applies not alone to bacteria, animal parasites, or filtrable viruses, but to proteins, which act as poisons to the tissues, either directly, or through split products. To a large degree they cause *specific defensive responses* on the part of the organism attacked, in so far as these may be determined by the selective activity of the so-called *antibodies* produced.

That these "*specific*" *antibodies* are not always specific with reference to the bacterium itself, but may react chiefly or only to substances liberated or elaborated as the result of a specific infection, is demonstrated by the later findings with reference to the Wassermann reaction, in which it is evident that the antigen best calculated to demonstrate the presence of *syphilitic* antibodies is obtainable from certain normal tissues (heart-spleen),* and

* Noguchi's antigen is the acetone-insoluble heart-spleen residue obtained by extraction with alcohol, evaporation, solution in ether and precipitation by acetone. A portion redissolved in ether and shaken up in salt solution until fine suspension is attained, is ready for use.

consists largely of cholestrin, sodium oleate and lecithin. On the other hand it is found that the *Treponema pallidum* itself or an extract of it cannot successfully be employed.

These disconcerting findings do not affect the fact that the Wassermann reaction seems clinically specific for syphilis.*

Bacteriolysis.—A direct knowledge of the phenomena attendant upon the destruction of bacteria in specific immune serum arrived by successive steps of which Pfeiffer's reaction was the most clarifying.

Bacteriolysis was first reported by Pfeiffer in 1894 who discovered that living cholera *vibrios* introduced into the peritoneal cavity of a guinea-pig previously rendered immune to cholera were rapidly destroyed after preliminary loss of motility.†

From this it was evident that the serum of an animal rendered immune to any bacteria or their toxins may, under proper and known conditions, destroy such bacteria and *dissolve* them (*bacteriolysis*).

It was shown further that exactly the same destructive process might be demonstrated in a test-tube ("*in vitro*"), containing living cholera-vibrios (or extracts of dead cultures), and the serum of a cholera-immune, provided that the serum had not been heated to 56°C. or more. Once so heated it was inert ("*inactivated*") and so remained until some fresh serum, *immune or unimmune*, was added when it immediately became "*activated*," and lethal for the specific microörganism.

It was thus shown that immune serum contains substances lethal for the specific species of bacterium producing the immunity and that the antibacterial activity depends in great part upon a "thermolabile" and a "thermostabile" substance, both of which must be present in the serum.

It also proved the specificity of the thermostabile (resistant to heat), substance, *and the lack of specificity in its thermolabile (non-resistant to heat) associate.*

Substance Sensibilatrice.—The "thermostabile" substance, "*Substance Sensibilatrice*" (*Bordet*), or "*Amboceptor*" (*Ehrlich*) is permanent when once formed for defense against the specific pathogenic microörganism, or its toxin.

The "Alexin" or "Complement."—The "thermolabile" body ("Alexin," *i.e.*, "Complement") exists in all fresh serum, but is relatively impermanent outside the living body.

The conditions described might be represented thus, in relation, for example, to cholera vibrios.

Fresh Normal "non-immune" serum ("complement" only) + cholera vibrios = No effect.

Fresh "Immune" serum unheated ("specific amboceptors" only) + vibrios = No effect.

Fresh "Immune" serum unheated ("complement" and "specific amboceptors") + vibrios = Bacteriolysis.

*The only exceptions now known being leprosy, sleeping sickness, scarlet fever and "yaws."

† The agglutinins were first thoroughly studied by Gruber and Durham in 1896, the "precipitins" by R. Kraus in 1897. The *action* of each was manifested in Pfeiffer's original procedure.

Fresh "Immune" serum heated ("specific amboceptors" only) + normal serum ("complement") + vibrios = Bacteriolysis.

It is evident that the specific antibodies cannot destroy the bacteria if complement (alexin) is lacking. Furthermore, we know that if the amount of complement added to an immune serum falls below a certain amount, or, if this complement is in part already bound by other immune bodies, bacteriolysis is incomplete (*"complement deviation"*).

COMPLEMENT (ALEXIN FIXATION).—*From the foregoing it is evident: (a) that complete binding and destruction of the invading bacterium demands specific amboceptors; (b) that these are helpless without complement; (c) that this substance itself becomes bound ("fixed") in the bacteriolytic process.*

Fundamental to Test.—It therefore follows that if any properly proportioned mixture, consisting of inactivated *immune serum, its specific antigen, and fresh complement*, were placed under conditions of time and temperature, most favorable to bacteriolysis, all *complement* would become *"fixed"* (bound) and, because of that fact, the serum could not afterward be used to supply complement for the "activation" of any other.

If such an inactivated serum was *not immune* to the specific antigen employed the "complement" would not become fixed, when added, and the serum could supply it to, and thereby activate, any other serum.

Thus, in the case of the reaction of freshly activated serum, from a patient suspected of a disease capable of conferring immunity, the behavior of its complement toward a known immune serum containing antigen and specific amboceptor, but no complement, determines the presence or absence of specific amboceptor in the patient's serum and the existence or non-existence, past or present, of the suspected ailment.

The Hemolytic System.—In hemolysis, the counterpart of bacteriolysis, the substance brought into solution is the hemoglobin of the red blood cell ("laking of the blood").

Positive Test for Hemolysis.—Rabbits may be immunized against the erythrocytes of the sheep, which, ordinarily, would produce hemolysis, and their immune serum will destroy washed sheep's erythrocytes and indicate the solution of their hemoglobin by assuming a red coloration.

Negative Test for Hemolysis.—If such immune serum is robbed of its complement by heating (inactivated), before the washed sheep's cells (antigen) are added, no hemolysis occurs and the mixture shows only a red sediment and translucent upper stratum.

These phenomena of hemolysis become extremely useful in connection with the complement fixation tests, as a means of determining the question of free or bound complement in suspected sera through a striking color change.

Application to Syphilis.—If to a heated (inactivated) serum taken from a patient *suspected* of syphilis, we add the correct amount of fresh complement (guinea-pig serum) and a *carefully proportioned* amount of the proper antigen, and incubate the mixture to promote the activity of any specific amboceptors which it may contain, we know that the complement will become wholly bound *if the patient is a syphilitic.*

If one has some of the serum of a rabbit immune to sheep's blood, heats it to destroy its complement, and treats it with washed sheep's corpuscles (antigen), all of the elements of an hemolytic system are present, save one, and it will produce hemolysis so soon as "complement" shall be supplied, and not before.

If, therefore, this is added to the foregoing incubated mixture and hemolysis occurs, it proves that the serum of the syphilis suspect was free from syphilitic antibodies, contained unfixed complement, and through it, was able to complete the hemolytic system, necessary to hemolysis.

If hemolysis does not occur in such an instance, syphilis is proven by the fact, thus established, that binding ("fixation") of the complement of the suspected human serum must previously have taken place and rendered the syphilitic serum incapable of activating any other.

All this sounds simple, but in reality involves a maze of complexities and opportunities for damaging error. The commoner methods of actual application of the test, as described by Zinsser* in his admirable book, are quoted here:

THE WASSERMANN REACTION

Titration of Hemolytic Amboceptor or Sensitizer

Titrations of both sensitizer and alexin should be made. For practical purposes it is quite enough to titrate the hemolytic sensitizer every few weeks and use a stated amount in successive reactions. The alexin or complement can then be titrated individually for each set of reactions. Examples of such preliminary titrations follow:

Rabbit injected 3 times at 5-day intervals with washed sheep corpuscles . . . . ., 3, 4, and 5 c.c., and bled 10 days after the last injection.†

This serum is inactivated at 56°C. for 20 minutes.

	Washed sheep corpuscles 5 per cent. emulsion in salt solution	Sensitizer	Fresh guinea-pig serum	Hemolysis
1	1 c.c.	0.01	0.1	+++
2	1 c.c.	0.005	0.1	+++
3	1 c.c.	0.003	0.1	+++
4	1 c.c.	0.001	0.1	+++
5	1 c.c.	0.0005	0.1	++
6	1 c.c.	0.0002	0.1	±
7	1 c.c.	.....	0.1	
8	1 c.c.	salt sol.		

* "Infection and Resistance." Hans Zinsser, The Macmillan Co., New York, 1914.

† In immunizing animals with blood cells for this or any other purpose it is necessary to wash the cells very carefully in salt solution. Unless this is done blood serum or plasma will be injected with them and the treated animal will respond by the formation not only of hemolysin but of precipitins for the serum proteins as well. When a subsequent hemolytic test is carried out, a precipitin reaction between the precipitin in the antiserum and serum adhering to the corpuscles will follow, and this, as we have seen, will fix alexin, obscuring other reactions which may be under observation.

In this case 0.001 c.c. still causes complete hemolysis of 1 c.c. of a 5 per cent. emulsion of sheep cells (volumetric measurement of cells sedimented in the centrifuge), and this amount ($\frac{1}{1000}$c.c.) is called the "hemolytic unit" of sensitizer; two units are then used in the reactions.

Against these cells alexin can, in each case, be titrated as follows:

Alexin Titration:

FRESH GUINEA-PIG SERUM PIPETTED FROM CLOT

	Red cells 5 per cent. emulsion	Sensitizer as above determined	Guinea-pig serum	Hemolysis
1	1 c.c.	2 units (0.002)	0.1 c.c.	+++
2	1 c.c.	2 units (0.002)	0.05 c.c.	+++
3	1 c.c.	2 units (0.002)	0.025 c.c.	±
4	1 c.c.	2 units (0.002	0.01 c.c.	

The smallest amount of alexin which completely hemolyzes the red cells (0.05 in this case) is the amount used. Since it is easier to measure larger volumes with accuracy, the alexin is diluted 1 to 10 in salt solution before use. A typical Wassermann reaction can then be carried out as follows:

SCHEME FOR WASSERMANN TEST

ADAPTED TO ORIGINAL WASSERMANN SYSTEM AFTER SCHEME OF NOGUCHI

	Test with unknown serum	Test with known positive syphilitic serum	Test with known negative normal serum	Test without serum to control efficiency of hemolytic system
Back row without antigen	Serum 2 c.c. + O Complement 0.1 c.c. + Salt sol. 3 c.c. 2.	Serum 0.2 c.c. + O Complement 0.1 c.c. + Salt sol. 3 c.c. 4.	Serum 0.2 c.c. + O Complement 0.1 c.c. + Salt sol. 3 c.c. 6.	O Complement 0.1 c.c. + Salt sol. 3 c.c. 8.
Front row with antigen	Serum 0.2 c.c. + O Complement 0.1 c.c. + Antigen (required amount in 1 c.c. salt sol.) + Salt sol. 2 c.c. 1.	Serum 0.2 c.c. + O Complement 0.1 c.c. + Antigen + Salt sol. 2 c.c. 3.	Serum 0.2 c.c. + O Complement 0.1 c.c. + Antigen + Salt sol. 2 c.c. 5.	O Complement 0.1 c.c. + Antigen + Salt sol. 2 c.c. 7.

Total volume, 3 c.c.

Total volume, 3 c.c.

O = Test-tube.

Place in water bath at 40°C. for one hour, *then add to all tubes washed red blood cells* (of sheep) *and hemolytic amboceptor.* These are previously mixed so that 2 c.c. contains the equivalents of 1 c.c. of a 5 per cent. emulsion of sheep corpuscles and 2 units of amboceptor. Again expose to 40°C.

If the serum tested is positive, tubes 1 and 3 should show no hemolysis, all the other tubes showing complete hemolysis in one hour.

Since many human sera normally contain small amounts of antisheep sensitizer, it is the habit of many workers to add the sheep corpuscles, without the sensitizer or amboceptor, and incubate for a half-hour. If, at the end of this time, no hemolysis has occurred either in the front or the back row, then amboceptor may be added. This technic avoids the possible error introduced by an excess of amboceptor, a condition which easily occurs when any large amount is normally present in the serum and in addition to this 2 units are added as in the test described above.

The foregoing represents the typical "Wassermann" as at present carried out in most laboratories. It may be carried out just as well and with greater economy of material by using one-half the amounts throughout. It is evident that the performance of the reaction calls for experience of serum technic, and knowledge of such reactions, so that fortuitous irregularities may be intelligently controlled. It is our opinion that the performance of routine Wassermann tests by workers without a thorough knowledge of the fundamental facts of serum phenomena is worse than useless in that insufficient attention to special conditions and to details may easily result in a positive reaction when syphilis is not present, and *vice versa.*

Many modifications of the Wassermann test have been suggested. Probably the most important is that of Noguchi. The chief justification for this modification is the fact that many normal human sera contain hemolysins for sheep corpuscles. For this reason many workers carry out the ordinary Wassermann technic without adding antisheep sensitizer or amboceptor until they have first observed whether or not the tested serum (in the "back row," without antigen) will not hemolyze the corpuscles without such an addition, adding the sensitizer only when this does not take place. This is advisable since the presence of any considerable amount of normal antisheep sensitizer in the human serum which is being examined (if added to the amount used in the ordinary reaction, 2 units), may so increase the total quantity that hemolysis will result even after most of the alexin has been fixed. Noguchi excludes this uncertainty by avoiding the use of the "sheep-cell-antisheep sensitizer" system entirely, substituting a hemolytic complex consisting of human cells and antihuman sensitizer, produced by injecting washed *human* corpuscles into rabbits.

His technic may be best illustrated in the following tabulation:

Reagents

1. Sensitizer prepared by injecting washed human blood corpuscles into rabbits.
2. 1 per cent. emulsion of washed human blood cells.
3. Alexin—fresh guinea-pig serum diluted with one and one-half volumes of salt solution, 40 per cent

The reaction is performed in the following way:

NOGUCHI'S METHOD OF COMPLEMENT FIXATION FOR THE SERUM DIAGNOSIS OF SYPHILIS

	Set for diagnosis Test with the serum in question	Positive control set Test with a positive syphilitic serum	Negative control set Test with a normal serum	
Rear row	a. Unknown serum, 1 drop* b. Complement, 2 units O c. Corpuscle susp., 1 c.c.	a. 'Positive syph. serum, 1 drop* b. Complement, 2 units O c. Corpuscle susp., 1 c.c.	a. "Normal serum, 1 drop* b. Complement, 2 units O c. Corpuscle susp., 1 c.c.	Incubation at 37°C. for 1 hour.
Front row	a. Unknown serum, 1 drop* b. Complement, 2 units O c. Corpuscle susp., 1 c.c. + Antigen	a. 'Positive syph. serum, 1 drop* b. Complement, 2 units O c. Corpuscle susp., 1 c.c. +Antigen	a. "Normal serum, 1 drop* b. Complement, 2 units O c. Corpuscle susp., 1 c.c. +Antigen	Addition of antihuman amboceptor, 2 units to all tubes. Incubation at 37°C. for 2 hours longer, then at room temperature.

Since the reaction is not a specific antigen-antibody union but depends on some substance liberated or produced by reason of the syphilitic infection, it is not out of question that other infections may give rise to a "positive Wassermann," and this, indeed, is the case. It was claimed for a time that a positive reaction may be obtained in tuberculosis, but this has been refuted by subsequent experience, and the earlier positive results probably depended upon faulty technic. There can be little doubt, however, that occasional positive reactions are obtained in cases of leprosy, scarlet fever, malaria, and trypanosoma infections.

The spinal fluid may be used instead of the blood serum in cases of syphilis of the central nervous system, but even here, as Citron† has shown, the results with blood serum are more frequently positive than those done with the spinal fluid itself. In isolated cases positive reactions have been obtained with ascitic fluids, pleural and pericardial exudates.

Controls.—In the first procedure it will be noted that tubes 1 and 3 contain a complete lytic system, *i.e.*, activated human serum, the one that of a known syphilitic; the other that of the suspect. No. 5 contains a serum *known* to be non-syphilitic. No. 7 contains no serum. Nos. 2, 4,6, 8, contain no antigen. All receive fresh complement (fresh, normal guinea-pig serum).

All are *incubated, merely*, for an hour. This incubation furnishes the ideal temperature, and adequate time, for the complete fixation of fresh complement in any syphilitic serum.

* When working with inactivated serum 4 drops (0.08 c.c.) should be employed. With cerebrospinal fluid 0.2 c.c. (not inactivated) is used.

(Taken from Noguchi's "Serum Diagnosis of Syphilis," Lippincott, 1910, p. 57).

† Citron. *Deut. med. Woch.*, 1907, No. 29, p. 1165.

Addition of Hemolytic System.—Adding the washed red-blood cells of the sheep and the serum of rabbits immunized against them, but no complement, must result in hemolysis in all tubes containing previously unfixed complement. This we know is true of all save tubes 1 and 3, and certainly not true of 3, which contains known syphilitic serum. It is evident that complement remains unbound *certainly* in every tube save 1 and 3; must be bound in both of these if syphilis is present in the suspect or remain free in 1 if the reaction is negative for syphilis.

From what has gone before it is obvious that the following events will ensue: (See page 907.)

Tube 1 (Suspect) may or may not fix the added complement. *Tube* 3 must fix complement for the source of the serum is a known syphilitic. No change in color.

Tubes 5 *and* 7 will not affect complement; the former being from a known normal, the latter lacking serum altogether.

Tubes 2, 4, 6, 8 contain no Antigen, the last also, no serum, hence complement remains unaffected.

The entire test depends directly upon the behavior of No. 1 *containing the suspected serum.*

If, together with all the tubes save No. 3, it shows the diffused red of the extracted and dissolved hemoglobin, it is not a syphilitic serum.

If it remains actually unchanged, or but slightly so, it is positive.

Results Obtainable with the Wassermann Test.—It is negative in at least 50 per cent. of syphilitics of the first stage (four to six weeks).

In tertiary cases at least 20 per cent. escape, although this figure is bettered if the so-called provocative treatment with anti-syphilitic remedies is first used.

In secondary syphilis the positives should run nearly 100 per cent., if no treatment has been had, or, if a period of four weeks, at least, has elapsed since its discontinuance.

Even imperfect treatment may abolish it, while not affecting the luetin test.

As stated previously, one should not accept negative findings in the fact of positive clinical evidence, or grounds for strong presumption.

The mere statement of the patient that a Wassermann has been made and was negative, should not be accepted in most instances until directly verified and never, if the test has been performed by anyone other than a serologic expert.

In cerebrospinal lues the Wassermann may fail occasionally in the blood serum and be positive in the spinal fluid, and *vice versa*.

The principle of the complement fixation test is now clinically applicable to the following conditions: (*a*) *Gonococcus infections* of at least four or five weeks' duration. (*b*) *Echinococcus invasion.** (*c*) *Glanders*. (*d*) *Typhus* fever, according to Olitzky's latest reports. (*e*) Tuberculosis. (*f*) Typhoid fever (Garbot's method with polyvalent antigens.)

* Probably a group reaction applicable to various *tenia*.

Antitoxins.—The name aptly expresses the character of these substances and in instance of natural immunity they may be shown to play a large part. So far as known, they occur primarily only as the result of Exotoxin activity. The production of antitoxin and its therapeutic application are too well known to require description here.

SCHICK'S TEST OF DIPHTHERIA IMMUNITY.—This test depends upon the results of the *intradermic* (not hypodermic) injection of diphtheria toxin in an amount representing one-fiftieth ($\frac{1}{50}$) of the minimum lethal dose for a 250 gram guinea-pig, as determined previously by proper laboratory tests.

The required dose may be so diluted as to represent a convenient bulk for injection (0.2 c.c.) and introduced by pinching up a fold of skin, introducing a very fine needle, with the barrel of the hypodermic syringe lower than the needle point, until the point of the needle is visible just under the epidermis, but not protruding. The test solution is then forced out and forms a bleb upon the surface. In most instances a typical inflammatory skin reaction develops by the end of thirty-six hours, but may be delayed until the lapse of seventy-two, and very rarely ninety-six hours.

True Reaction.—The best test of the true reaction is apparently a decided tenderness to pressure associated with decided reactive inflammation.

Induration is likely to be present and is best detected and estimated by pinching up the area involved.

Doubtful reactions and pseudo-reactions are common and demand that the individual exhibiting them be given a prophylactic dose of diphtheria antitoxin, unless the observer has had a wide experience with the test.

Recent reports attest the genuine value of the procedure, both in relation to preventing the disease and checking epidemics, and in sparing suspects from the unnecessary injection of prophylactic doses.

Dr. A. Zingher has devised a simple outfit for the distribution of toxin for the Schick test,* and this provides for the simultaneous application of a control test which eliminates almost wholly the danger of misinterpretation of the pseudo-reactions, which are caused by *protein* content of the test serum, not by the toxin itself. This outfit, as previously described by him, "consists of a capillary tube which contains a little over one minimum lethal dose of a ripened diphtheria toxin, a small rubber bulb for expelling the toxin, and a 10 c.c. bottle of normal saline solution for diluting the toxin. Every 0.2 c.c. of the dilution represents one-fiftieth minimum lethal dose, the amount used in the Schick test.

"The outfit for controlling the positive, pseudo and combined reactions is similar to the Schick test outfit, but the toxin in the capillary tube has been heated to 75° C. (167° F.) for five minutes. Heating the toxin at this temperature destroys the soluble diphtheria toxin, but does not appreciably affect the protein of the diphtheria bacillus."

Precipitin Test for Human Blood.—This is an extremely important, wonderfully delicate, and accurate, forensic test and has been used extensively also to determine species relationships in animals (Nuttall).

* Jour. A.M.A., Vol. 66.

Technic.—One uses the serum of a rabbit so immunized as to yield precipitation in dilutions up to 1 to 10,000 under proper titration against human serum. With 1 to 1000, clouding should occur in three minutes at room temperature, and be very distinct in eight.

This constitutes the "*Antiserum.*" Five tubes are used as indicated below:

Test.—

Tube 1.	Unknown human serum 1 to 1,000	1.0 c.c. +	Antiserum 0.2 c.c.
Tube 2.	Unknown solution to be tested	1.0 c.c. +	Antiserum 0.2 c.c.
Tube 3.	Unknown solution to be tested	1.0 c.c. +	Normal rabbit serum 0.2 c.c.
Tube 4.	Salt solution	1.0 c.c. +	Antiserum 0.2 c.c.
Tube 5.	Unknown solution	1.0 c.c. +	Salt solution 0.2 c.c.

(*Fornet and Müller*).

In each case the antiserum is first introduced and the solution to be tested is allowed to flow gently and slowly down the side of the inclined tube upon the surface of the underlying fluid.

At the junction of the two a fine white ring will appear in tubes 1 and 2 if the test is positive, the others remaining unchanged. The reaction may be positive for human blood in stains many years old, if these have been continuously dry and in the dark.*

Meningococcus Test.—(*Vincent-Bellot.*) To 3–6 c.c. of centrifugated cerebrospinal fluid, add one drop of meningococcus serum. Set the test liquid aside for 8 to 12 hours at a temperature of 50 to 53°C.

These investigators claim that in meningococcus infections a precipitate is demonstrable even though the specific microörganism is not to be found.

Agglutination.—The most important of the agglutination reactions are discussed elsewhere.

It should be remembered that they are specific when dilutions of not less than 1–40 to 1–60 are used and are as readily demonstrable with dead bacteria as with the living organisms.

In dilutions under 1–20 the normal agglutinins of the serum may prove deceptive factors.

As stated elsewhere, dead bacteria, living ones taken from solid cultures, or those grown in bouillon, may be used, but the latter should not be too young.

Though more or less devitalized, bacteria are not killed by the process of agglutination alone.

Agglutination Reaction.—Our present understanding of the phenomena constituting the so-called agglutination reaction which has proven so valuable in the diagnosis of certain obscure diseases depends upon the known fact that injected erythrocytes or other cells, not those of the animal into whose blood they have been introduced, or, bacteria similarly injected, produce in the blood serum of the animal so treated, the peculiar quality of agglutinating and precipitating, *in vitro*, these foreign elements, and even of

* Extracts of the muscles of the neck and leg of a mummy of the first Egyptian Empire yielded a faint reaction according to Meyer, Munch. Med. Wochen., 1904, Vol. 51, No. XV.

producing agglutinins and precipitins specific for a given microörganism. This involves the assumption of agglutinins and precipitins as products evolved during adaptation or immunization. The clinical application of this process has proven of value in a number of cases and is best illustrated by typhoid fever in which disease is found its first clinical use through the work of Widal based upon the previous investigations of Pfeiffer and Gruber and Durham, who worked along purely bacteriological lines seeking to apply the phenomena to the identification of bacteria.

The agglutination reaction of typhoid, paratyphoid, colon infections, dysentery, cholera, plague, Malta fever and to a much less degree of pneumonia and tuberculosis have proven valuable.

An early and definite reaction is chiefly marked in typhoid, paratyphoid, Malta fever and cholera. In paratyphoid one is sometimes embarrassed because of the fact that the paratyphoid bacilli of different epidemics may not react. In dysentery there are three types of bacilli to be dealt with, viz.: those of Flexner, Shiga and Hiss, while in plague the reaction is not constant and occurs too late to be of use, and the same objection of inconstancy applies to tuberculosis and pneumonia. (For Widal Test see Typhoid Fever.)

Anaphylaxis (*"Hypersusceptibility," "Allergy"*).—This phenomenon, which may be found to constitute the very foundation of immunity, was reported fully first by Hericourt and Richet in 1902.

Isolated observations were made by Magendie (1839) and S. Flexner (1894) clearly stated the basic phenomena.

Their discovery first established the fact that the production of increased tolerance and resistance to antigens of any nature attained through the usual methods employed in the production of artificial immunity, have decided limitations and demand a close adherence to the terms imposed by certain conditions.

This subject is so beset with contradictory opinions and conflicting observations at the present time as to make an intelligent discussion impossible in a book of this kind.

Speaking broadly, the most important phenomena are those of abnormal sensitization and excessive response of body tissues to foreign proteins, from whatever source, reaching the blood or lymph in an unaltered state.

A specific sensitization may be inherited, may result from inunction, surface applications, inhalation, ingestion, irrigations, enemata, or any other method related to the taking of food or the administration of medicament.

The most striking clinical analogies at once suggested are: *idiosyncrasies with relation to foods, drugs, odors, and the like, hay-fever and spasmodic asthma.*

It is proven experimentally that specific sensitization may pass from mother to child, or be passed on from one directly and artificially sensitized animal ("active anaphylaxis") to another ("passive anaphylaxis").

The amounts of foreign protein necessary to produce specific sensitization are incredibly small.

$\frac{5}{100,000,000}$ of a grain of crystallized egg albumin can produce demonstrable sensitization in a guinea-pig (H. G. Wells).

The chief phenomena are: (1) *Excessive reaction.* (2) *Decreased duration of symptoms.* (3) *A shortened incubation period.* (4) *A fleeting period of immunity to subsequent injections of the same foreign protein (anti-anaphylaxis).* (5) *Absence of complement from the blood of the animal affected.* (6) *Diminished blood-coagulability.* (7) *Leukopenia.*

SERUM DISEASE.—The first clinical observations on anaphylaxis were made by von Pirquet in a case of diphtheria. We now know that any foreign protein in solution, when injected into the living tissues, may so sensitize the individual to the particular protein employed, as to produce pathologic systemic or localized reactions of a character wholly foreign to any symptoms originally produced or an exaggerated response within a greatly shortened period.

The same statement holds with respect to many substances regardless of the method or route of their introduction, and it now appears that hypersusceptibility may also represent maternal inheritance or result from the direct transfer of serum from a sensitized to an unsensitized animal. Furthermore the hypersusceptibility may last for years in certain instances.

Symptoms of Serum Disease.—The chief manifestations of serum disease are (*a*) Urticaria. (*b*) Swelling of lymph nodes. (*c*) Edema, which may be shifting and transitory and in some instances seriously threatens life by invasion of the larynx. (*d*) Joint pains. (*e*) Fever. (*f*) Albuminuria and casts.

It is true also that it is the *amount of serum, from the same class of animals,* and not the antitoxin content which is the determining factor. When second injections of diphtheria antitoxin are demanded, after a lapse of one week or more, the use of the concentrated sera, and, if possible, of antitoxin prepared from animals other than the horse, are elements which make for safety.

Death may, but very rarely does, occur from the therapeutic use of sera, and in such instances seems to result from the same condition which determines *exitus* in the anaphylactic shock of the guinea-pig, viz. *acute bronchial spasm,* resulting acute distention of the lungs, and asphyxia. The suggestive resemblance of such a critical and lethal phenomenon to the attack of ordinary bronchial asthma is striking and led to the suggestion of anaphylaxis as the basic cause in 1910.

OPSONIC THEORY*

It is not strange that the earlier enthusiasm regarding the opsonic diagnosis and therapy has been followed by disappointment in certain directions, for the results so far obtained have not yielded a reward commensurate with the tedious, delicate and elaborate technic required, nor is this technic

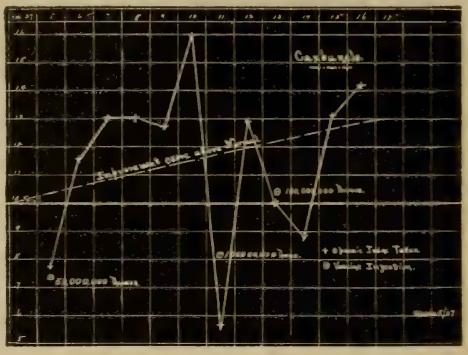


FIG. 475.—Record curve of patient with large furuncle on back of neck. (Courtesy of F. M. Houghton.)

free from serious faults, especially as regards the preparation and, more especially, the use of vaccines, but the procedure may yet prove to be the key to a larger knowledge and more accurate diagnosis, prognosis and therapy. All medical progress seems to partake of the nature of a persistently lengthening ascending spiral.

FIG. 476.—Collecting blood for serum.

Metchnikoff described phagocytosis; Denys and Leclef, the increased activity of the phagocytes in the presence of blood serum. Leishman developed a method for measuring phagocytic activity and Wright and Douglas formulated a new application of the doctrine of phagocytosis which is of sufficient scientific and historic interest to justify the retention of its

* For the illustrations, with one exception, the author is indebted to the courtesy of Dr. E. C. L. Miller.

description in this edition even though it has already fallen into disuse both on the diagnostic and therapeutic side.

These experimenters have found that the blood contains a limited amount of certain bodies necessary to prepare germs for destruction by the phagocytes of the body. It appears that if white blood corpuscles are thoroughly and repeatedly washed in normal salt solution until every trace of blood plasma is removed and then brought into contact with certain germs, the result is almost negative as regards phagocytosis. If, however, a little blood serum be added, the germs are at once acted upon by the white cells, and, furthermore, if the microbes brought into contact with the washed cells without blood serum have been previously incubated with such serum, they fall easy victims to the phagocytic cells, unless the serum has been previously heated to 65°C., in which case no phagocytic action appears.

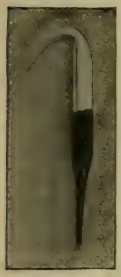


FIG. 477. Pipette with blood.

It thus becomes evident that normal blood contains a certain substance ("*opsonin*") destroyed by heating, but otherwise capable of rendering the bacteria vulnerable to the attack of the phagocytes or fit for phagocytic digestion.

It is evident, moreover, that the action of the phagocytes is dependent not upon any quality inherent in these cells, but upon the amount of an extracellular opsonic substance contained in the serum of a given blood. Certain bacilli, *e.g.*, those of diphtheria, are immune to opsonic action, but may be taken up to some extent by washed leucocytes (spontaneous phagocytosis).

FIG. 478.—Grinding bacteria.

Further experiments establish the fact that in relation to different varieties of germs it is possible to establish a normal index of phagocytic activity, this index being represented by the number of germs of a given sort taken up by a definite number of washed leucocytes obtained from the blood of any individual, normal or abnormal, but treated with normal serum, and that in such cases the index is practically constant, whereas, in persons suffering from diseases caused by the different germs, the opsonic index is variable both as to individuals and as to the time or stage of the disease

at which the estimation is made. Yet again it is found that if a given individual suffering from a certain disease, such as tuberculosis, receives a fixed dose of dead bacilli, said injection is followed by a change in the opsonic index. The first effect is to depress the phagocytic power of the patient's blood

FIG. 479.—Emulsifying bacteria.

(negative phase); on successive days, however, the opsonic index rises above normal (phase of flow and reflow) and finally reaches a maximum (high tide) at last reaching the stage of recession (the ebb phase). From the negative phase to that of high tide several days may be consumed, then follows for two or three days the gradual return to the normal level. If at this period a second inoculation be made the same phenomena are observed with a shortened and lessened negative phase and higher flow.

While subject to many inaccuracies and not adapted to general diagnostic use, the presence of these phenomena is of some diagnostic, prognostic, and therapeutic interest, and furnishes an interesting sidelight upon relative individual susceptibility to the infectious diseases.

Technic.—Some blood of a normal individual, or, better the "pooled" blood of several, and a corresponding amount of that of the patient or suspect is drawn by gravity and capillary action into separate tubes (Wright's tubes) with capillary extremities and a curved shorter limb. The upper limb is then heated and sealed in the flame and the blood is drawn by the resulting condensation of the rarefied air into the body of the tube which is inverted and centrifuged to bring it into the longer arm, then allowed to rest until the clot begins to separate, and again centrifuged. Finally,

FIG. 480.

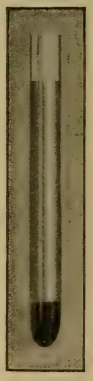


FIG. 481.

Blood and citrate solution before and after centrifugalizing. (Plain tubes.)

the capsule is cut across above the fluid level and the serum forming the upper stratum is removed with a pipette. A bacterial emulsion is then prepared from cultures of the required germ (bacillus tuberculosis, gonococci, staphylococci, etc., sterilized by heat) by grinding the germs in an agate mortar with a sterilized 0.85 per cent. solution of sodium chloride until an apparently uniform mixture is obtained. After sedimentation and removal of the upper layer, this coarse emulsion is drawn into Wright's capillary tubes as described above, centrifuged several minutes to remove clumps, and the opalescent supernatant layer then constitutes the test emulsion.

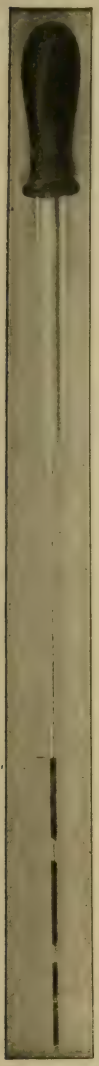


FIG. 482. FIG. 483.

FIG. 482.—Opsonizing pipette, showing the equal parts of blood serum, bacterial emulsion and leucocytes.

FIG. 483.—Mixture ready for incubation.

To procure the washed leucocytes, a few (10–15) drops of blood (from either the patient or another person) are drawn into a centrifuge tube containing sterile salt solution plus 1 per cent. of sodium citrate. After thoroughly mixing, the liquid is centrifuged, and, after the clear upper layer is pipetted off, again centrifuged, after which the upper surface of the precipitate will be found to consist largely of leucocytes which may be removed with a pipette and used for testing.

The *four test solutions* have now been obtained, viz.: (1) *normal serum*, (2) *suspected serum*, (3) *bacterial emulsion*, (4) *washed leucocytes*. Into a marked or otherwise graduated sterile nipple pipette with a long capillary extremity one draws equal parts of the washed leucocytes, suspect serum, and bacterial emulsion, separating the three portions by aspirated air bubbles; these equal portions are thoroughly mixed by alternating expulsion and respiration, and finally sealed in the capillary pipette by heat and placed in the incubator for fifteen minutes at a temperature of 37 to 40°C. A control tube is prepared in the same manner, using normal serum. Finally films are made in the same manner, stained in the usual way and examined with an oil immersion $\frac{1}{12}$ lens. The number of bacteria contained in 50 leucocytes is counted for both the test and control films, and the bacterial count per leucocyte in the patient divided by the average number contained in the leucocytes of the control film; *i.e.*, if the normal serum shows 5 and that of the patient 2.5 per leucocyte, the index is represented by $\frac{2.5}{5} = 0.5$. The normal index is but slightly variable, the minimum for the tubercle

bacilli being fixed at 0.8 and the maximum at 1.2. In most chronic infections, such as lupus, furunculosis and tuberculosis, and acute infections of the type of septicemia or Malta fever the index is uniformly and persistently low

FIG. 484.—Mixing test solutions.

FIG. 485.—Drop ready to spread.

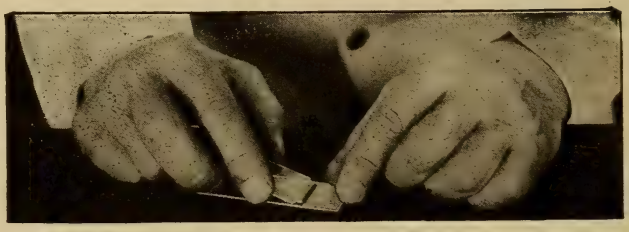


FIG. 486.—Making smear.

(0.4 or less). In the former one deals with a localized or "shut-off" process, in the latter with a blood-stream infection.

In cases where the infection is but imperfectly or intermittently shut off, as in certain cases of advancing pulmonary tuberculosis or tuberculous

joints, the index may show marked variations. For diagnostic purposes the T. R. is used in the usual manner in the case of tuberculosis and the resulting phases as previously described give to some extent, diagnostic, prognostic, and therapeutic indications.

Measuring the Dose of the Vaccines.—As it is necessary to use an exact or approximately correct dosage of any given germ, various procedures have been devised. One of the simplest consists in preparing an emulsion

FIG. 487.—Smear.

of the germs with salt solution and making a mixture consisting of 1 part of the germ solution, 1 part normal blood and 3 parts of salt solution. A count will determine the number of corpuscles and the proportion of germs to blood corpuscles and hence the number of germs per cubic millimeter. From 100,000,000 to five or ten times that quantity may be required to maintain the highest and most sustained positive phase, together with the minimum extent and duration of negative phase which is the therapeutic optimum.

THE INFECTIOUS DISEASES

TYPHOID FEVER (*Enteric Fever, Typhus Abdominalis*)

Endemic.

Definition.—*This acute general infection is endemic rather than epidemic, is especially frequent in the late summer and early autumn, rarely affects babes, is most prevalent between the ages of ten and twenty-five, rare in persons above sixty and especially affects the adult male.*

Seasonal incidence.

Characteristics.

It is caused by the bacillus typhosus and characterized by continued fever, enlargement of the spleen, a peculiar exanthem, diarrhea or constipation, tympanites, abdominal tenderness, marked nervous symptoms, the diazo-reaction in the urine, the presence of the causative organism in the blood during the early days of illness at least, and the agglutination reaction of Widal.

Eberth's bacillus.

Etiology.—*The bacillus typhosus* of Eberth (1880) is a small motile rod, flagellated, Gram-negative, staining readily, constantly present, easily recovered and grown in pure culture and capable of producing the disease in certain apes, thus fulfilling Koch's law.

Fulfills Koch's law.

Its development in the body results in the formation of agglutinins and precipitins responsible for the almost pathognomonic test of Widal.

Vitality of germ.

Outside the body it resists cold and moderate dry heat, but fortunately, *direct sunlight destroys it in a few hours* and it is readily killed by the ordinary antiseptics. *In water it lives but a few days if saprophytic organisms are present and in ice rarely longer than ten days or two weeks,* though in rare instances living germs have been recovered from surface ice after a period of nearly five months.

Water and ice.

Soil.

In superficial soil and in the feces they may live for months unless exposed to direct sunlight or other unfavorable conditions, and this applies to dust and filter sand as well as ordinary earth.

Modes of conveyance.

In milk, sweet or sour, in butter or in cheese, they may persist for months, and infection arises ordinarily through germs cast off in the feces or urine and indirectly conveyed to the water or food supply of the individual.

Though possible, direct contagion, under ordinary precautions, is rare, but the germs may be conveyed by food, clothing, soiled fingers, house flies, and even by dust.

Chronic carriers.

Certain individuals may become chronic carriers of virulent organisms and living centers of infection. One polluted spring may poison a whole city, and milkmen using water from infected wells for cleansing utensils or with less innocent purpose, frequently convey contagion to their patrons. Salads may be dangerous when their components are grown on soil fertilized by infected material, and even oysters bedded along lines of sewage flow may become active sources of infection.

Green salads.

Oyster beds.

Distribution of the Germs in the Body.—The infection is a general one, and almost any body fluid, secretion or excretion, may contain them. The urine is a peculiarly dangerous source of infection, containing germs in at least one-third of all cases, and oftentimes for long periods after apparent recovery. The organism is constantly present in the feces during the active

The urine a menace.

PLATE IV.

Typhoid Ulceration.
(From "Infectious Diseases," C. B. Ker.)

stages of the disease and *spleen, blood, and rose spot cultures* yield positive results in the majority of instances. It *may* be found in the bile and even in sweat, sputum, serous exudates or foci of suppuration.*

Modes of Entrance.—It is probable that all infections occur through the gastrointestinal tract and in the vast majority of cases it is the intestine and its associated structures that show the most marked lesions.

Incubation Period.—The incubation period ordinarily is from 10 to 14 days, but great variations are encountered.

In certain instances where the time of the ingestion of infected food has been definitely known, certain prodromal symptoms have appeared within seventy-two hours, whereas in other rarer instances the onset of the disease has been postponed for thirty or even forty days.

Varieties.—Following Osler, we may group cases as follows: (1) *Typhoid fever with marked enteric lesions.* (2) *Those of slight enteric lesions.* (3) *Cases with no discoverable intestinal lesions.* (4) *Mixed infection.* (5) *Pseudo-typhoid cases or those due to the so-called paratyphoid groups.*

The first group comprises the vast majority of all cases. Groups two and three represent extremely rare findings. Group four should be confined to those cases in which the secondary infection is caused by germs which favor the growth and intensify the action of the original germ. Such are the ordinary pyogenic cocci, the pneumococci, and the colon bacilli.

The fifth group comprises those cases denominated *paratyphoid.* In regard to these it may be said that they are due to the so-called paratyphoid bacillus of Achard and Bensaud (1896), *and the disease cannot be differentiated by its clinical manifestations*, which may be identical with those of true typhoid fever. The absence of the Widal reaction with the Eberth bacillus in a case clearly typhoidal in type is the best diagnostic evidence if paratyphoid cultures are unobtainable. If such can be had a specific diagnosis may be readily made by the methods of Widal or Pfeiffer.

Clinical typhoid fever may in fact be due to either the bacillus typhosus, bacillus paratyphosus A., or bacillus paratyphosus B., but the paratyphoid fevers usually are milder, of shorter duration and more rapid and acute onset. Hemorrhages are rare and perforation does not occur, but the anatomic lesions differ only in intensity from those of true typhoid fever.† Mild types.

In the Serbian army the failure to secure protection by the usual routine vaccinations led to the identification of a paratyphoid "C" which proved efficient.

There are certain features which justify one in making a tentative diagnosis of paratyphoid infection pending laboratory reports. These diagnostic factors may be grouped as follows:

* It is quite possible that in a considerable number of cases it may be recovered from the duodenal secretions very early. It has been so obtained by means of cultural methods long after convalescence.

† L. D. Bristol in reporting on paratyphoid "A," emphasizes the importance of applying agglutination tests with that strain as well as with "B" in doubtful cases. He reminds us also that demonstrable splenic tumefaction is often lacking in the paratyphoid "A" cases.

In paratyphoid the gastrointestinal symptoms usually are more severe than in true typhoid; the onset is more likely to be abrupt or rapid and an initial chill is reported in many cases; the temperature rise and termination are more rapid in paratyphoid, though cases of crisis have been reported. Herpetic eruptions of the facial or buccal type are relatively common in paratyphoid and very rare in true typhoid. (See also page 933.)

The adoption of protective vaccination has robbed typhoid fever of its terrors as an army disease. In the American forces during the Great War just closed a triple vaccine was used and as a result there were but 297 cases in the entire army on June 30, 1918.

PATHOLOGIC ANATOMY.—The germ may be recovered from the blood, mesenteric glands, spleen, bone marrow, and intestinal lymph structures and readily cultivated. The essential and almost constant lesions are those found in the agminate glands known as Peyer's patches, and the solitary follicles of Lieberkühn in the ileum and jejunum, the process being one of medullary infiltration. The simple follicles of Lieberkühn are found in both the large and small intestine; Peyer's patches, throughout the ileum and the lower jejunum. The follicles are at first grayish white and prominent and the inflammation increases during a week or ten days, then terminating by fatty degeneration and absorption, or more often in necrosis and ulcer formation involving the follicles alone, or exposing the submucosa and muscularis at the base of *oval* or round ulcers, which, unlike those of tuberculosis, tend to parallel the long axis of the bowel and lie opposite the mesenteric attachment. This process results in deep and extensive sloughing, the neighborhood of the ileo-cecal valve being usually the point of maximum change. The ulcer border may be undermined, the edge regular or irregular, and the stage of actual necrosis ordinarily represents the third and fourth weeks of the disease. (See Plate.)

Typhoid vs. Tuberculous ulcers

Hemorrhages, trivial or serious, may occur at any time, from the beginning of the ulcerative process to the completion of that of healing, but death from hemorrhage *after cicatrization* is a rare event.

Perforation with septic peritonitis occurs in about 5 per cent. of all cases and the danger of excessive tympanites during the later weeks of typhoid is evident. The perforation is ordinarily found in the ileum, rarely in the cecum or adjacent portion of the colon or vermiform appendix.

Usual sites.

The spleen is enlarged and extremely soft, rupture may rarely occur, either with or without traumatism, abscess is rare, infarction not unusual. *The liver* is hyperemic and shows signs of parenchymatous degeneration, rarely abscess, and the *gall bladder* not infrequently *contains the germs* and may be the seat of a cholecystitis. *Kidneys.* An acute nephritis or suppuration is rare, cloudy swelling and glandular degeneration more frequent. *Heart lesions.* Endocarditis and pericarditis are extremely rare. Granular and fatty degeneration of the myocardium is common, but is seldom a cause of death. *The arteries and veins.* Emboli and thrombi are not uncommon, venous thrombosis being the rule and having its most frequent seat in the femoral veins. Such a thrombosis constitutes one of the

complications of later typhoid. *Respiratory organs.* Splenization and hypostatic congestion are extremely common, infarction occurs in about 5 per cent. of all cases, but abscess, gangrene, and pleurisy are extremely rare.* *Nervous system.* A specific meningitis in typhoid is extremely rare, severe headaches and delirium common. *Mesenteric glands.* Their marked involvement is constant.

SYMPTOMS AND DIAGNOSIS.—*In the absence of the laboratory aids, no disease is at times more difficult to diagnose in its earliest stages, none save influenza or syphilis more interesting and protein in its forms, none more difficult to forecast and few more exacting in their demands upon the therapeutic resources and good sense of the clinician.*

Protean.

Many cases are remarkably clear in their development and run an uneventful course, but the practitioner must recognize the *atypical* and beware of hasty conclusions in those apparently straightforward. The work of the last decade has greatly simplified the diagnosis of typhoid for those who have at hand the aid of the clinical laboratory or the simple means to be described, and clinical and microscopic tests have added enormously to our positive knowledge of the character and frequency of variations in type.

Obscure cases.

Simplified diagnosis.

Major Symptoms.—(*a*) *An insidious onset.* (*b*) *A continuous and somewhat characteristic temperature.* (*c*) *Enlargement of the spleen.* (*d*) *Rose spots.* (*e*) *A lowered pulse-temperature ratio.* (*f*) *A low leucocyte count.* (*g*) *Ehrlich's diazo-reaction.* (*h*) *Agglutination test of Widal.* (*i*) *The positive blood culture during the first days of illness.* (*j*) *The recovery of typhoid bacilli from the stools, duodenal contents, or urine.*

Classic symptoms.

Minor Symptoms.—Of the more or less inconstant minor symptoms, one may mention *nose-bleed, gurgling, and tenderness in the right iliac fossa*, the so-called *typhoid tongue* with its V-shaped red tip and brilliant edges, the *pea-soup stools*, becoming a *brilliant ochre* in the later stages, *headache and delirium.*

Inconstant phenomena.

Complications.—The two most important are *hemorrhage from the bowel* and *perforation of the intestines.* The former is characterized by symptoms of concealed hemorrhage; the latter by profound shock.

Hemorrhage.

Two chief symptoms often signalize a large hemorrhage, even in the profound stuporous toxic patient, and should be carefully watched for and promptly noted by the nurse. They are (1) *a sudden drop in temperature;* (2) *coldness or even clamminess of the body surface.*

A sudden drop in blood pressure (below 90 mm. Hg. systolic) and temperature, abrupt severe abdominal pain,† and profound pallor are suggestive of perforation and the pain may be sharply localized with tenderness and defensive rigidity of the abdominal muscles and in some cases is gradual in onset. An insidious onset is relatively rare, yet cases come to autopsy which have previously yielded no definite signs of perforation or hemorrhage.‡

Symptoms of perforation.

Variants.

* Ulceration of the larynx has been reported, but has never been observed by the author.

† Pain may be absent or undetected in 10 or 12 per cent. of such cases. *Tenderness* is demonstrable in about three-fourths of the cases.

‡ The profoundly toxic state of many of these patients is sufficient in many instances to suppress pain and tenderness alike.

Shallow breathing induced primarily by the pain and tenderness and later by abdominal distention if that be present, is usually observed.

The remaining symptoms of later development are those of the ensuing peritonitis. One should seek for tactile crepitation over the xiphoid tip and costal arch areas, obliteration of splenic and hepatic dulness, movable dulness in the flanks, and audible friction on auscultation over the liver, spleen and intestinal coils. The systolic blood pressure is said to fall sharply in hemorrhage and rise abruptly in perforation, but according to the author's observations is likely to show a decided primary drop in both conditions.

Blood pressure.

In perforation the primary drop is followed by a somewhat deliberate secondary ascent of 20 to 60 mm. of Hg. (Norris), occupying from two to six hours, and succeeded by a return to low figures.

Norris finds that the average systolic blood pressure in this disease runs as follows: First week, 115 mm.; second week, 106 mm.; third week, 102 mm.; fourth week, 96 mm.; fifth week, 98 mm. (auscultatory readings).

In somewhat more than half the cases of perforation a leucocytosis occurs.

The sudden drop in the fever and acceleration of the pulse usually present may be misleadingly absent.

Successful treatment by surgical means demands prompt recognition and the early diagnosis is often extremely difficult.

In both hemorrhage and perforation the mental condition may suddenly become clear and, *occurring abruptly in such conditions*, this should always excite suspicion and cause a thorough search for other phenomena of these complications.

If hemorrhage be suspected the bowel should be cleared by enemata in order that early demonstration of the blood may be made.

The *sudden* appearance of abdominal distention is also a suspicious circumstance.

Oral toilet.

Proper care of the mouth has greatly reduced the frequency of *otitis media*, a condition readily overlooked when present in dull or stuporous patients, and bandaging of the lower extremities when the patient begins to sit up or walk usually prevents a *saphenous phlebitis*.

Bandaging the legs.

Insidious onset.

The Typical Case (*Seen rarely nowadays in its complete form*).*—There is first a period of *general malaise* (lassitude, aching head and limbs, loss of appetite, etc.), during which (or later) *nose-bleed* may occur with diarrhea or constipation, more generally the latter.

This condition progressively increases, slight *fever* is apparent and the patient takes to his bed. *The fever rises each night higher than the night preceding, receding each morning about one degree below the temperature of the preceding evening.* The *face becomes flushed*, the *tongue* is *heavily coated, save the tips and margin*, which may remain a bright angry red. An antecedent *diarrhea* may now increase *or* replace *constipation*, or the latter condition may persist. *Mental hebetude* is marked, and the *pulse*, heretofore somewhat slow and soft, *becomes weaker and tends toward dicrotism.* At the

Rising fever.

Gastro-intestinal signs.

* Before the days of refined diagnostic methods a case had to be reasonably typical to be recognized as typhoid fever.

end of a *week or ten days pale red points, like flea bites*, easily blanched by pressure, appear over the abdomen and lower chest usually in small numbers, and the *spleen may be readily palpable. Tympanites* may be a prominent and distressing symptom. By the end of the second week the fastigium is reached and all symptoms are intensified. Headache and hebetude are replaced by a *low muttering delirium*, and the *pulse* grows more *rapid and dicrotic.* The *heart sounds are weaker*, there is a tendency to *congestion of the lung bases*, the *tongue grows dry* and hard and sordes tend to accumulate upon the patient's lips and gums necessitating the most scrupulous cleanliness, the *attitude of* the patient indicates *profound weakness*, he sinks down in bed, lies constantly in one position, which must frequently be changed by the efforts of the nurses or ward attendants. If diarrhea is present, the *pea-soup stool* of the earlier period *is replaced by the light ochre stool* of the latter typhoid. *Tympanites* is likely to be *excessive* and increases the danger of perforation.

"Rose spots."

Mental state.

Decubitus.

Stools.

During two weeks the temperature is practically continuous, all symptoms are intensified, hemorrhage or perforation may occur and tendency to hypostatic pneumonia increases. In the *fourth week there is a recession of symptoms*, fever gradually subsides, the mind clears, the spleen shrinks under the ribs, tympanites lessens and the heart sounds and pulse are of better quality. Such a case offers no difficulty in diagnosis, but one must consider the common variations.

Period of recession.

Occasional Sudden Onset.—Our experience during the Spanish-American war has shown *how frequently there may be a sudden onset with chill and high fever.*

Often atypical.

Period of General Malaise.—*Nose-bleed* is frequent but neither constant nor limited to typhoid fever. *Gurgling and tenderness in the right iliac fossa.* This much overrated symptom may be found in any diarrheal condition, and any *extreme early tenderness is more suggestive of an appendicitis than of typhoid fever;* moreover, in many cases of typhoid these symptoms are altogether lacking.

Inconstant symptoms.

The Typhoid Tongue.—Such a tongue is strongly suggestive of typhoid, but is not always present, and may occur in other conditions associated with high temperature and gastrointestinal disturbances.

Fever.—"Step-ladder" temperature is somewhat rare *in its typical form*, and the use of cold baths has robbed it of much of its symmetry; moreover, as before stated, fever may occasionally rise rapidly with or without an initial chill, instead of gradually as in a typical case, and terminate either by crisis or by lysis in a few days or after several weeks.

The So-called Abortive Typhoid.—The *abortive typhoids are much more common than was formerly supposed*, and still more extraordinary are those extremely rare cases lacking temperature, which have been verified by the most modern and exacting clinical tests.

Afebrile typhoid.

Diarrhea.—This symptom is present only in a *minority* of the cases on a modern dietary, regular movements or moderate constipation being the rule.*

The exception.

Rose Spots.—These, when present, are valuable diagnostic aids and a

Present in two-thirds.

* Speaking from personal observation.

great source of comfort to the practitioner groping for a clew. They are present in about two-thirds of all cases, ordinarily few, scattered, and limited to the lower chest and abdomen, they may be profuse and widely distributed, but unfortunately, are not limited to typhoid.*

Often obscured.

Enlargement of the Spleen.—In association with the general symptoms of typhoid this sign is of great value but often an existing tympanites makes its determination impossible, and, moreover, it may be found in paratyphoid, acute miliary tuberculosis, malaria, septicemia and some of the tropical fevers. The spleen is often only just palpable.

Disturbed Pulse Temperature Ratio.—A low pulse rate associated with high fever is a valuable confirmatory sign of typhoid infection though by no means limited to this disease. *It is, furthermore, a valuable prognostic sign* indicating light infection or good resisting power. It should be remembered that pneumonia offers the same symptom, and in either disease one may be misled in elderly persons, in whom a slow pulse may be the result of an obstructive aortic lesion or arteriosclerosis.

Seldom necessary.

Recovery of the Bacillus from the Stools, Blood, or Urine.—The introduction of the Widal test and the simplification of the blood culture has made the first (difficult and unsatisfactory) method of investigation of little use to the practitioner. *Spleen puncture* will often yield a pure culture, but is neither necessary nor justifiable save in the rarest instances. *Blood from the rose spots affords an easy method of cultural diagnosis in many instances.*

Mental state.

Nervous Symptoms.—*These are essentially those of the "typhoid state,"* and *may* be seen in any disease associated with *overwhelming toxemia.* The low muttering delirium of the typical typhoid, and less often the mental hebetude (torpor) may be entirely absent, or, in rare instances, replaced by an acute maniacal delirium, which constitutes a most serious complication.†

Sudorous.

Misleading Variants.—*Of the many curious variations* in the general form of this disease may be mentioned (*a*) that associated with *profuse and exhausting sweats* which strongly suggest septicemia, pyemia, certain forms of malaria or ulcerative endocarditis; (*b*) those in *which the onset is so distinctly pulmonary* in its type as to lead to a diagnosis *of pneumonia* alone without regard to the underlying infection; (*c*) *the tonsillo-typhoid form, often accompanied by erythema and simulating scarlatina;* (*d*) the *renal form* in which symptoms of nephritis are prominent and misleading, and (*e*) the many variations due to complicating *abscess and necrosis.*

Septic.

Pulmonary.

Scarlatinal.

Renal.

Value of clinical picture.

The Important Factors in Accurate Diagnosis.—*The essential thing is a proper appreciation of the lesser value of individual symptoms, and the greater one of the grouped signs.*

Valuation of single symptoms.

Aside from the Widal test or the blood culture, no single clinical symptom suffices for a diagnosis of typhoid, but *many combined* reduce error to a mini-

* A case coming under the author's observation a short time ago presented the typical rose spots, but failed to react to the Widal test, and proved to be a case of miliary tuberculosis. Several such have been observed.

† Nowadays a considerable number of true typhoids pass through the whole course of their illness without any marked mental disturbance.

mum, and of those mentioned nearly all are *important in combination*, but of slight significance if isolated.

Cardinal points.

Four symptoms have been reserved for separate discussion because of their great individual value. These are, first, *a low leucocyte count;* second, *Ehrlich's diazo-reaction;* third, *the agglutination test of Widal;* fourth, *the blood culture.*

It has been amply proven that nine-tenths of all cases of typhoid, unless complicated by a secondary infection, yield a subnormal or low normal leucocyte count, which tends to progressively decrease as the disease increases in severity.

The exceptions represent, chiefly, blood concentration from vomiting, diarrhea, or cold baths. A count of 15,000 may be regarded as the maximum thus produced, and, on the other hand, even in complications counts as low as 1000 have been reported. *Absence* of a high count in the relation to a suspected complication does not furnish absolute proof of its non-existence, but the *presence* of a high count with no profuse diarrhea or vomiting is good proof of one. A moderate secondary anemia is practically invariable in all cases.

Recent investigations by Himmelheber show that the lowest count coincides with the maximum of toxemia and that the leucocyte reduction first affects the neutrophiles, and later, the lymphocytes. The latter then increase, and by the end of the second week a relative lymphocytosis may be present. The "coagulation time" of the blood is increased during the height of the fever (favoring hemorrhage), and shortened during convalescence (inviting thrombosis).

Value in negation.

Ehrlich's Diazo-reaction.—This much maligned, misunderstood, and faultily applied test is one of genuine value in the exclusion of typhoid, but cannot be used save with a thorough understanding of the conditions under which it appears, its limitations and the true typhoid reaction color. The test recommended is that of Ehrlich as modified by C. E. Simon and the author (see under "Urinalysis").

Limitations of the Diazo-reaction.—It cannot be too emphatically stated that *the presence of a true typhoid diazo-reaction does not prove the disease to be typhoid;* with equal emphasis it may be said that *absence of reaction in a supposed case, provided the test be made at a proper stage of the disease, amounts to almost positive proof that typhoid is not present.* This statement, iterated and reiterated by the author ten years ago, has, in the last few years, been abundantly confirmed by other observers using it in connection with the agglutination test. *It may appear as early as the fourth day of a typhoid and is almost invariably present by the end of the first week or ten days. It becomes intensified as the disease progresses, but rapidly fades as soon as the acme of infection is passed.* This latter fact is an extremely important one, and a failure to recognize it has led to many errors. The reaction is present in many of the *acute exanthematous* diseases, but if made with the author's higher dilution, as described elsewhere, will be found absent in *malaria, most cases of appendicitis, pneumonia,* and the *earlier stages of acute miliary tuber-*

Period of persistence.

Diseases excluded.

culosis. Indeed, its late appearance in the last disease is of considerable diagnostic value in that connection.*

Practically pathognomonic.

The Agglutination Test of Widal.—*Aside from the blood culture this is by far the most important and positive test of typhoid fever.* It was first announced to the medical profession by Widal in 1896.† This test really depended upon an earlier discovery by Pfeiffer (May, 1893), who found that cholera vibrios mixed with the serum of an immune animal and injected into the peritoneal cavity of a guinea pig lost their motility and changed their form, and later on, finding that the typhoid germ reacted in a like manner with the blood serum of typhoid patients, he applied the test to the identification of the germ. Widal *reversed* the process and applied it to the diagnosis of the disease. *The test is as nearly pathognomonic as any in the range of medicine, and in its simplest form may now be applied by any practising physician.*‡

Rapid rough method.

The method originally employed by the author, though rough, was surprisingly definite in its results and required merely an agar-agar (*i.e.*, solid) culture of typhoid bacilli, a medicine dropper, a platinum loop, and distilled water. The loop being made of fine wire was so constructed as to leave a central opening of not more than 1/16 inch. The tests were made either with the fresh blood or the dry film, the latter being treated with a drop of distilled water before testing. Instead of the "hanging-drop" method, the ordinary flat preparation was used with equally good results. It was only necessary to place a drop of distilled water upon each of two clean microscope slides, take a small particle of the culture with a sterilized platinum loop, stir it into the drop of water, sterilize the loop, and from a drop of blood obtained from the suspect allow the tiny loop to fill and stir this film into one of the drops containing the bacilli. A cover-glass was then dropped upon it, it was rimmed with vaseline, placed

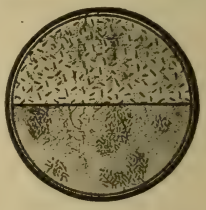


FIG. 488.—Typhoid agglutination test. (Widal.) Upper segment shows the freely moving germs. The lower the typical "clumping."

* The present neglect of the reaction is due in part to the availability of the crucial bacteriologic and serologic tests and in part to the fundamental difficulty inherent in any test depending upon color interpretation. It is extremely simple if instructions are followed absolutely.

† Bulletin Medicale, 1896, p. 618. The papers of Wyatt Johnston (New York Medical Journal, Oct. 31, 1896) and of the author (Medical Record, Nov. 14, 1896, and Dec. 6, 1896), being the first reports published in this country.

‡ At the present time one may obtain the so-called typhoid agglutometer (Parke, Davis & Co.), by means of which the test may be applied without the use of the microscope. The agglutometer comprises three tubes containing: (*a*) A sterile permanent suspension of typhoid bacilli. (*b*) A control tube of the same nature for the comparison of reactions. (*c*) A dilution tube containing fluid for properly diluting the serum previous to testing. (*d*) Tube for the collection of the blood to be investigated. (*e*) A pipette for the withdrawal of the serum. (*f*) A puncture needle. The test is made by adding 2 drops of the serum to the fluid in the diluting tube and this diluted serum is distributed in specific quantities in three tubes of suspended material. If the reaction is positive floccules appear in one or more of the tubes in one or two hours, the reaction being readily determined by a comparison of the treated tubes with the control.

under the microscope and a reaction was indicated by loss of motility, and grouping of the germs, *half an hour* being the time set for a positive reaction, reference being made to the control slide for error due to pseudo-reactions or preformed clumps.

The methods now in general use quite properly provide for exact dilution and the use of bouillon cultures,* and require laboratory facilities.

A macroscopic test.

During this early period in tests thus made the author found that if a relatively large amount of the solid culture material was used a positive reaction could be determined in the same time and by the same method, without a microscope, through the gradual appearance of a dust-like film or haziness, due to agglutination, which became visible when the surface of the cover-slip was viewed tangentially against the light. This was easily proven by the microscope to represent the agglutination reaction.

It may be added that oil-immersion lenses are not absolutely necessary to the performance of the test, those of moderately high power being quite sufficient.

Slides and cover-glasses should be washed in pure water before using, to remove any powerful antiseptics. Blood should never be taken upon paper or cardboard, but rather upon glass, porcelain or tinfoil.†

Time of Appearance.—If the fresh whole blood be used, the reaction may be found as early as the fourth day of the disease. It is persistent throughout the illness and in some cases for a year or more, but its exact duration has not been determined. Cases of delayed or even absent reaction have been reported, but the author has found few which have been delayed beyond the first ten days of the illness. *A single negative test should never* be considered sufficient.

As early as the fourth day.

Period of persistence unknown.

Value of the Test.—Even when working with dried blood, competent observers report positive results in at least 95 per cent. of the cases tested. It corrects innumerable errors of observation and must be given not only first place among *readily available measures* in the diagnosis of typhoid, but a value more than equal to that of all other *symptoms* combined. It should by now be universally invoked by city and country practioners alike, and its teachings accepted in a proper spirit of humility.

Its use imperative.

The Blood Culture.—*Unlike all other tests, this is of greatest value and constancy during the first week of the disease and of diminishing importance as the local changes increase.*‡

An early test.

It is almost invariably positive during the first week and present in from 80 to 90 per cent. of cases early in the second week.

Constancy.

Test.—The technic itself is extremely simple. The blood may be taken from the lobe of the ear or the finger-tip, exactly as in the case of the ordinary

* Young bouillon cultures are not dependable.

† To the city practitioner who may send his patient's blood to the laboratory of his city or state, and obtain a prompt report, these statements may seem of slight importance, but it is sometimes most comforting to the country practitioner to anticipate by several days the report of a distant laboratory. The same statement applies to the diazo-reaction.

‡ It is quite uncertain after the tenth day of established clinical typhoid fever.

blood count, from 30 to 40 drops being milked into a test-tube containing bile medium (inspissated ox bile, 30.0; peptone, 2.5; water, 250 c.c.). A better method consists in the use of a sterilized hypodermic syringe and the direct withdrawal of blood from a superficial vein.

A report can be made in from twelve to twenty-four hours, motile Gram-negative bacilli being often demonstrated in the original solution after twelve hours of incubation. If not then present, a solid agar culture receives a few drops of the original bile medium and suspected blood and is incubated for twelve hours. For absolute accuracy, tests should be applied for colon and paratyphoid.

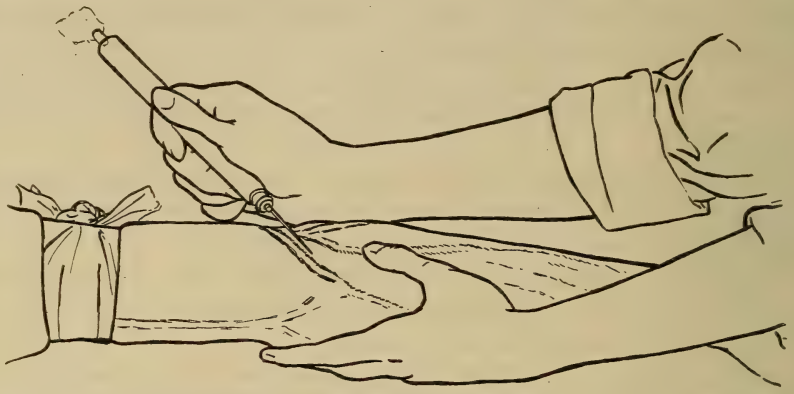


FIG. 489.—The usual method of obtaining blood for blood cultures. (*After Todd.*)

In view of the value of blood cultures in the diagnosis of obscure infections a brief reference to the simple method of securing blood and culture medium made available to all practitioners by the Keidel vacuum tube may be desirable.

Keidel Vacuum Tube.—Directions for Use.—Cleanse the patient's arm at the bend of the elbow with soap and water, and with alcohol and ether, in the usual manner. Place a constricting band around the arm above the elbow and after removing the connecting cap and stylet from the needle, introduce the needle into any prominent vein that may present itself. When the needle has been satisfactorily introduced into the vein, break off the sealed point of the vacuum tube (of 50 c.c. capacity), containing the culture medium within the rubber tubing, holding this tubing between the fingers and thumbs of both hands. As soon as the blood begins to flow, remove the constricting band and allow 5 c.c. of blood to enter the tube, then quickly withdraw the needle, replace the protecting tube and thus reassemble the outfit, which may then be carried to the laboratory. If it is necessary to mail the tube, remove the rubber tubing under aseptic precautions, and carefully seal the broken end of the tube, that contains the culture medium and specimen, in a Bunsen or alcohol flame. (See Fig. 490.)

Decisive test.

DIFFERENTIAL DIAGNOSIS OF TYPHOID FEVER.—Widal's reaction or the blood culture when practicable will almost invariably eliminate

any doubt that arises, but in its absence one must depend upon the grouping of symptoms.

Acute Miliary Tuberculosis.—This differs from typhoid in its irregular temperature, the fever being often of the inverse type; its chills and sweats, more rapid emaciation, the fact that rose spots are usually absent, the late appearance of the diazo-reaction and splenic tumor, the failure of the Widal test and sometimes in the discovery of definite pulmonary symptoms.

The family history, past health record, or known exposure to infection is often suggestive, *but it must be remembered that typhoid and acute miliary tuberculosis may co-exist and that in both diseases there is a tendency to abnormally low leucocyte counts and low blood pressure.*

A peculiar bilateral, universal, thoracic hyperresonance has been present in nearly every case of general miliary tuberculosis observed by the author.

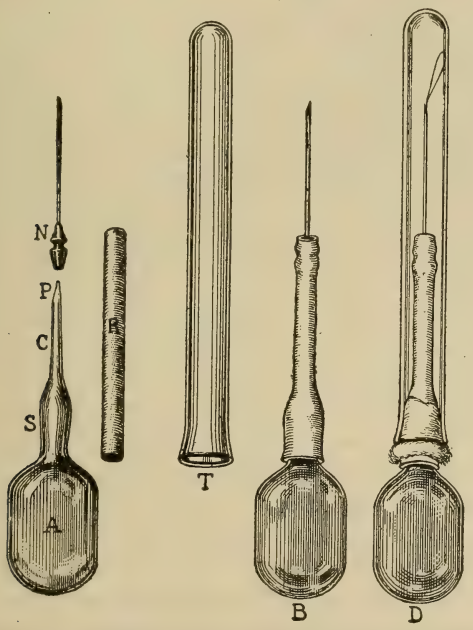


FIG. 490.—Keidel's tubes. Simple sterilized tubes, carrying a needle and containing culture medium are now readily procurable at a small cost.

Septicemia.—As this disease is usually characterized by a distinctly "*typhoid state*," the *focus of suppuration must be sought*, and the irregular temperature, with possibly chills and sweats, and a marked leucocytosis may point to a true diagnosis. It gives the diazo-reaction exactly as does typhoid, but does not yield a Widal or the typhoid bacillus in the blood.

Blood cultures may be decisive and reveal the dominant microörganism.

Ulcerative Endocarditis.—These present much the same features with the added help of *localized symptoms*, yet under superficial methods, most cases of malignant endocarditis of the acute type are mistaken primarily for typhoid, and many of those of *endocarditis lenta* for typhoid, tuberculosis or malaria.

Sepsis and local signs.

The symptoms of chronic recurrent ulcerative endocarditis are, however, more distinctly septic, cardiac lesions, almost invariably, are pronounced, and precordial oppression and dyspnea are rarely absent. Cutaneous petechiæ are especially common on the extremities, and in the chronic recurrent type due to the streptococcus viridans, the *painful*, *tender*, transient, cutaneous nodules are almost pathognomonic. Leucocytosis is usually marked.

Blood cultures if positive will lack the typhoid bacillus and show the causative organism which is usually a streptococcus.

Salpingitis.—*Salpingitis* is of course to be thought of in all obscure infections.

Cholecystitis.—As a complication of typhoid this condition is not uncommon and though usually occurring during the second week it may be encountered in the early days of illness. Perforation of the gallbladder has occurred and should always be kept in mind as a rare possibility.

Appendicitis.—This constitutes a not infrequent source of error, operations for appendicitis being made without justification and cases of appendicitis being treated as typhoid. Ordinarily, the localized symptoms and sudden onset with perhaps a previous history of similar attacks serve to make the diagnosis clear. *Early* localized tenderness in the appendiceal region and a leucocytosis are rare in typhoid and in appendicitis the early colicky pain is almost invariable and blood cultures and the Widal test are negative.

Error common.

Meningitis.—This is sometimes to be distinguished from the cerebral form of typhoid only by the absence of the diazo-reaction, the failure of the blood cultures and Widal test, but ordinarily no confusion of the two diseases is possible. Lumbar puncture and the examination of the spinal fluid is now a ready means of differentiation.

A bastard syndrome.

Simple Continued Fever.—This can never be positively differentiated from the early stages of typhoid fever unless the two crucial tests are applied. As a matter of fact, we may reasonably assume that no such thing as simple continued fever exists. It merely represents an unsolved problem in infection and, nowadays, may usually be placed under its proper specific designation.

Pneumonia.—This is a frequent complication of typhoid, but as an independent disease may be recognized by its sudden onset, marked localizing pulmonary symptoms, high leucocyte count, absence of true diazo-reaction in high dilution (1–100), negative blood cultures and Widal test.

Psittacosis.—An epidemic chronic enteritis of parrots which when transmitted to man presents a clinical picture of typhoid complicated by atypical pneumonia. Inasmuch as the germ *may* be agglutinated by typhoid serum, though in lower dilutions, and strongly resembles it in cultural characteristics, the observer's opinion is likely to be based upon the past handling and feeding of infected parrots and the occurrence of house epidemics.*

Malaria.—The remittent form can at times be differentiated only by

* As a matter of fact the bacteriologic findings are extremely varied and reports are contradictory. It is probable that in most instances the disease in man would lack the agglutination test.

finding the plasmodium in the blood, the absence of a diazo-reaction and a negative blood culture and Widal test. In any malarial country a mistake in diagnosis is extremely frequent if clinical symptoms alone are considered.

Influenza.—This protean disease may sometimes closely simulate typhoid but lacks the rose spots, enlarged spleen, diazo-reaction, positive blood culture and Widal.

Trichinosis.—The early stage of trichinosis and typhoid are similar, but edema of the face, muscle tenderness, and eosinophilia in the former and lack of the cardinal tests of typhoid, soon permit accurate differentiation.

Tubercular Peritonitis.—This may closely simulate typhoid fever but lacks the cardinal features of *developed typhoid.*

Brill's Disease.—This, a mild form of typhus fever, lacks the chief symptoms of typhoid and can hardly cause confusion.

Paratyphoid.—As before stated, this disease exactly simulates typhoid and can only be differentiated positively by cultural methods and the agglutination tests. It unquestionably furnishes an explanation of the cases in which a failure of the Widal test has been reported in the face of a group of symptoms convincingly typhoidal in type and such failures can scarcely be proven without cultural or paratyphoid-agglutination tests.

It is only during the past few years that the frequency of paratyphoid infections has been recognized.

As stated previously, the disease ordinarily is milder and its mortality much less than is the case with typhoid fever of the classical type. It is true, nevertheless, that certain epidemics have been reported in which the symptoms were extremely severe and the mortality great. (See also page 922.)

Conclusion.—It is evident that, both in direct and differential diagnosis, in certain cases the agglutination test, the blood culture, and the negative diazo may be the only determining factors.

PROGNOSIS.—The mortality of typhoid varies greatly according to the age of the patient, his condition at the time of its onset and the character of the epidemic, if such exists. It varies in private practice from 5 to 15 per cent., in public hospital practice it may reach 20 per cent. Much depends upon the character of the hospital population and methods of treatment employed. The mortality of different epidemics varies widely in the presence of virulent infection, a picked body of men such as would be represented by a newly mustered volunteer regiment furnishing a low death rate. On the other hand, the same regiment after a long campaign is likely to furnish an extreme mortality.* The female furnishes a higher mortality than the male, and the so-called walking typhoid, *i.e.*, the ambulatory form, presents, as might be expected, an excessive death rate.

Private vs. public.

Varying mortality.

Sex.

* This is well illustrated by a comparison between the light death rates of such of our own volunteer regiments as remained in camp, but out of service during the Spanish-American war, and the British regiments in South Africa among whom the death rate was nearly trebled in cases occurring during or after the active campaign.

The frightful incidence of typhoid in armies has been overcome to a gratifying extent by the present method of prophylactic immunization.

Ominous events.

The temperature curve seems not to figure greatly, but pronounced nervous symptoms, high pulse rate, and excessive meteorism are of bad portent, and such complications as hemorrhage and perforation threaten life, the latter being almost invariably fatal even if prompt surgical measures are instituted.

Hemorrhage occurs in about 5 per cent., and perforation in about 3 per cent. of the cases. *There is a marked tendency to relapse, especially if improper feeding be permitted during the period of convalescence, and it is seldom wise to depart from the modernized routine fever diet until the temperature has been normal for a week,* though it sometimes requires the finest judgment to decide whether fever is due to an obscure complication or the persistence of the infection and intestinal changes.

Relapse.

The best guide is the condition of the abdomen; freedom from tenderness and rigidity (general or localized) being the safest indication for an increased dietary.

COLON BACILLUS INFECTIONS

However much of a factor the colon bacillus may be in infections of the gallbladder, urinary bladder, and the peritoneum, when it suddenly gains access to that tissue under conditions reducing greatly its resistance, it seems wholly probable that its attacks are usually limited to more or less devitalized tissues.

It is certain that the more critical analysis, associated with the modernizing of methods, is greatly reducing the number of ailments formerly accredited to this normal and usually benevolent inhabitant of the intestinal tract.

One may admit the following activities:

(*a*) *Terminal infection, i.e.*, producing toxemic symptoms in moribund or near-moribund patients.

(*b*) *Peritonitis* in some instances of perforation, strangulation of a hernial sac content and the like.

(*c*) *Cholecystitis.*

(*d*) *Cystitis* and *pyelitis.*

(*e*) *Occasionally an enteritis* in conditions of impaired vitality.

(*f*) *Pseudo-typhoid.*

It is probable that its rôle as an etiologic factor will undergo further shrinkage with the passage of time.

INFLUENZA

(*"La Grippe," "La Cocotte," "La Follette," "The Pleasant Acquaintance," "The Jolly Rant," "The Knock-me-down Fever," "Catarrhal Fever," "Epidemic Catarrhal Fever"*)

Historic Note.—Influenza has been recognized as a clinical entity for over 2000 years, having been reported first by Hippocrates in 426 B.C., who recognized its epidemic type and described the chief outstanding symptoms of the disease. Its misnomers.

Influenza appeared in the Greek army at the siege of Syracuse and destroyed thousands of soldiers. In 1728 it drove the Imperial Court of Russia from the city of Moscow. In 1782 it so crippled the fleet of Admiral Kempenfeldt as to drive it back to port. Ancient epidemics.

The epidemic of 1918–19, occurring during that critical period when the cantonments of the United States Army contained the very flower of the youth of our country, brought home to our people as never before a realization of its fearful destructiveness. Epidemic of 1918.

The author's duties in War service took him at this time from hospital to hospital and from camp to camp, and the Fall of 1918 held for him more of tragedy than any other period in his life. The extraordinary morbidity and the terrific mortality, affecting as it did chiefly the choicest and best of American youth, was appalling and unutterably depressing.

Epidemic Variants.—The epidemic and pandemic nature of the disease has long been demonstrated but this epidemic showed also the peculiar tendency of the disease to vary in virulence in different countries and even in the various centers of infection in the same country. This variability was manifested both with respect to the incidence and the mortality. Curiously enough the Expeditionary Forces seemed to have suffered far less than did those undergoing training in the United States. Geographic distribution.

It is now known that there is a fairly definite cycle of recurrence with respect to this disease which occurs in periods of from 28 to 30 years, and it is also certain that the type remains essentially the same, though wide variations exist as to its severity. The epidemic of 1918 was much more virulent in type than that of the early 90's. It is believed that over 500,000 deaths resulted from the former, whereas the probable mortality of the latter epidemic was comparatively small, though considerable. Periodicity of epidemics.

General Comment.—Like cholera, influenza seems to follow lines of commerce in its spread and one can hardly escape the belief that, while essentially it is epidemic, it nevertheless is always present in some form throughout a large portion of the world. Furthermore, there is a definite tendency to the recurrence of the individual epidemics themselves after a period varying from six months to a year, and during such recurrences the type of the disease may be markedly modified. This certainly was true in the recurrences of the epidemic of the early 90's according to the recollection of the Recurrences.

author. In the case of that of 1918 this country was fortunate in escaping any severe repetition of the original infection.

Older theories as to cause.

Etiology.—The cause of influenza remained a mystery until very recent times. Hippocrates ascribed it to "Divine wrath," and Sydenham to some "occult and inexplicable changes wrought in the bowels of the earth." Weber spoke of a "negative state of electricity," whatever that may have meant, and recommended the wearing of socks lined with non-conductors. The prevalence of fogs of an extraordinary character, the appearance of comets, earthquakes, and various other disturbances, were held by many to be concerned in its causation, yet more than two and a half centuries ago there were advocates of the "*contagium vivum.*"

Pfeiffer's bacillus.

The Influenza Bacillus.—In 1892 Pfeiffer described small, non-motile bacilli, recoverable from the secretions of those suffering from influenza and staining readily with concentrated alkalin methylene blue and carbol-fuchsin. These took the dye so deeply at the ends as to resemble diplococci. They proved to be Gram-negative, were cultivable with difficulty on blood agar, and in smears tended to form groups. He did not succeed, however, in absolutely establishing this organism as the cause of the disease nor can it be said even at this time that we know positively that, unassociated with other organisms, *Bacillus influenzae* is capable of producing true influenza.

Nevertheless the evidence in its favor has been strengthened greatly by observations made by competent observers during the epidemic of 1918. In general there was a tremendous amount of variability in the reports submitted even from the Army hospitals, some failing to find Pfeiffer's bacillus at all and others reporting it only in a limited number of cases. On the other hand it is significant that observers of undoubted trustworthiness were able to recover the organism in nearly all cases coming under their observation, some reporting as high as 93 per cent. positive findings. (Stillman and Pritchett.)

Specificity unproven.

So far, the specificity of this organism has not been proven for man, but Blake and Cecil have shown recently that when cultures, whose pathogenicity has been heightened by animal transmission, are introduced into the trachea and bronchi of monkeys there is produced in these animals a disease closely resembling or identical with true influenza, the parallelism extending even to the production in some instances of the peculiar hemorrhagic pneumonitis which constituted the chief cause of mortality in the recent epidemic.

Many strains.

One of the interesting developments of the recent studies of *Bacillus influenzae* is the discovery that many strains are obtainable varying considerably in their pathogenicity. This has been held as being against the specificity of the organism but such an assumption would seem to be far-fetched. On the other hand such variation reasonably may be associated with the peculiar variability in the symptomatology of influenza in different epidemics and especially in the recurrences following them. It is worthy of note that during epidemics Pfeiffer's bacillus is present in a large number of normal individuals.

Contagiosity.—The disease is highly contagious and, with respect to virulence and transmissibility of its pathogenic organism, it may be said that it is probable that the height of virulence and the maximum of contagiosity is attained at the very beginning of an attack. If this be true, as seeems probable, it goes far to explain both the rapid spread of the disease early in epidemics and the extreme difficulties attending the prevention of its spread. Highly contagious.

After the experience of 1918 it would seem that no measures however radical would be unjustified in the presence of an epidemic. The wearing of face masks, the prevention of all public meetings so far as humanly possible, and the isolation of the individual patient, all countribute to the limitation of the spread of the disease. Prevention of spread.

Under such conditions as have prevailed heretofore, in all probability practically every person who did not possess individual immunity acquired influenza.

Immunity.—With respect to the production of immunity in those having the disease, little can be said positively. It is probable that for a limited period such immunity may be acquired. With respect to the production of immunity by artificial means through the use of sera, dead cultures, or what not, it can only be said that the value of these various procedures remains unproven. Immunity.

Age and Sex.—This disease affects the male and female about equally and attacks with especial severity and frequency young adults. Those individuals under 16 and over 40 years of age are less frequently subject to attacks, or, at least to its severer manifestations. Pregnant women, if far advanced, showed a fearful mortality in the epidemic of 1918. Pregnancy.

Incubation.—Hitherto the period of incubation has been unknown, but observations made during the recent epidemic show that in a large majority of the cases it is of forty-eight hours' duration. In a larger number of instances it may occupy three days; in a still smaller number of instances one day; and possibly the period may be extended to a week or ten days in rare instances.

Types of the Disease.—In general, one may speak of "true epidemic influenza" and of "endemic-epidemic influenza." The former is by far the more serious and fatal type but, aside from the severity of its symptoms and complications, is practically identical with the second form. From a purely clinical standpoint we speak of "*simple influenza*," in which only the basic characteristics of the disease are present, the duration being short, and of "*influenza with complications*." Chief types.

In 1918 especially, the dominant complication was a peculiar hemorrhagic type of pneumonia, these cases furnishing almost the sum total of the excessive mortality experienced.

Moreover, while possessing certain well-defined peculiarities, influenza is one of the most protean of acute diseases if we consider the differences to be noted in its various epidemics and especially the variance associated with its recurrences. Protean.

It would appear that the epidemics of different years assume certain predominating features. Some are especially catarrhal, coryza and bronchitis being the prominent features, and pneumonia a frequent complication. In others, nervous and even cerebral manifestations play a considerable part; and in yet others gastrointestinal symptoms are unduly prominent. *Furthermore, while distinctly a febrile disease, it often presents an afebrile form or one in which the fever is so slight as to lead to an under-estimation of the importance of the attack.*

Such variants are especially common in the endemic-epidemic form and to a less degree in the definite recurrences.

Its diagnosis in the absence of a definite epidemic and even in the presence of one is rendered obscure oftentimes by its simulation of a multitude of diseases as a result either of certain selective points of attack or the peculiarity of its complications.

Diseases simulated.

Among these confusing conditions may be mentioned cerebral and cerebrospinal meningitis, cerebritis with hemiplegia or paraplegia, intractable neuralgia, pleurisy, pneumonia of the ordinary type, jaundice, excessive vomiting, diarrhea, dysentery, nephritis, suppurative or non-suppurative otitis media, endo- or pericarditis, thrombosis, embolism, psychasthenia, melancholia, or mania.

In the epidemic of 1918 there was little opportunity for error along any of these lines and it is probable that in the past some of the confusing complications just mentioned were due to conditions now much more readily and certainly recognized, such, for example, as sinusitis, the most frequent cause of so-called intractable neuralgias and violent, long-persisting headache.

Cardinal points.

In view of these complicating conditions there is but one safe ground for the clinician to occupy, and that is to recognize and act upon the fact that in the absence of an epidemic, influenza may sometimes be recognizable only through the recovery of the bacillus and the abrupt appearance of a degree of exhaustion entirely out of accord with the other symptoms of acute infection presented.

Mistakes in diagnosis must inevitably be common, excusable, and fortunately in most instances without serious results, inasmuch as the treatment of most complications is essentially that of the disease which they simulate.

Unfortunately this statement did not hold true in the terrible epidemic of 1918 for it was necessary to treat even *suspects* with extraordinary care in order that the excessively common and fatal complication, hemorrhagic pneumonia, might be avoided.

SYMPTOMATOLOGY.—Simple Influenza.—*Onset.*—One of the most characteristic symptoms of influenza in all of its forms is the astonishing abruptness of its onset. In many instances a person in perfect health becomes in a few moments a sick patient; in others there may be during a period of several hours certain prodromal symptoms, usually a sense of weakness in the legs and back, often in the joints, and in a certain proportion of cases associated with headache more or less severe.

Common type.

Pain.—The pains described may be very severe; the pain in the back, for example, in cases of the severer type may equal that of the prodromal pain of smallpox. The headache is usually frontal, but may be occipital, general, or even hemiparietal.

The pains are frequently associated with a considerable degree of hyperalgesia. Hyperalgesia.

Fever.—Fever comes on promptly and attains its maximum early, enduring in simple cases from three to five or six days and subsiding ordinarily by lysis. In preceding epidemics the ending of the fever seemed usually to terminate the attack save for the exhaustion and weakness remaining. In the epidemic of 1918, however, this was not true and it was found to be unsafe to assume that the ending of the febrile period might permit the patient to resume his physical activity. In a host of instances the failure to recognize this fact led apparently to the oncoming of the most dreaded and fatal complication, namely, pneumonia. Indeed, it seemed to be proven beyond contradiction that the only safeguard against this extremely fatal pulmonary complication lay in a prompt recognition of the initial symptoms of the disease, the immediate putting of the patient in bed, and insistence upon his remaining there for several days after all active symptoms seemed to have disappeared. Dangerous over-sight.

Coryza and Bronchitis.—The degree to which coryza, tonsillitis and bronchitis dominate the clinical picture in influenza varies widely in different epidemics. In the epidemic of the early 90's coryza was a very prominent feature, and to a less degree this statement is true of the epidemic of 1918. Tonsillitis in some epidemics has been a prominent feature. In the last one it was only occasional. On the other hand, bronchitis is one of the relatively fixed features of influenzal attacks and in the last epidemic was very prominent. An harrassing cough with substernal pain or distress developed early, and it is significant when taken into consideration with the frequency of pneumonic complications and the type of pneumonia present, that the degree of substernal pain was great in so large a proportion of the cases. In no other respect than this last did the bronchitis differ from the simple influenzal form. The sputum was not characteristic, though claimed to be so by some observers, but followed the usual changes, being mucoid, mucopurulent, and sometimes streaked with blood. Coryza. Tonsillitis. Bronchitis. Cough and pain. Sputum.

Prostration.—The degree of prostration present in simple influenzae is always marked and oftentimes excessive. In any event it is wholly out of proper relation to the fever and other symptoms present in the given case. Oftentimes it is peculiarly persistent and it has seemed to the author that in the older epidemics this was the case in a great many of the relatively or almost wholly afebrile cases. Prostratio marked or extreme.

Circulatory System.—Nearly all writers unite in stating that cardiac complications are rare in influenza though some describe a cardiac type of this disease. Such assertions are perhaps dangerously misleading. It may be granted that endocarditis and pericarditis are rare, but on the other hand it has seemed to the author that myocardial toxemia is marked Heart. Myocardial toxemia.

even in cases of moderate severity and profound in those of the severer type, especially when pneumonic complications supervene.

Undoubtedly the greater number of cases of definite toxic myocarditis are overlooked and this statement is more or less justified by the author's experience following his return from Army service and the resumption of a practice devoted almost exclusively to cardiovascular disease. An astonishingly large proportion of the cases reporting for diagnosis and treatment at that period dated their symptoms of decompensation from an influenzal attack.

Of cardinal importance.

Cyanosis.—One of the characteristic features of the severer types of influenza in the 1918–19 epidemic was the cyanosis present and by some observers this seems to have been regarded as one of the symptoms of uncomplicated cases. This assumption no doubt is erroneous, and any marked degree of cyanosis must be considered as indicating the presence of a complicating pneumonia or profound cardiac weakness.

The Pulse.—The pulse rate in influenzal cases is not accelerated usually to a degree conforming to the physiological rule of pulse and temperature ratio and even though a complicating pneumonia ensues the same statement holds true. Indeed *pulse acceleration is one of the important factors suggesting the onset of a pneumonia or other complication*, and if this rate exceeds 120, other causes being eliminated, and pneumonia is present it adds greatly to the gravity of the prognosis.

Important factor.

Leucocyte count.

The Blood.—The low leucocyte count in influenza cases is a somewhat striking symptom and may reach low figures, 2000 or less per cubic millimeter. Even when a pneumonic complication occurs the rise in the count is slow and the total seldom exceeds 15,000 or 20,000 leucocytes per cubic millimeter of blood. The average leucocyte count in uncomplicated cases is about 6000 per cubic millimeter.

Albuminuria.

Genito-urinary Tract.—Practically all severe cases of influenza will show febrile albuminuria with hyaline and finely granular casts in the urine. On the other hand, nephritis as a complication is extremely rare.

"Abdominal influenza."

Gastrointestinal Symptoms.—These usually are not pronounced in mild cases, though in certain epidemics or recurrences after epidemics they may be so pronounced as to justify the use of the term "abdominal influenza."

In the epidemic of 1918 they were often present in the pneumonia cases, taking the form most frequently of absolute anorexia, nausea and tympanites. In many instances vomiting was severe and, in some, an exhausting diarrhea occurred. In many of the cases with severe tympanites, an extremely acute abdominal pain occurred which misleadingly suggests a surgical condition.

Erythema.

Cutaneous Manifestations.—Many observers writing of the epidemic of 1918 report the frequent occurrence of an erythema affecting chiefly the face, the chest, or the back to a greater or lesser degree. In some instances this seems to have been so decided as to resemble scarlet fever. Instances

Macular eruption.

of the appearance of a macular eruption resembling measles have been reported and in a few cases disseminated papular eruptions have been described.

Respiration.—The respiratory rate is not markedly increased and this is true to an extraordinary degree even in the presence of a complicating pneumonia so that *any decided increase in the respiratory rate is of importance as suggesting the onset of pneumonia* and a high respiratory rate in an established pneumonia is of ill omen prognostically.

Onset of pneumonia.

It may be said in this connection also that during the epidemic of 1918 the pneumonia cases were free to an extraordinary degree from actual dyspnea or complaint of respiratory distress, though occasionally a feeling of suffocation was experienced and was very troublesome and harrassing.

Dyspnea.

Cough.—Even in simple influenza the cough may be and usually is extremely harrassing and not infrequently paroxysmal. This was particularly true of the epidemic of 1918 and in many of the pneumonia cases it constituted a most serious and exhausting favtor.

It has seemed to the author that since the original epidemic of 1918 there has been a tendency to the occurrence of paroxysmal cough following what seemed to be ordinary "colds." These may or may not represent residual endemic influenzal manifestations. In some instances they are extremely persistent, enduring over long periods, the cough being most troublesome at night, and oftentimes leading to emesis before relief is obtained.

Recurrent coughs.

"Colds."

Emesis.

Nose-bleed.—Epistaxis was a frequent occurrence in the epidemic of 1918.

Epistaxis.

Comment.—*It is important to remember,* first, *that pneumonia is the most frequent and deadly complication of influenza;* second, *that in the form occurring so commonly in the* 1918 *epidemic it was not recognizable by auscultation and percussion in its earliest stages;* and third, *that in a large proportion of such cases it was never positively diagnosticated.*

It is necessary, therefore, to remember primarily that uncomplicated simple influenza endures but a few days and in most instances after the third day is on the decline. If then, the symptoms continue actively beyond this period, the temperature persisting or rising again after reaching low figures or the normal, the signs of prostration increase rather than diminish, the rate of the pulse and respiration is accelerated, and particularly if cyanosis develops in the patient, pneumonia is present in all probability whatever may be the physical signs elicited in the chest.

Cardinal points.

ACUTE HEMORRHAGIC PNEUMONITIS.—The influenza epidemic of 1918 owed its mortality almost wholly to the complicating bronchopneumonia of a peculiar type and to the profound toxic effect exerted upon the heart. This form of pneumonia possessed characteristics which were so nearly constant as to suggest a distinct influenzal form.

Peculiar broncho-pneumonia.

Pathology.—In the first stage, the lungs were moist, greatly engorged and bloody, subpleural extravasations of blood being manifest. The picture was one of acute inflammatory hemorrhagic edema rather than a true pneumonia of the usual type, and a large number of the cases escaped diagnosis on account of the curious divergences from the usual symptomatology together with a suppression of the commoner physical signs of pulmonary consolidation.

Hemorrhagic edema.

Tracheal engorgement.

The bronchial and tracheal mucous membranes were unduly engorged at the outset and with the progress of the disease from the hilus outward the lungs became more voluminous and more intensely hemorrhagic.

Cyanosis.

The peculiar deep cyanosis which was one of the characteristic features of these cases was attributable apparently in part to the condition of the lung but also to the profound toxic influence exerted upon the heart.

As demonstrated by Selby, the lung apices and the peripheral portion of the bases remained unaffected.

On section the lung yielded much fluid, thin and bloody. The tissue was quite friable in the later stage and areas of hemorrhage were frequent.

One of the peculiar developments in connection with the pneumonia cases of this type occurring at the beginning or during the height of the epidemic in 1918 was the absence of decided pleural exudate and even of any considerable amount of fibrin on the pleural surfaces. Histologically, in the earlier stages the alveoli were filled with exudate, serous or hemorrhagic; polymorphonuclear leucocytes were relatively scarce, and the dominant cell was the mononuclear.

Cell destruction.

A striking feature of the disease in its later stages was the apparent tendency to disintegration of cells, in the capillaries and alveoli alike, and often a necrosis of the alveolar walls.

Other features of importance were the occurrence of interstitial emphysema and acute bronchiectasis in many cases. Desquamation of bronchial epithelium was marked and there occurred oftentimes an hyaline deposit along the alveolar and bronchiolitic walls.

Changes in the Skeletal Muscles.—Not infrequently marked degeneration of the skeletal muscles was observed in the fatal cases of influenza in 1918.

Cardinal points.

Symptoms of Influenzal Pneumonia.—*The greatest stress should be laid upon the importance of persisting fever, renewal of fever after primary subsidence, the appearance of cyanosis, an increased respiratory rate even though this be only moderate, an increased pulse rate, and a leucocyte count above normal, as symptoms indicating the onset of a pneumonia. It is especially necessary to stress these points as the physical signs of the condition are so slight, misleading, inconstant and slow in their development.*

Selective site.

Most observers unite in reporting that the earliest signs appeared in the region of the lower angle of the scapula and consisted of simple crepitant rales or small consonant rales which might be only temporary in their appearance, coming and going at varying intervals.

Signs of consolidation, if present at all, seldom appeared until twenty-four hours or more later. The clinical signs of consolidation were relatively infrequent. As shown in Selby's article, which is quoted freely in this section the pneumonia began in a large proportion of the cases in the left lung. Resolution, when it appeared, was usually relatively slow, termination by crisis being the exception rather than the rule.

Roentgenographic Findings.—As an interesting incident of his War service the author was privileged to see a portion of the radiographic work

done by Major John Hunter Selby, Chief of the Roentgen Ray Section, Walter Reed Hospital, in cases of influenzal hemorrhagic pneumonitis.

The X-ray pictures were so clarifying with respect to the beginnings and extension of the pneumonic process and so great a degree of accuracy in prognosis was shown by Major Selby that he feels that a brief consideration of this investigator's findings should be included in this article.

In all 470 patients were referred and of these 386 were found to be suffering from hemorrhagic pneumonitis. So far as possible all cases were submitted daily to radiographic examination by means of the ward unit made available in army hospitals.

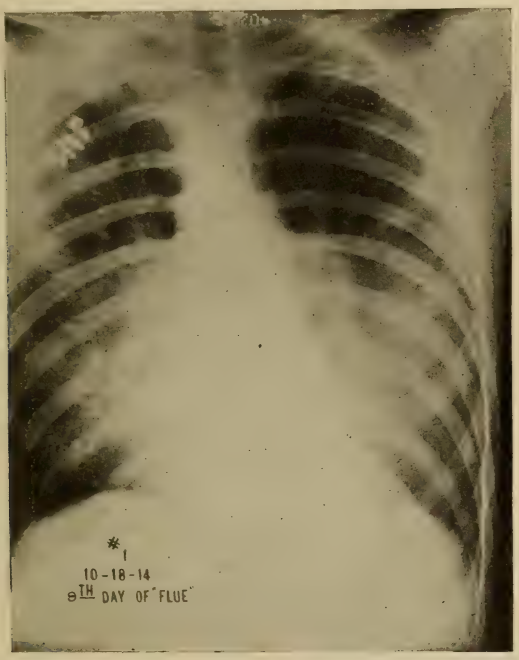


FIG. 491.—The initial film reveals the presence of hemorrhagic pneumonitis in the adjacent portions of the upper and lower left lobes and also in the adjacent portions of the lower and middle right lobes. Note that the density is most intense at the hilum and fades out toward the periphery. Note the clearness of the costophrenic angles on the right and left sides.

The following quotation is taken from Selby's article in the American Journal of Roentgenology.*

Time of onset.

"*Mode of Onset.*—Hemorrhagic pneumonitis usually began coincidently with, or soon after, the incidence of a secondary rise in temperature,† occurring from one to five days after the patient had run what was apparently a typical influenza course. Less frequently it appeared on the fourth or fifth

* American Journal of Roentgenology, 1919, Vol. 6, page 211.

† This observation was noted early in the epidemic by Lieut. J. Harkavy and personally communicated to me. This enabled us to detect a larger number of early cases than otherwise would have been possible.

day after the initial onset of influenza, where the temperature curve was atypical, in that the characteristic fall did not occur. In a few instances, it was detected the day after the patient went to bed.

"*Characteristic Roentgen Ray Appearance.*—In the earliest stage demonstrable by the roentgenogram hemorrhagic pneumonitis is recognized as a faint filmy haze opposite the level of the lower angle of the scapula. The mesial portion of haze is partially obscured by the outer portion of the normal hilum shadow. This hazy area enlarges in all directions and frequently it is observed that the adjacent portions of the upper and lower lobe are involved simultaneously. The process may advance so rapidly as

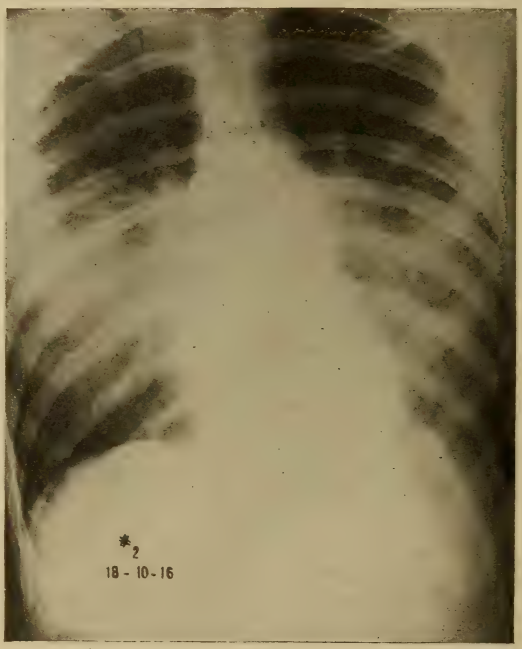


FIG. 492.—Compare with Fig. 491 and note the extent of spread in 48 hours.

Fulminent cases.

to include the greater portion of all lobes in the same side of the chest within forty-eight hours. In fulminating cases all five lobes may become blood-logged and death ensue within forty-eight hours. Hemorrhagic pneumonitis invariably began as a unilateral process, and in 82 per cent. of our cases the left lung was primarily involved. It was never seen to begin in any peripheral portion of a lobe, but invariably appeared in the region of the lung roots. It was also noted that the involvement never began simultaneously in two or more widely separated areas, but it was found to spread from the original site. Later it may develop around the roots of a lobe on the opposite side. The peripheral portions were the last to fill with blood. The true apex and the lower borders of the lower lobes were never involved. We discovered this characteristic absence of apical costophrenic involvement early in the epidemic and requested a check by autopsy. The autopsy con-

Starting points.

Interesting observations.

firmed this, so that we were enabled to utilize the sign in detecting pleuritic involvement. In other words, we found that in every case where the roentgenogram showed a haziness or a total density over the apex or costophrenic angle, it was indicative of a pleuritic complication or lobar pneumonia. In the vast majority of cases we found pleural effusion responsible.

"*Course.*—The hemorrhage showed a tendency to disappear within three days where the area of involvement did not reach a greater diameter than a silver dollar. In moderately advanced cases, *e.g.*, where the invasion was arrested when the involvement included no more than the lower proximal half of the upper left lobe and the adjacent proximal portion of the lower left

Course.

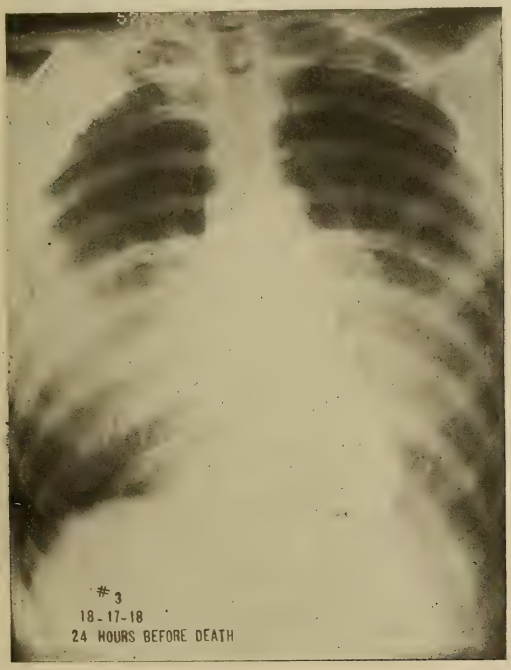


FIG. 493.—The patient was too ill to hold his breath and died within 24 hours after this examination.

lobe, the condition would slowly but progressively fade away, usually disappearing by the twelfth day unless complicated by some other intrathoracic condition. If in such a case there had been a progressive clearing demonstrated over a period of several days followed by an interruption, a complication usually appeared soon thereafter, the most frequent one being a pleural effusion. This often changed into empyema.

"During the height of the epidemic the cases which showed extensive involvement of all five lobes usually proved fatal regardless of whether the involvement developed slowly or rapidly. Later in the epidemic, the virulence diminished and the percentage of fatalities in extensive involvements became conspicuously less. In some cases the process of absorption

Compoete bilateral cases.

of the hemorrhage was noted in one lobe while a complication would develop in the region of another lobe.

"*Prognosis.*—Soon after these daily roentgen ray observations were instituted it was found that the cases presented certain definite characteristics by which a prognosis could be offered with much accuracy. The second or third film usually sufficed to base our predictions on. Our prognosis was made utterly regardless of the clinical data. We based it upon the rate, direction and extent of the spread, interpreted in the experience furnished by the earlier cases.

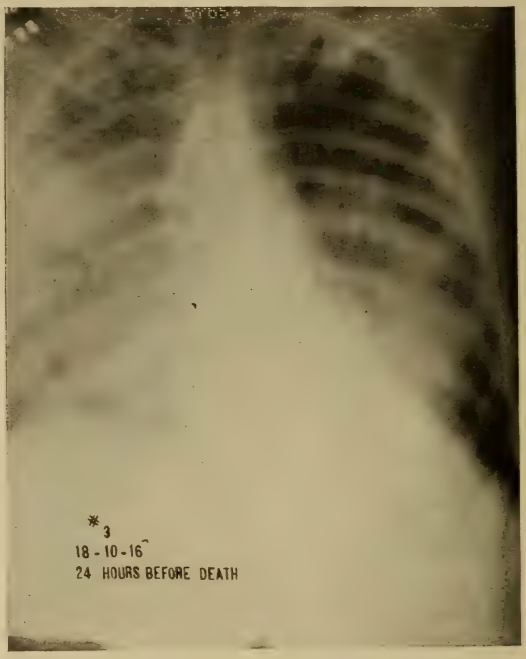


FIG. 494.—Another case showing extensive involvement of lower and middle right lobes, lower portion of upper right and adjacent portions of upper and lower left lobes. The patient was too ill to hold his breath but this figure illustrates clearness of costophrenic angles and true apex. The patient died 24 hours after this was taken.

"The rapidity with which the hemorrhage spread to include the lobes opposite the original hemorrhage bore a direct relation to the gravity of the case. *We did not observe a fatal case in which the hemorrhage remained a unilateral condition, even though all lobes on the affected side became extensively involved.*

"*Ordinarily if the patient survived seven days after the onset of the hemorrhagic pneumonitis, the prognosis was favorable.* However, the convalescence in many cases was interrupted by one or more of the previously enumerated intrathoracic complications. Empyema was the most frequent to occur after the seventh day in hemorrhagic pneumonitis.

Remarkable prognoses.

"In our series, where the roentgen ray observations were possible early in the course of the disease, we erred in two cases where a grave prognosis

had been rendered. In one, all five lobes were involved and a bilateral empyema developed subsequently. This case was interesting in that an entirely different organism was recovered from the right pleura than from the left. In the other case, the convalescence was complicated and prolonged.

"*Theory of Invasion.*—The serial radiographic studies upon the trachea, bronchi, bronchioles, alveoli and their capillaries, interpreted in the light of the autopsy findings, support the conception that the causative organism, whatever it be, gains access through the respiratory tract. It lodges and proliferates in the lower portion of the trachea or in a main bronchus, whence

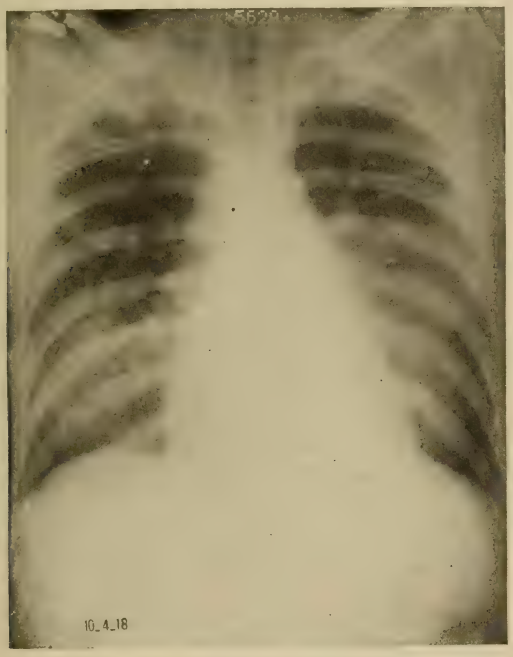


FIG. 495.—Another case showing extensive hemorrhagic pneumonitis in lower right lobe, lower left lobe, and adjacent portions of upper left lobe.

the organisms are distributed by contiguity, reaching first the bronchi and alveoli in the immediate vicinity. These individual colonies elaborate the toxin which produces local destructive change in the cells lining the bronchi, the alveoli, and also the capillary walls, the result being a frank pernicious hemorrhage. The spread is perhaps augmented by the flow or inhalation of the infected bloody exudate into other portions of the respiratory tract. The process seems to develop by spread in contiguity from the original focus in the main bronchus to the alveoli.

Mode of extension.

"The serial radiographs proved that the hemorrhage invariably commenced in the central portion of a lobe nearest the hilum. Later it spread in all directions simultaneously. It is plausible to assume that when the hemorrhage becomes extensive enough all approaches to the peripheral portions of the lobe are likely to be blocked off by the presence of the rapidly

Primarily "central."

accumulated blood in the distributing bronchi. If this is true, the air circulation is retarded and the organisms cannot be transported either by air or by the flow of exuded blood into the more distant alveoli. Autopsy consistently confirmed the conspicuous absence of hemorrhagic pneumonitis in the true apex and in the costophrenic borders of the lower lobes. It was also noted that the alveoli situated nearest the pleura are relatively free from hemorrhage.

Curious immunity.

"This absence of hemorrhage at the apex and in the costophrenic borders, together with the delayed involvement of the peripheral portions of the

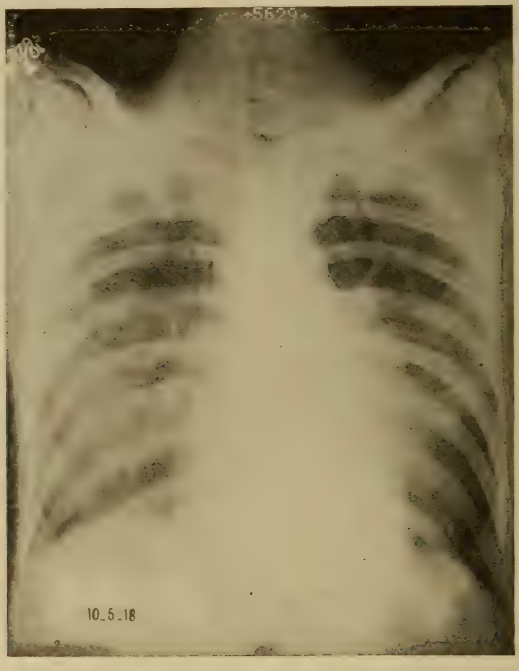


FIG. 496.—Compare with Fig. 495 and note extent of spread in 24 hours. Both costophrenic angles remain clear.

lobes, strengthened the theory that the hemorrhage is due to the toxins elaborated locally. It is also obvious that the hemorrhage cannot be explained on the assumption of a state of hypostatic congestion because the autopsy findings failed to confirm hypostasis in the most dependent portions of the lower lobes. Thus it is obvious that the condition is in no way a septicemia.

"We found it advisable to divide our 470 cases into the following groups:

(*a*) Hemorrhagic pneumonitis uncomplicated. These presented the typical hazy spreading shadow produced by the increased condensation. No discrete mottling was present, nor was there any conspicuous enlargement of the mediastinal glands. No pleuritic complications were present.

(*b*) Hemorrhagic pneumonitis associated with a conspicuous enlargement of the mediastinal glands, but without discrete mottling.

(*c*) Hemorrhagic pneumonitis associated with discrete mottling, but without conspicuous enlargement of the mediastinal glands.

(*d*) Hemorrhagic pneumonitis associated with conspicuous enlargement of the mediastinal glands and with a definite discrete mottling over the pulmonary area.

(*e*) Hemorrhagic pneumonitis complicated by some form of pleural involvement, pericardial involvement and mediastinal empyema.

(*f*) Adenitis unassociated with hemorrhagic pneumonitis, discrete mottling, or any other form of pulmonary or pleural pathology discernible by roentgen ray.

(*g*) Adenitis and fibrosis unassociated with other intrathoracic pathology discernible by roentgen ray.

(*h*) Adenitis without hemorrhagic pneumonitis or discrete mottling but associated with some other intrathoracic pathology.

(*i*) Discrete mottling of the pulmonary area uncomplicated by hemorrhagic pneumonitis, enlargement of the mediastinal glands, or any other intrathoracic pathology discernible by roentgen ray.

(*j*) Discrete mottling associated with conspicuous enlargement of the mediastinal glands unassociated with any other intrathoracic pathology discernible by roentgen ray.

(*k*) Mottling unassociated with hemorrhagic pneumonitis and enlargement of the mediastinal glands, but associated with some other intrathoracic pathology.

(*l*) Hemorrhagic pneumonitis associated with pulmonary tuberculosis.

(*m*) Hemorrhagic pneumonitis associated with lobar pneumonia.

(*n*) Lobar pneumonia uncomplicated by hemorrhagic pneumonitis.

(*o*) Cases in which there was definite clinical evidence of pulmonary pathology but in which the serial roentgenographs failed to confirm the presence of a pulmonary lesion.

"Naturally the cases presenting a discrete mottling offered the greatest difficulty in interpretation, for we did not have preliminary plates by which an estimate might have been made of the previously existing chronic lesions; such as peribronchial adenitis, chronic parenchymatous, tubercular changes, etc. However, we were soon enabled to identify the mottling by autopsy. It was found to be due to the discrete interstitial lesions, commonly held to be streptococcic in origin. In our cases the sum total of mottled types was so small as to be conspicuous.

"The total number of patients studied roentgenographically was 470. Of these 82 per cent., or 386, presented hemorrhagic pneumonitis complicated or uncomplicated by one or more of the intrathoracic conditions already noted. Of the remaining 84 patients, 33 failed to show any lung pathology by the roentgenogram, although clinically there was evidence of so-called bronchopneumonia, 7 were Group (*g*) (mediastinal adenitis), 13 were Group (*i*) (discrete mottling uncomplicated); the remaining 31 could not be classified, as the presence of fluid completely obscured the original pathology.

"Of the cases which came under roentgen ray observation early enough,

the hemorrhagic process was found to appear primarily in the left lung in 82 per cent. This is a striking fact, but no less remarkable than the observation that in our 91 cases in which there was unmistakable evidence, the initial invasion appeared in the lower left lobe eighty-five times to six in the upper left lobe.

"Group (*a*) includes a few cases where autopsy revealed a small amount of pleural fluid, which roentgenographically was not present twenty-four hours before death. Presumably this developed as a terminal process. The importance of serial roentgenograms in influenza has been amply proven in this clinic, chiefly in detecting and identifying pulmonary complications in the early stages.

"We had no deaths from Group (*f*) or (*g*) and so far we have not seen a single instance of fatal influenza unassociated with an intrathoracic complication."

Actual vs. relative mortality.

Prognosis in Influenza.—*As has been said, influenza is a disease of enormous actual but low relative mortality;* that is to say that, considering the universality of an epidemic and the enormous numbers of individuals attacked, the death ratio is small, yet the total number of deaths is enormous and the indirect mortality adds greatly to the figure. The enormous mortality, the fact that it kills the adult by preference, and especially those engaged in active productive work, has opened the eyes of sanitary authorities to the necessity for quarantine regulations, both as applied to the State and to the household.

As to the prognosis of simple uncomplicated influenza, one may say that there is only a small mortality save in individuals suffering from chronic disease, greatly debilitated, or of an advanced age.

The onset of pneumonia at once converts the case into one of extreme danger.

Pregnant women suffer very greatly, usually abort especially if the pregnancy is advanced, and show a mortality which in certain instances has been reported as reaching 60 per cent.

In the epidemic of 1918 it was found that those individuals who survived for a week or ten days after the onset of pneumonia usually recovered. Furthermore, as has been stated by Selby quoted in this article, those cases which showed only a unilateral pneumonic involvement yielded a relatively slight mortality.

As stated previously, the heart suffers seriously from the toxemia of this disease and those who carry a chronic heart lesion are more greatly endangered during the attack and are prone to show its effects for long periods thereafter. A failure to appreciate this fact results very seriously for an enormous number of cardiopaths and every physician should watch the heart carefully during convalescence and prolong this so far as is necessary to secure for the patient the best possible reëstablishment of his myocardial reserve.

ASIATIC CHOLERA

("*The Death Blow*")

Definition.—*An acute infection caused by the comma bacillus of Koch and characterized by profuse rice-water diarrhea, violent muscle cramps and collapse.*

Historic Note.—It is one of the most ancient of diseases in the East, but of comparatively recent development in the United States, the first epidemic having occurred in 1832. An ancient scourge.

FIG. 497.—Cholera Bacillus (comma bacillus or spirillum choleræ of Koch). Non-sporogenous, flagellate, motile, parasitic, saprophytic non-chromogenic aërobic and facultative anaërobic, liquefying, spirillum, readily stained but Gram negative.

Etiology.—Koch reported his discovery of the comma bacillus in 1884. The germ retains its vitality for a week or more on foodstuffs, such as butter, milk, and meat, lives but a day or two in oysters, but finds sewage a good culture medium. Water is a great conveyor and flies also carry it, but it travels no faster than man, following the trade channels, whether these be railways, canals, rivers, or steamship lines, tending to spread from India, where it is endemic, to all parts of the civilized world. The great pilgrimages, fairs, and festivals serve to disseminate it widely; filth, overcrowding, and infected water and food being part and parcel of such gatherings.* Readily conveyed. Difficult to control.

The disease is readily conveyed by fomites, but is essentially water-borne. The city of Hamburg in 1892–93 had 18,000 cases with a mortality of over 40 per cent., whereas Altona, a suburb, had but 516 cases. Both cities drank the same water, *but Altona filtered hers*. One convalescent workman from Marseilles once infected the water of a large reservoir, and through it a large district of the city of Paris.

Cholera is a hot weather and low altitude disease, the germ is readily destroyed by sunlight, but vast quantities of bacteria are discharged in the stools and many objects may serve as indirect carriers. It is not highly contagious and scrupulous care in regard to the dejecta permits nurses and physicians to perform their duties without fear of infection. Age and sex seem to cut no figure as regards disease incidence, and neither absolute nor relative immunity is well proven.

Morbid Anatomy.—Rigor mortis is extraordinarily rapid, producing horrible post-mortem movements of the extremities, lower jaw and even the eyes. The tissues are dry and shrunken, the blood is thick and tarry, and there is an intense congestion of the stomach and intestines and cloudy swelling and parenchymatous degeneration of the viscera.

Incubation Period.—*The incubation period is from two to five days.*

* The British have found the utmost difficulty in checking its development and dissemination. Native visitors to holy shrines bathe in holy wells and drink of the precious water which contains the washings of many bodies. Sick and well are crowded together, dirt abounds, ventilation is deficient, and in event of an epidemic the care of the sick and the proper disposition of the dead are alike neglected.

Preliminary Stage.—*When present, this lasts but a few hours and presents malaise, marked mental depression, headache, diarrhea, nausea, vomiting and colicky pains. In most instances the onset is abrupt.*

Typical attack.

Stage of Purging and Collapse.—This is characterized by profuse *and almost constant serous discharges from the bowel,* accompanied by *tenesmus, intense thirst, and excruciating muscle cramps* especially affecting the abdomen and calves of the legs. *Vomiting* is almost incessant, *acidosis profound,* the *countenance Hippocratic, cyanosed and shriveled, the skin wrinkled, moist and cold, the superficial temperature greatly subnormal, though the rectal temperature may reach* 103 *to* 105°F., *the blood pressure minimal* (60–75).

Profound toxemia.

The hemoglobin percentage is high and the blood is obtained with difficulty. The pulse soon becomes excessively rapid, weak, flickering, or even absent at the wrist, the albuminous urine scanty or suppressed, the tongue and throat dry, yet the patient sweats profusely. The stools are of the characteristic "rice-water" type and the patient becomes comatose or passes into the third stage. The duration of this stage of collapse is from a few hours to two days.

"Rice-water stools."

Third Stage (*Reaction*).—All symptoms gradually subside and a tedious convalescence ensues. Various late eruptions may occur.

The Blood.—There are both *polycythemia* and *leucocytosis* in the algid stage, the red cells varying from 6,000,000 to 8,000 000 and the leucocytes from 14,000 to 60,000 per cubic centimeter.

Acetonemia and uremia must be guarded against even in this stage.

Prognosis.—The mortality varies from 25 to 75 per cent. in different epidemics, death may occur in relapse, in cholera typhoid, when the delirium is marked, and coma rapidly supervenes, or the patient may die even before the rice-water stools appear (*cholera sicca*).

Diagnosis.—In the absence of an epidemic, cases of poisoning by certain minerals, ptomains, or mushrooms may cause difficulty.

The so-called *cholerine* is a very mild form of the disease, accompanying certain epidemics, and isolated cases offer great difficulties in diagnosis.

Cholera nostras (cholera morbus) in its severer forms exactly simulates true cholera, but is usually a paratyphoid infection.

Positive Differential Symptoms.—*The germ must be recovered and cultivated from the stools, and the diagnosis thus absolutely established.*

In the presence of an epidemic a *tentative* diagnosis may be made in a majority of the cases from the appearance of the vibrios in a smear from fecal mucus and the "scintillating motility of the organisms" as viewed in the hanging-drop.

Expert handling is absolutely necessary to positive diagnosis which involves the application of the agglutination test with known immune sera, the use of Pfeiffer's method, and the nitroso-indol reaction.

BUBONIC PLAGUE

(*"Black Death," "Pest," Malignant Adenitis*)

Definition.—A *virulent epidemic disease,* highly infectious but not markedly contagious, caused by the *bacillus pestis* of Yersin, and characterized

by *high fever, great prostration*, the *formation of buboes, a tendency to hemorrhage*, both subcutaneous and from mucous membranes, and *a remarkably high leucocyte count.*

Historic Data.—It is as old as the Pyramids. After the sixth century it repeatedly visited Europe and Great Britain, killing during less than one century in London alone, 161,344 people; indeed, the epidemic of 1665 caused 68,000 deaths, the total population of London being at that time under 500,000. The Indian plague of 1889 is said to have been responsible for at least 250,000 deaths and in the fourteenth century it is said to have carried off not less than one-fourth of the total population of Europe. It is endemic in China and India, and a frequent visitor to the Philippines. "The Killer."

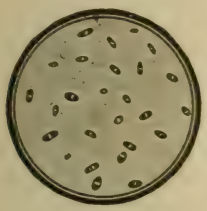


FIG. 498.—Bacillus of Bubonic plague (bacillus pestis of Yersin). Minute, non-motile, non-sporogenous, non-chromogenic, pleomorphous, aërobic and facultative anaërobic organism, easily cultivated and stained, but Gram negative.

Etiology.—*It is distinctly a rat disease*, and its development and spread occurs under conditions favoring the development and multiplication of these animals. The germ is an encapsulated, short, round microörganism, discovered in 1894 by Yersin and Kitasato working independently. It lives long and thrives in urine, sputum and fecal matter.

The disease is communicable, chiefly through droplet infection in pneumonic cases, but it is probable that only a small proportion (3 per cent.) of the cases are so contracted.

So far as the human being is concerned, nearly all cases acquire the disease through the fleas infesting infected rats, the chief carrier being the *Epimys norwegicus*.

In California, ground squirrels have been proven to be carriers.

Morbid Anatomy.—That of an acute infection with profound toxemia. The lymph vessels are chiefly affected, the glands being enlarged, edematous, and hemorrhagic, or showing suppuration and external ulceration. The bacilli are generally distributed, the kidneys and spleen are hyperemic and the lungs may show pneumonia and infarction. Primary and secondary carbuncles, ecchymoses and dermatitis may be present and the viscera generally show fatty and parenchymatous degeneration with marked hyperemia.

PESTIS MAJOR.—Symptoms.—*Incubation period two to ten days. Prodromal period from twenty-four to forty-eight hours. Pallor, vertigo, and profound muscular weakness are associated with nausea, vomiting, and diarrhea. Nose-bleed is frequently present and mental depression marked. The fever rises steadily until just before the active stage.*

Active Stage.—*This is characterized by chill, high fever* (104 to 106°F.), *and rapid pulse* (120 to 200). *The expression is anxious, countenance livid, conjunctivæ congested, the skin dry and hot. After three or four days buboes appear in from 70 to 80 per cent. of the cases. The inguinal and femoral glands are affected primarily in 60 per cent. of the cases. Suppuration is a favorable* Buboes.

Plague spots.

sign, but gangrene may supervene. Petechial or extensive ecchymoses may appear (plague spots), as may other skin eruptions of various types. Any mucous membrane may be the seat of hemorrhage.

High counts Virulent types.

The Blood.—The leucocytes average more than 90,000 to the cubic centimeter, and there is marked polycythemia, the average count of red cells being about 7,000,000 per cubic centimeter.

Varieties of Pestis Major.—(*a*) *Septicemic plague.* In this form buboes do not appear, the organism is overwhelmed and death occurs in a few days or hours. (*b*) *Pneumonic plague.* In this the lesions are chiefly and ordinarily those of broncho-pneumonia, the sputum is loaded with germs and about 90 per cent. of the cases die.

Misleading form.

Pestis Minor.—This mild form has *slight fever of short duration, glandular swelling with or without* suppuration, and may easily be misdiagnosed. Such *cases, unfortunately, are the usual forerunners of epidemics.*

The Diagnosis.—Lacking an epidemic, the diagnosis must sometimes depend upon the recovery of the germ, as the agglutination reaction, though often obtainable, is not absolute and occurs late in the disease. Reliance is to be placed chiefly upon: (*a*) *Prevalence of an epidemic.* (*b*) *Profound exhaustion.* (*c*) *Anxious countenance and mental depression.* (*d*) *Buboes which are characteristically edematous.* (*e*) *Petechiæ.* (*f*) *Hemorrhage.* (*g*) *Leucocyte count.*

Cardinal points.

Pathognomonic Sign.—*There is but one, viz., the recovery of the bacillus from the blood, urine, sputum, or feces and its positive identification.*

Usually easy.

Differential Diagnosis.—*Tuberculous adenitis.* Fever absent or intermittent, development slow and *comparatively* painless; this could hardly be confounded even with pestis minor. Syphilis presents initial lesion or cicatrices, sore throat, characteristic eruption of mucous patches and its buboes do not suppurate. *Chancroid,* initial lesion, strict localization of bubo. *Hodgkin's disease and gonorrheal bubo* should not be confusing, but nevertheless *are* sources of disastrous error.

Prognosis.

The mortality is always large and even in those inoculated with antiplague serum it is said to be 14 per cent. In some epidemics it runs from 50 to 95 per cent.

DENGUE (deng-ga)

("*Break Bone*" or "*Dandy*" *Fever*)

Definition.—*A mosquito-borne tropical and subtropical disease of unknown origin, low mortality, high infectivity, and slight contagiousness, characterized by its sudden onset, severe muscular and joint pains, irregular rashes, double febrile paroxysm, and tendency to relapse.*

A mosquito host.

Etiology.—It is probably due to an ultramicroscopic organism as Ashburn and Craig found blood highly infective, even though passed through a porcelain filter and as reported by H. Graham, of Beyrout, Syria, in 1903, the *Stegomyia fasciata* is probably the intermediary host and source of infection. Ashburn and Craig failed to find the parasite reported by Graham though

confirming his report that the mosquito acts as a carrier of the specific filtrable virus. Filtrable virus.

Incubation Period.—Three to six days, no prodromata.

Symptoms.—*The onset is extremely abrupt with chill, high fever, and rapid pulse, headache, muscular, joint and deep-seated bone pain associated with extreme tenderness. Both large and small joints may be swollen. Red and enlarged lymph glands are frequent and, often, long persistent vomiting may occur, the face is deeply congested, and a rash of short duration, irregularly distributed, and variable in character is often present. Initial symptoms usually include extreme pain in the eyes* and a blotchy facial erythema.* Abrupt onset. Arthritic onset.

On the second, third, or fourth day *a crisis occurs with the usual phenomena, and is followed by a period of remission of all symptoms, lasting from twelve hours to three days;* **then follows a reinvasion** *less severe than the first, terminating in a second crisis after three or four days or even a few hours, and during this stage a somewhat characteristic rubeolar eruption may appear, first on the hands then over the body, but always most markedly over the hands, wrists, elbows, and knees, occasionally forming a diffuse red rash by coalescence. Desquamation follows and is usually furfuraceous.* Remission. Initial and terminal erythemas.

A second remission and third paroxysm may occur, or the first remission may terminate the disease. In more severe cases, jaundice, black vomit, marked albuminuria and hematuria may occur and suggest "yellow jack." A decided leucopenia is observed in this disease. Severe forms.

Differential Diagnosis.—(See Yellow Fever.)

Prognosis.—There is practically no mortality.

YELLOW FEVER

("Yellow Jack," "Bronze John")

Definition.—A virulent infectious but non-contagious disease, characterized by sudden onset, high fever, jaundice, black vomit, and slow pulse.

Etiology.—At the present time it is believed that the *Leptospira icteroides* of Noguchi may be the specific casual factor. A commission, headed by the later Walter Reed, U. S. A., in 1900–01 proved the *intermediary host and transmitter of the filtrable virus* to be the mosquito known as the *Stegomyia calopus fasciata*. This commission furnished one of the most brilliant examples of scientific acumen and heroic self-sacrifice in the history of medicine. Dr. Lazear became infected and died. Non-immune volunteers slept for twenty days in the stained sheets taken from the beds of yellow fever patients, their heads wrapped in soiled, bloody cloths, yet all these, being protected from mosquitoes, escaped the disease. Those isolated for long periods and then exposed to multiple inoculation by mosquitoes that had fed upon yellow fever patients, in nearly every instance developed the disease. It was found that mosquitoes required twelve days to develop virulence, and retained their infectivity for eight weeks or more. A subcutaneous injection of yellow fever blood produced the disease even though the blood were defibrinated and passed through a porcelain filter. Major Reed's great work. Heroism of volunteers. Period of virulence. Blood infective.

* This may also be marked in yellow fever.

Simple preventive measures.

The experiments proved that the only quarantine measures necessary were those calculated to destroy mosquito life in houses or on shipboard, and that the disease is not conveyed by fomites; further, that to control an epidemic it is only necessary to screen the sick and the houses of the well, destroy all resident mosquitoes, and to treat marshes, stagnant pools, water barrels, and other breeding places with petroleum which destroys the surface-breathing larvæ.

Substantial proof.

Through such measures, Santiago, Havana, and other towns were rendered free from yellow fever by the government medical authorities after the Spanish-American war.

Seasonal Influences.—All that was previously written regarding these matters now resolves itself into a discussion of mosquito life and propagation, and it has always been recognized as a summer and early autumn disease, killed by frost. **Race.** The whites are chiefly affected, though negroes are not wholly immune and visitors contract the disease more readily than those acclimated. **Sex.** There is equality of susceptibility, though on account of greater exposure males chiefly predominate. **Age.** It resembles typhoid in its preference for the young adult. Old people and infants ordinarily escape, the latter probably from minimum exposure. **Immunity.** One severe attack protects for life.

Morbid Anatomy.—Diffuse nephritis, gastric hyperemia with hemorrhagic stomach contents, hemoglobinemia, general glandular enlargement, cutaneous hemorrhages, and deep jaundice are the usual findings.

"A bolt from the blue."

Symptoms.—*The first stage strikes "like lightning from a clear sky," with chill and high fever* (103 to 106°F.) *accompanying a singularly slow pulse* (80 to a temperature of 104°F.); there is *excruciating pain in the loin and in the leg muscles, profound muscular weakness, violent headache, sore throat and vomiting, and the mental depression is often marked. A slight jaundice commencing in the eyes and extending to the chest, arms, and body may appear even in this stage.*

Early jaundice.

Albuminuria may be marked as early as the second or third day.

There is photophobia frequently associated with intense orbital headache; vomiting, increasing in intensity until black vomit may appear; the tongue is swollen and beefy or often small and pointed, and the gums are tumid. The pulse is not primarily slow, feeble, and compressible, but tends to decline even with rising temperature.

Black vomit.

Remission.

Physiognomy.—The *expression is thought by Guiteras to be quite characteristic. The cheeks and conjunctivæ are congested, the eyes staring, humid and ferrety, the general expression "anxious."*

Characteristic facies.

Second Stage (duration from a few hours to thirty-six hours).—On the third or fourth day all symptoms abate leaving the pulse slow (even 30 or 40) and compressible, the temperature may be normal and recovery follows in mild and favorable cases, though convalescence is often protracted and accompanied by irregular fever.

Third Stage.—In severe cases a stage of intensification of the initial symptoms and collapse follows a remission. Its duration is from one to three days. The temperature usually rises slowly to 102° or 104°F., or may

rise steadily until death, though the pulse may fall to 60 or even 30 beats per minute. The *jaundice deepens, black vomit occurs, albuminuria is invariable, hematuria commences, total urinary suppression is not unusual, and hemorrhage may occur from the nose, gums, lips, rectum, or any other mucous membrane, the skin may be of a deep mahogany color, and various irregular eruptions may occur. The late Dr. West, of Galveston, of wide experience and a keen observer, always insisted that the renal symptoms overshadowed all others in severe cases. A "butcher-shop odor" is marked in this disease.*

Falling pulse.

Hemorrhages.

Renal symptoms.

Diagnosis.—Essential Data.—Mild cases ("acclimatation fever") may be unrecognizable, but in marked cases one especially notes: (*a*) *Sudden onset without prodromata.* (*b*) *Presence of an epidemic or history of exposure.* (*c*) *Physiognomy.* (*d*) *Low initial pulse rate with high temperature.* (*e*) *Falling pulse rate and volume with rising temperature.* (*f*) *Excessive gastric irritability.* (*g*) *Early jaundice and later extreme pigmentation.* (*h*) *Black vomit, epistaxis and bleeding gums.* (*i*) *Albuminuria with a tendency to suppression* (predominance of renal symptoms). (*j*) *General tendency to hemorrhage.* (*k*) The *apparent* slight loss of red cells per cubic centimeter is a marked feature in view of the hemorrhagic condition.

Faget's sign.

Differential Diagnosis.—*Dengue.*—No mortality, joint symptoms usually more pronounced and *persistent*, yellow fever pulse-temperature ratio absent, jaundice seldom marked and rarely as *early* as the third day, black vomit usually absent, renal symptoms not so prominent or so early, rashes common and somewhat characteristic, and hemorrhages rare save hematuria.

Seldom offers real difficulty.

Malaria.—Plasmodium in the blood, sequence of phenomena usually widely different, enlarged spleen, response to quinin, renal symptoms less prominent, jaundice later, facies of yellow fever absent, albuminuria delayed and hemorrhage rare. Only the irregular forms of malaria can by any possibility be confounded with yellow fever. *Acute yellow atrophy, Weil's disease and relapsing fever lack the chief symptoms of "yellow Jack."*

Prognosis.—It is largely determined by the height of the original fever. In Sternberg's analysis of 269 cases, of those characterized by an initial temperature of 105 to 106°F., 61 per cent. died; of 104 to 105°F., 30 per cent. died; of 103 to 104°F., 6 per cent. died; of 103°F., none died. *Recovery may ensue in any case, however severe, but convalescence is slow.*

Relation to fever.

MALARIA

(*Marsh Miasm*)

Definition.—*An infectious disease caused by the hemameba or plasmodium malariæ and characterized in its common forms by periodic paroxysms, consisting of chill, fever, and sweating periods, and by its response to quinin.*

Historic Note.—One of the most ancient of diseases, malaria has been carefully studied in all ages, and when, in 1880, a French army surgeon, Laveran, described its special organism, and Patrick Manson and Ross, a little later, demonstrated the rôle of the mosquito as intermediary host, it

Mosquito intermediate host.

was found that little was needed to make all previously recorded and accepted observations fit the newer theory. Everyone knew that the season of maximum prevalence of malaria was the spring, summer, and early fall, that it prevailed most extensively in the tropics, that it tended to follow water courses and haunted the neighborhood of stagnant pools and fresh, brackish marshes, that salt marshes were free from it, and that sandy-bottomed *fishy* pools might abound without malaria. Its curious horizontal spread, the fact that dwellers in upper stories might escape it, the danger of night exposure, its rapid extension with the drift of prevailing winds, its disappearance under drainage and cultivation of land, all these things were thoroughly understood, even by the ancients, and each and every one corresponds to the life history of the mosquito. The carrier is the female anopheles, a mosquito never absent from malarial districts, although anopheles may be present in non-malarial districts, the organism being absent. The world-wide distribution of malaria makes it a disease of great importance, although, generally speaking, of low mortality.

Conditions governing incidence.

Fifteen thousand are said to die annually of malaria in Italy. It has been estimated that over 3,000,000 cases occur annually in Russia. Japan, India, China, and the Philippines suffer greatly, and the yellow races seem to have little or none of the immunity so evident in the Congo-black or pure negro, the Japanese, Chinese, and East Indian being readily infected.

Characteristics of the Mosquito.—There are probably over 500 varieties, the general appearance and character being too well known to require description. Like others of the order of diptera, they lay eggs from which come the larvæ after two or three days of warm weather. These feed energetically on the water-borne organic matter and are air breathers, floating on the water, or possessing a respiratory funnel placed near the tail; hence they may be easily destroyed by the use of petroleum on the surface of their breeding places. The grown male insect is a vegetarian, the female seeks a mixed diet and sucks the blood, not alone of man, but of mammals generally, and, it is said, of birds, reptiles, and even of fishes. The time required for full development is about thirty days in warm summer weather and the hatching process may be repeated several times during the season, one pair of insects producing millions of their kind.

Air breathing larvæ.

Easily destroyed.

The Special Characteristics of the Anopheles.—Length of palpi and proboscis about equal, the former four-jointed in the female and three-jointed in the male. The straight filamentous palpi are held almost parallel with the proboscis, the wings are often spotted, the abdomen is pilose with no scales, the legs are long, ending in dentate claws, and the nucha has posterior scaly cornua.

Color in Relation to the Mosquito.—Nuttall has reported some interesting observations in regard to the color preferences of mosquitoes. He found that of the various colors ranging from white to black, the former was the least, the latter the most attractive. The observations are of distinct value in relation to the proper colors for clothing for dwellers in mosquito-infested communities.

Dark colors attract.

Classification of the Organism.—As will be seen from the plates illustrating the cycle of development in the human body, the malarial organism is

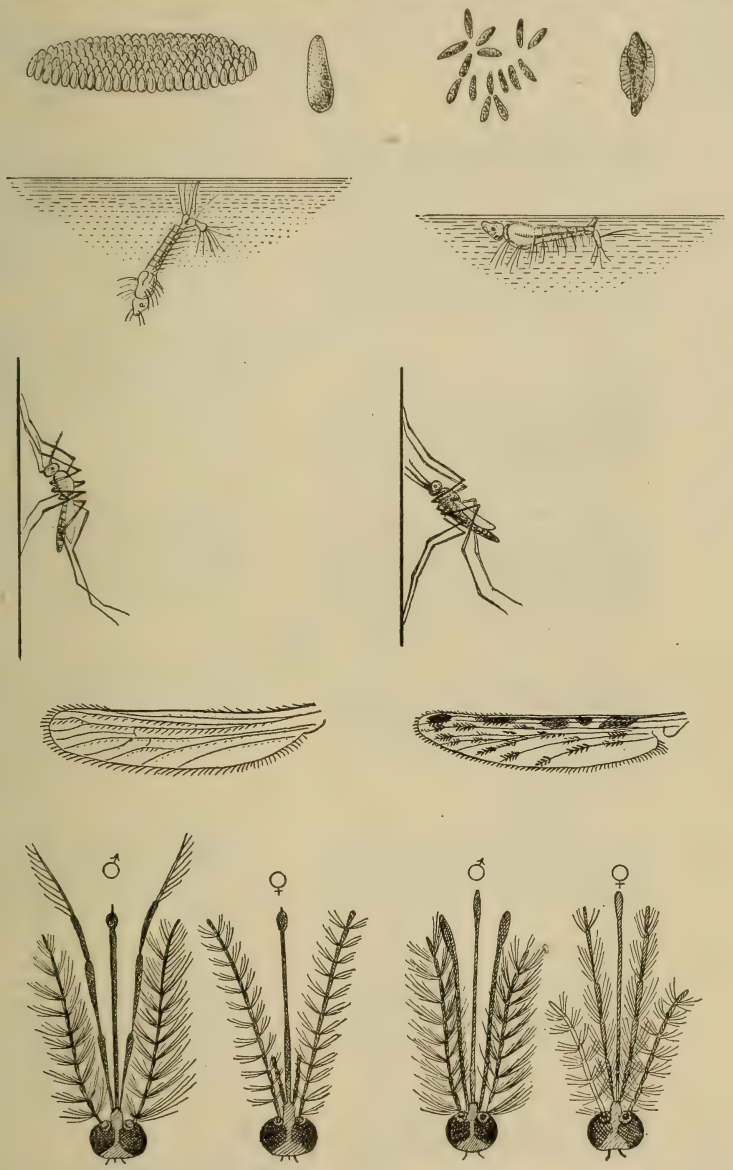


FIG. 499.—*Culex* (left) and *anopheles* (right) mosquitoes and larvæ. In the above figure note the culicine egg raft, 45° angle position of siphonate larva, parallel attitude of resting mosquito, non bulbous palpi of male and short palpi of female as contrasted with the anopheline star or ribbon arrangement of eggs, horizontal attitude of asiphonate surface larva, brad-awl attitude of resting mosquito, spotted wings, bulbous palpi of male and long palpi of female mosquito. (*From Jordan after Kolle and Hetsch. Stitt.*)

easily recognized and differentiated by blood examination and the expert observer can also closely predict the time of segmentation and recognize double or triple infections.

The clinical divisions correspond to the old time relationships, based upon the frequency and regularity of the seizures.

The tertian organism (*p. vivax*), *requiring forty-eight hours for its development,* brings about a paroxysm on each third day, the interval of apyrexia being one day.

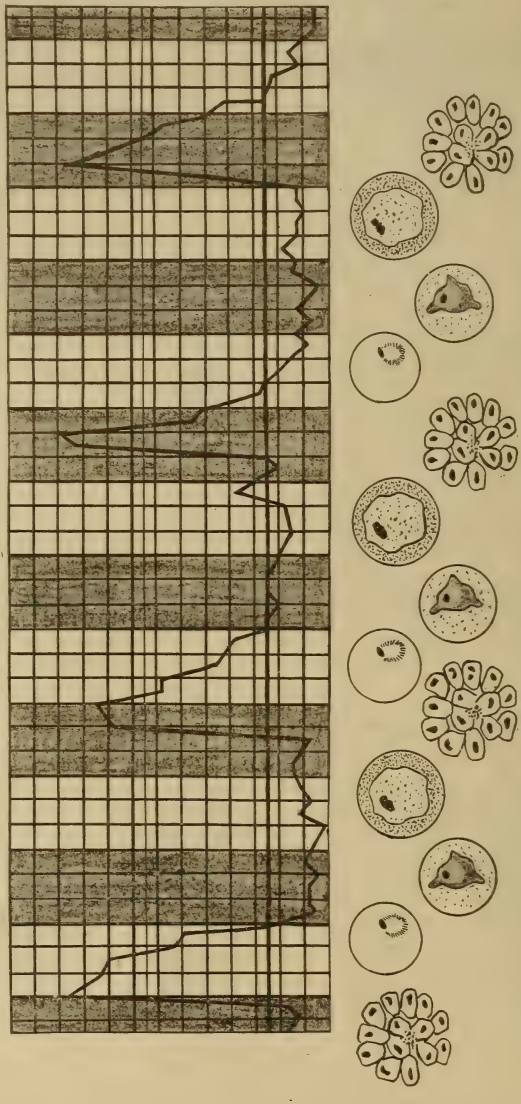


Fig. 500.—Malarial fever. Simple tertian form. The developmental cycle in this form occupies forty-eight hours. Hence attacks recur on each third day, the wholly free interval occupying one day. (*After Marchiafava and Bignami, modified.*)

The quartan (*p. malariæ*), *requiring seventy-two hours for its development, causes a paroxysm on each fourth day, with two days of apyrexia.* If two tertian organisms are developing, the paroxysm assumes a daily type; in the case of double quartan infection, attacks appear on two successive days, leaving one day of apyrexia. If, as rarely happens, there be a triple quartan

Double infections.

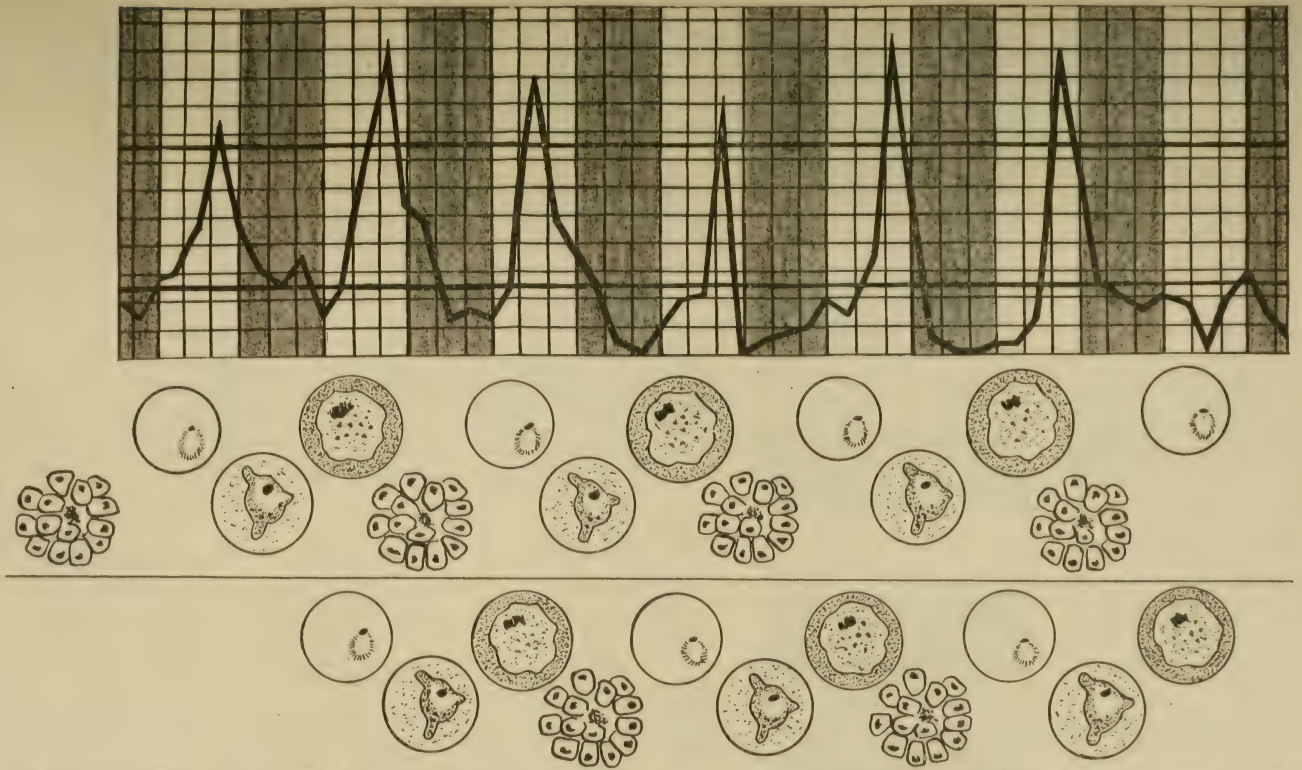


FIG. 501.—Malarial fever. Double tertian infection. Quotidian fever. Alternating vertical and shaded areas each represent a twelve-hour period. Below, the upper figures show the primary cycle; the lower, the secondary cycle, both in the proper order of development and correct relationship to the fever curve above. Sporulation and the resulting attack occur every forty-eight hours in simple tertian, but the presence of a second generation produces a daily seizure, as is clearly indicated by the time alternation of the sporulation as shown above. (*After Marchiafava and Bignami, modified.*)

infection, the disease assumes the quotidian or daily type, as in the case of the double tertian.*

Severe form.

Estivo-autumnal Plasmodium (*p. falciparum*).—This is irregular in its development and manifestations and is the variety which causes the severer, more chronic, malignant, or pernicious types of the disease. In the United States the tertian organism is exceedingly common; the quartan rare. The accompanyng plates give a description of the various forms and show their progressive development at the expense of their erythrocyte host.

Quartan rare.

Evolution of the Organism in the Mosquito.—Two days after the ingestion of human blood containing sexual forms (gametocytes) small pigmented

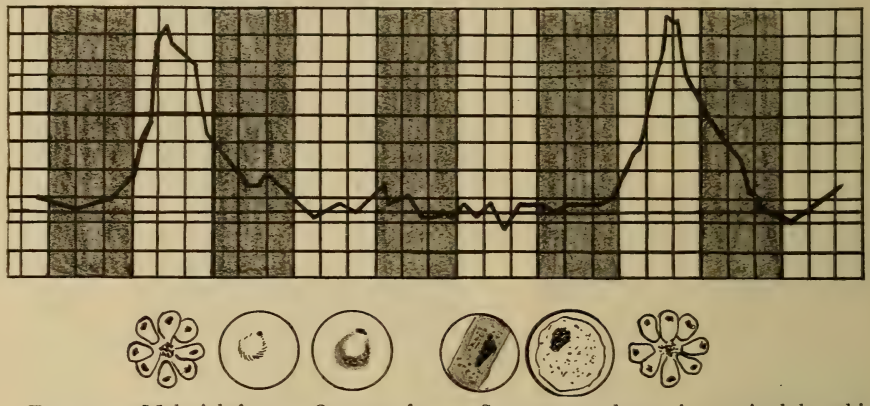


FIG. 502.—Malarial fever. Quartan form. Seventy-two hours is required by this form for the development of its cycle. Hence segmentation and the associated malarial paroxysm falls on each fourth day, the wholly free periods occupying two days. Double and even triple infections may reduce the apyretic interval to a single day or to zero. (*Marchiafava and Bignami, modified.*)

granular bodies (zygotes) are formed which appear in the muscular coat of the mid-intestine of the mosquito host as oökinetes. These *zygotes* result from the conjunction of the spermatozoa-like, actively motile flagellæ of the microgametes of the male bodies (microgametocytes) with the female cells (macrogametes) and represent, therefore, the fertilized female element transformed into a worm-like body (vermiculus or oökinete) which penetrates between the cells and develop the oöcyst.

Five days later striation appears representing the *sporoblasts* which contain and later set free the tiny sickle-shaped embryos (*sporozoites*) by the rupture of the parent oöcyst. These enter the salivary glands, and thence pass by the venenosalivary duct to the pharynx of the mosquito and are thus passed into the blood of man.

The sporozoites of the mosquito cycle and the spores of the human cycle (merozoites) are essentially like bodies and in the human host may pursue either a sexual or an asexual development.

The human cycle is primarily and for several generations asexual or

* Craig, Manson and others have reported a quotidian type of organism which they call *Plasmodium falciparum*.

sterile, producing *schizonts* only, but later the sexual forms (*gametes*) are developed, the one *male* (*microgametocyte*), the other *female* (*macrogametocyte*). The former has a clearer protoplasm, stains less deeply blue, has less

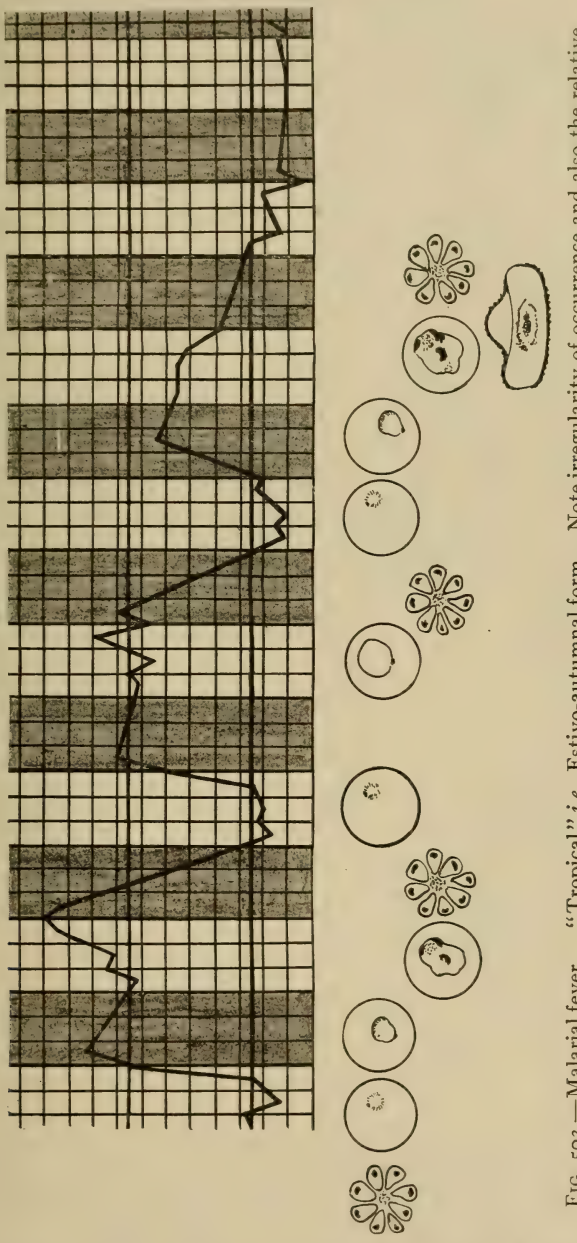


FIG. 503.—Malarial fever. "Tropical" *i.e.*, Estivo-autumnal form. Note irregularity of occurrence and also the relative persistence of the febrile stages.

pigment, more chromatin and a more centrally placed nucleus than the latter. (See Fig. 408.)

Cultivation of the Parasites.—The first successful cultivation of the plas-

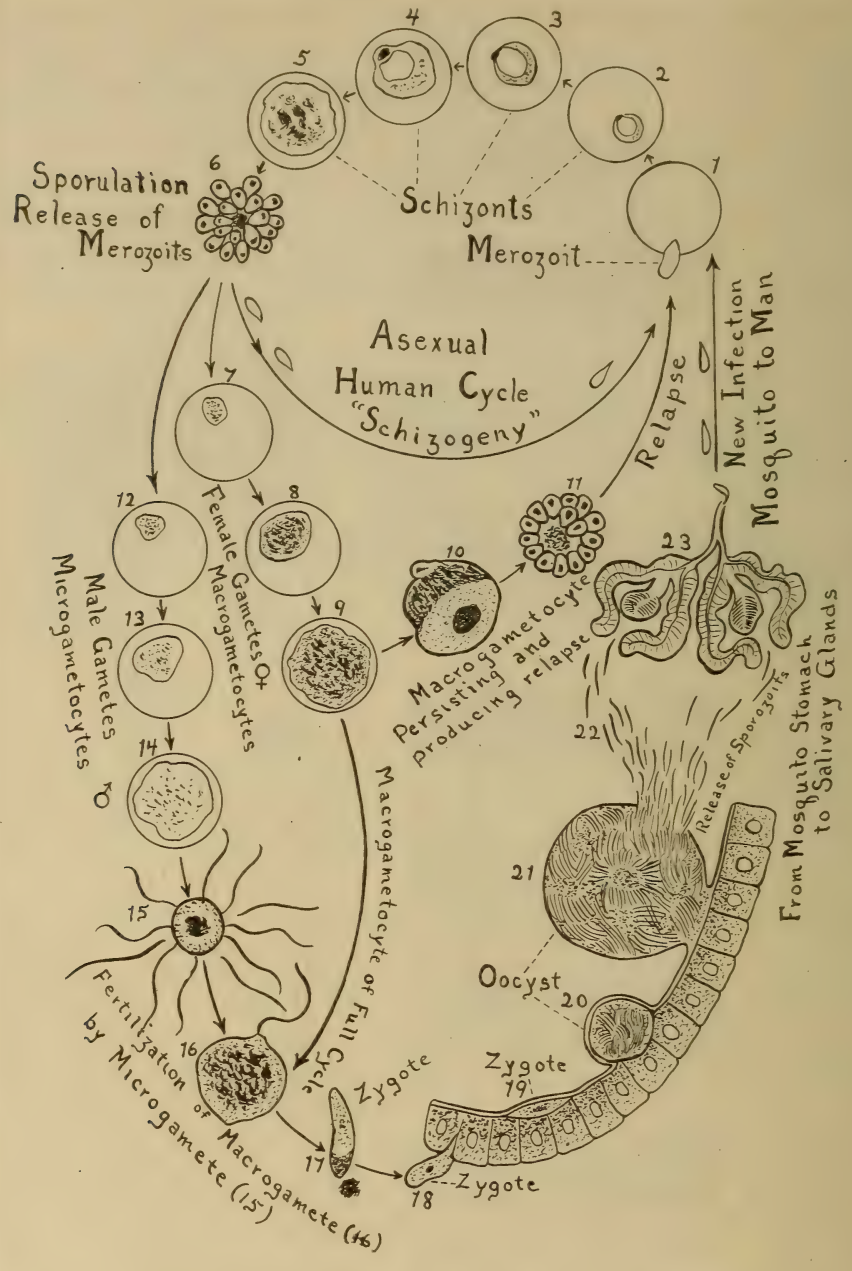


FIG. 504.—Developmental cycle of the malarial parasites. (1) Youngest form. (2, 3, 4, 5) Stages of enlargement and pigment formation. (6) Sporulation. (7, 8, 9) Female forms. (10, 11) Female forms going on to sporulation within the human body and producing relapse. (12, 13, 14) Male forms, development terminating in a conjunction with female form of the sexual cycle within the body of the mosquito. (15) Flagella-like microgametes formed from male cell. (16) Conjunction of male and female elements. (17, 18, 19) Zygote (impregnated female form) passing under epithelial lining of mosquito stomach. (20) Development of sporoblasts and formation of oöcyst. (21, 22) Rupture of mature oöcyst and release of sporozoites which pass to salivary glands of mosquito (23), and thence to human blood by inoculation. (*After Blanchard and Schilling, greatly modified.*)

modia was reported by Bass of New Orleans, using the upper layer of centrifugated defibrinated blood taken from the human host, a trifling amount of glucose being added.

Examination of the Blood for Malarial Organisms.—Fresh blood on a warm stage gives the most satisfactory results in these examinations, but it is quite possible to detect them in the dried and fixed specimens, stained as in ordinary blood examinations. If the old triple stain be used, or the better stain of Wright, already described elsewhere, the organism will be found to have taken the blue. *The common mistake born of inexperience lies in the failure to search primarily for pigment in both red and white blood cells, and in the readiness with which the unpigmented forms are confounded with artefacts.* In nearly all specimens taken at a proper time pigmented organisms may be found within the erythrocytes and pigment granules in the leucocytes. *In the tertian variety the pigmented forms appear from eight to sixteen hours after the preceding chill, and the large pigmented organism* is best obtained eight hours before the paroxysm. It is useless to hunt for tertian or quartan forms if quinin has been taken for twenty-four hours or more.

Fresh blood best.

Look for pigment.

Best time to search.

Stippling of the erythrocyte host may be visible in stained specimens (Giemsa's stain). This may take the form of brick red tiny dots in the tertian form.

In the estivo-autumnal infections, specimens deeply stained with Maurer's modification of the "Romanowsky" may show dark red-violet spots of irregular and variable form ("pernicious spots").

Immunity.—It would appear that natural complete immunity to the malarial organisms is very rare.

Relative immunity is not uncommon and is especially marked in the negroes of malarial countries.

Symptoms.—The *tertian and quartan forms* are manifested by typical paroxysms whatever the frequency of their occurrence, and the order of events is as follows: (*a*) *The cold stage:* after a rigor lasting from one-fourth to one and one-half hours and occurring usually in the mid-forenoon or early afternoon, the temperature, which rises with and during the chill, reaches a maximum (104 to 107°F.) and the cold stage is succeeded by (*b*) *the hot stage,* which presents all the symptoms of a sthenic fever which reaches its fastigium in an hour or two and declines to normal during the next six, eight or ten hours, its recession being marked by the coming of (*c*) *the sweating stage* during which all symptoms abate and the patient feels well. These attacks vary greatly in duration, those of the severe type lasting from eight to fourteen hours. The spleen enlarges if they are repeated on several days, and if they are long continued, tends to become permanently hypertrophied (ague cake). In spite of the marked evidences of infection there is a normal leucocyte count or more frequently a leukopenia. Herpes labialis is a common manifestation. Albuminuria may be present during the attacks or persist if they are frequently repeated, and the gastrointestinal disturbances accompanying the fever may be profound.

"Fever and ague."

Cold stage.

Hot stage.

Sweating stage.

"Ague cake."

Herpes and albuminuria.

The estivo-autumnal form, due to the organism of that name, may at

DESCRIPTION OF PLATE V

Quartan, 1, 2, 3, 4, 5, and 6, show the development from the hyaline form to the mature intracellular (6) and large extracellular (10) forms; 11 shows vacuolization of an extracellular form; 7, 8, 9, show segmentation stages; 12, the flagellate form (sexual cycle, microgametocyte).

NOTE.—In the tertian and estivo-autumnal organisms the same phases are shown.

NOTE.—

A. Relative depth of color in the erythrocyte host, deepest in the quartan, lightest in the tertian, often brassy with cell deformity in the estivo-autumnal (tropical form).

B. Comparatively coarse, scant, and dark granules of the quartan as compared with the tertian.

C. Tendency to shrinkage in the erythrocyte host of quartan *vs.* large pale host of tertian.

D. Peripheral arrangement of pigment in developmental stage of tertian and quartan followed by central grouping initiating segmentation. The pigment in the quartan may form an oblong band across the erythrocyte host.

E. Note greater regularity in quartan as compared with tertian forms.

F. Note relative number of segments.

G. Note greater density and clearer outline of quartan forms.

H. Star-like arrangement of pigment in early segmentation stage of quartan.

I. Relatively small flagellate forms of quartan and estivo-autumnal.

J. The peculiar ovals and crescents of the developed estivo-autumnal form, its scantily pigmented spherical form (35) and the ring bodies of the early stage.

PLATE V.

1, 13, 23.—Schizonts (youngest ring forms).
8, 20, 29.—Sporulation.
9, 21, 30.—Release of new merozoites.
25, 26.—Double infection by young schizonts.
32. Female gametocyte (macrogamete).
34. Male gametocyte (microgamete).

its onset be definitely periodic, usually like tertian but with longer paroxysms. Ordinarily, owing to the irregular development of the parasite crops, the fever tends to assume the remittent (bilious remittent) type, and in such cases the temperature curve may be lower than in the fastigium of the regular form. Jaundice is common, gastrointestinal symptoms and splenic enlargement marked. Under this head the most diverse types of malaria must be included, representing on the one hand the larval types, or dumb agues, often manifesting themselves by persistent headache, neuralgia, general malaise, dyspepsia, anemia, etc., and, at the other pole, the pernicious form of the disease, encountered especially in the tropical and subtropical countries. These assume various types, are often limited to a certain small area and are peculiarly fatal to the unacclimated.

"Dumb ague" sequelæ.

Choleraic and comatose forms.

Classification of Pernicious Forms.—(*a*) *Choleraic.* Symptoms, low or slightly elevated temperature, vomiting, choleraic diarrhea and stools, frequently a jaundice, cyanosis, feeble, rapid pulse, collapse and high mortality. (*b*) *Comatose form.* This is preceded usually by a typical malarial paroxysm which is succeeded by stupor, hyperpyrexia, Cheyne-Stokes breathing, and every symptom of collapse, stupor deepening into coma. (*c*) *The hematuric or hemoglobinuric* form. This is characterized by the predominance of renal symptoms, associated at times with bleeding from the mucous membranes and includes undoubtedly the so-called "black-water fever." It is very fatal.

Blackwater fever.

Usually estivo-autumnal.

Comment.—These pernicious forms are usually though not invariably associated with the estivo-autumnal organism; in the choleraic type masses of organisms may be found in thrombi of the intestinal vessels; in the cerebral or comatose form, in the vessels of the brain. The strong resemblance to yellow fever presented by these cases will at once be noted (see "Yellow Fever").

Malaria in Infants.—Fusco has found malaria in many infants living in malarious districts and states that in such cases the symptoms may be most misleading.

Fever may be brief, the chill absent, or replaced by convulsions, vomiting, mere pallor or cyanosis.

The spleen is almost invariably enlarged and in 85 per cent. of Fusco's cases the estivo-autumnal organism was present.

Chronic Malaria (*Malarial Cachexia*).—The chief characteristics of this form are the enlarged spleen, anemia, sallow skin, and persistent malaise. In many instances there is a tendency to chronic diarrhea, dysentery, or marked chronic gastrointestinal disturbance of a vague and indeterminate type. In some cases there are accessions of fever, perhaps actual paroxysms occurring irregularly, and persistent and repeated examinations will usually reveal the estivo-autumnal organism.

Misleading symptoms.

Complications.—Persistent neuralgias, often regularly or irregularly periodic, persistent anemia, gastrointestinal difficulties, and chronic nephritis are among the commoner complications and, more rarely, actual changes in the brain, spinal cord, or peripheral nerves may occur, producing paralysis, ataxia, or even symptoms of disseminated sclerosis.

Diagnosis.—The recognition of the regularly periodic form is not difficult, but in general no accurate diagnosis can be made without an examination of the blood, the two elements of importance being the plasmodium and the leucopenia. Clinically dependence must be placed oftentimes upon the effect produced by quinin given in large doses and in solution. The differential diagnosis as regards typhoid and yellow fever has been discussed under those diseases.

Blood examination and therapeutic test.

RELAPSING FEVER

(*"Famine Fever," "Seven-day Fever"*)

Definition.—An acute contagious disease due to the *spirocheta Obermeieri*, (*Spirocheta recurrentis*), and in different countries to *spirochetae* differing somewhat in their characteristics (*S. novyi*, *S. duttoni*, in African tick fever; *S. carteri* in that of India). It is characterized by recurrent febrile paroxysms, each being of from five to seven days' duration and associated with an acute onset and critical termination. An attack does not confer immunity.

Fig. 505.—Spirillum of relapsing fever (*spirocheta Obermeieri*). Motile, flagellated, spirillum readily stained but not cultivated and Gram-negative. The organism is usually longer than is indicated in the illustration.

Etiology.—The disease is one of filth and is highly contagious. The spirillum is unquestionably the cause of the disease but cannot be cultivated.

Morbid Anatomy.—There are no special lesions. Enlargement of the liver and spleen being common, together with catarrhal inflammation of the gastro-intestinal tract.

Symptoms.—*The symptomatology is summed up in the definition. It represents repeated attacks (usually two) of high fever of sudden onset terminating by crisis, alternating with afebrile periods.*

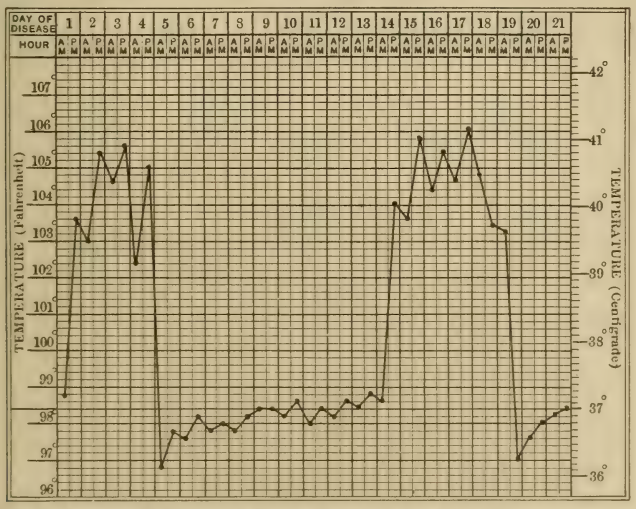

DAY OF DISEASE
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21
HOUR
A P A P A P A P A P A P A P A P A P A P A P A P A P A P A P A P A P A P A P A P A P A P A P
TEMPERATURE (Fahrenheit)
107 106 105 104 103 102 101 100 99 98 97 96
TEMPERATURE (Centigrade)
42° 41° 40° 39° 38° 37° 36°

Fig. 506.—Clinical chart of relapsing fever showing the febrile movement upon the fourteenth day.—(*From Wilcox's Fever Nursing.*)

DESCRIPTION OF PLATE VI

Malarial organism of types shown in Plate III but treated with Wright's stain.

QUARTAN PARASITE.

1. Non-pigmented form.

2, 3, 4. Young organisms showing chromatin bodies (red) and pigment granules.

5, 6. Full-grown parasites, the former intracellular, the latter extracellular.

7, 8. Presegmentation forms.

9. Segmentation.

11. Normal blood cell.

12. Flagellate body.

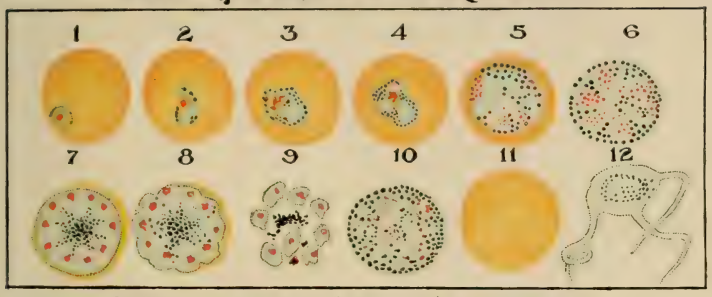
TERTIAN PARASITE.—The forms are self-explanatory in connection with the preceding description.

Estivo-autumnal Form. Various ring forms are shown together with the characteristic crescents, Nos. 32, 33, 34 (Micro- and Macrogametocytes).

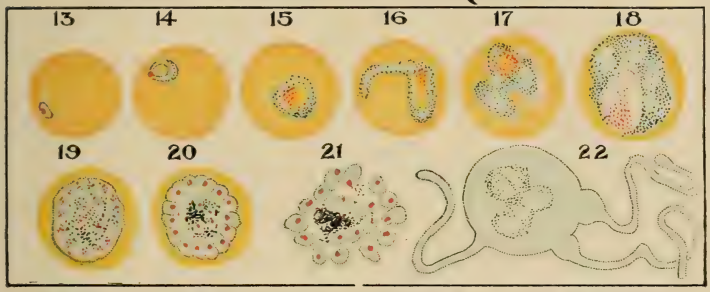
(In both this and the preceding plate the drawings are almost entirely based upon personal observation, though certain forms have been adapted from Thayer's valuable monograph.)

PLATE VI.

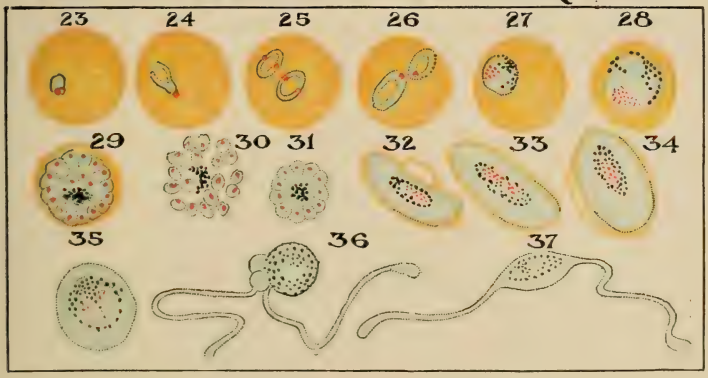
THE QUARTAN PARASITE



THE TERTIAN PARASITE



THE ÆSTIVO-AUTUMNAL PARASITE



The diagnosis is at once established in most, but not all cases by an examination of the blood which reveals the long spirilla which are in length about three times the breadth of the red cell. The usual complications of fever of this type may be present, *i.e.*, pneumonia, nephritis, hyperpyrexia, jaundice, ophthalmia, and renal, gastric, or intestinal hemorrhage, yet in general the disease is uncomplicated, the prognosis favorable, the mortality low (2 per cent.).

The inoculation of susceptible animals is occasionally required if the organism cannot be recovered either in the apyrexial or febrile period.

TYPHUS FEVER

(*"Spotted Fever," "Ship Fever," the "Black Assize," "Jail Fever"*)

Definition.—*An acute self-limited infection, the specific cause of which is probably a microörganism known as Rickettsia prowazeki, its intermediary host the body louse, highly infectious and characterized by its sudden onset with high and continuous fever, its termination by crisis on or about the fourteenth day, and its association with a petechial eruption, a mottled skin and a characteristic odor and facies.*

Probable cause.

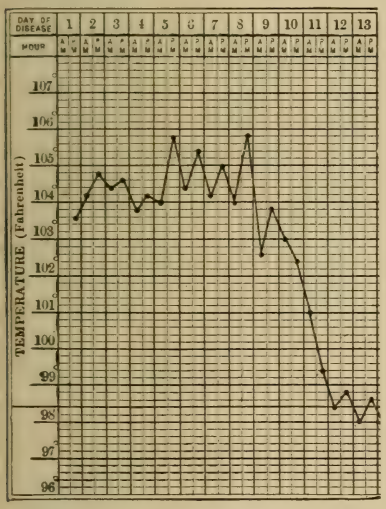


FIG. 507.—Clinical chart of typhus fever ending in recovery.—(*From Wilcox's Fever Nursing.*)

Etiology.—Ricketts described in 1910 the occurrence of certain bacterium-like microörganisms in body lice and his observations were confirmed later by Hegler and von Prowazek. Studies were made later by Rocha-Lima, who named the organism rickettsia prowazeki. The Commission to study typhus operating under the Red Cross Societies conducted a large amount of experimental work in 1920 which seems to have definitely established this organism as the causal factor of the disease although as yet it has been impossible to cultivate it.

In smear preparations the organism is ovoid, occurs in pairs, filaments, or as bipolar staining rods. The organism is Gram-negative and can be satisfactorily demonstrated only with the Romanowsky stains, of which Giemsa's modification is most satisfactory.

A Valuable Test.—Anderson and Goldburger have introduced a valuable test for atypical cases. A guinea pig is injected with the blood of the suspect by the peritoneal route and will show the typical fever curve if typhus is present.

Transmission.—Contrary to the belief formerly held up to the last few years, typhus fever is not readily, if at all, directly communicable from man

to man and practically all cases are now known to be dependent upon the bite of infected body lice.

The conditions under which it develops make it extremely difficult to avoid epidemic spread, but it is now known that in hospitals those actually attending typhus cases seldom acquire the disease unless they become infected with body lice from outside sources. On the other hand, those employees of the hospital who have to do with the reception and preparation of patients at the hospital run a very considerable risk of acquiring the disease.

Seasonal Occurrence.—The disease is distinctly seasonal as well as epidemic in type, being most prevalent during the later winter months, diminishing as the spring advances and reaching its minimum of incidence during the summer.

Historic Note.—This ancient disease, which owes its name to de Sauvages (1760) and was first described by Frascatorious of Verona in the sixteenth century, was confounded with typhoid fever until the early part of the nineteenth century, and even the clear distinction made by Louis did not bring about its separation from typhoid until nearly half the century had passed. Once a common and fatal disease,* it has now become one of the rarest and, save in the mild forms known as "Brill's disease," is limited to insanitary, semicivilized and densely populated countries or communities. Overcrowding, bad air, and dirt have always been its chief promoters. It is highly infectious and conveyed usually by the body louse (P. vestimenti).

A banished scourge.

Easily preventable.

The Great War brought about renewed interest in and extended study of typhus fever as a result of serious outbreaks in certain of the Eastern countries. Fortunately the Western war front was not affected at all. The first serious primary outbreak occurred in Serbia in 1915. At this time the disease still exists in Poland and Russia.

Morbid Anatomy.—There are no characteristic changes beyond those of an intense febrile infection.

Incubation Period.—The period of incubation varies from seven to fourteen days.

Immunity.—Absolute immunity over a period of many years is conferred by a single attack and individual immunity probably occurs. Artificial immunity has not been established as yet.

Violent onset.

Rapid prostration.

Eruption.

Symptoms.—*The symptoms characteristic of typhus are—a strikingly abrupt onset of fever, often moderate at first, tending to advance daily and assume a continuous type, at the outset accompanied by chilling or actual chills with marked nausea or actual vomiting, headache, backache, profound prostration, the rapid development of a typical "typhoid state," and the appearance of an eruption at the end of the fourth or fifth day.*

The eruption, at first like rose spots, soon becomes darker and fades to a brownish discoloration and is associated with a dim subcuticular mottling.

Crisis.

The termination is usually but not invariably by decided remission or a sharp crisis. Relapse is rare and the complications are those attending any profound febrile infection with marked toxemia.

* It is said to have killed 1,000,000 of the British people during the eighteenth century.

Variants.—As in so many other infectious diseases, many atypical cases occur often of a most misleading character. Both the fever and the exanthem may be lacking and many mild cases occur. Doubtless these facts lead to an exaggerated notion of the extent of natural immunity.

The stormy manifestations usually associated with its onset may be preceded by several days of fever and other prodromal symptoms.

Physiognomy and Odor.—*An odor often described as "mousy" or, by Ker, as that of "rotten straw" is quite constant even in well-kept patients, and the physiognomy is that of a "drunken rounder," the eyes being bleary and bloodshot, the face bloated and swollen.*

Diagnosis.—This must depend still upon the clinical symptoms inasmuch as cultural methods are too slow to be of use during the attack.

Prognosis.—The mortality may reach 50 per cent. in old people, and varies from 10 to 20 per cent. in children and young adults. The heart suffers profoundly during the height of the illness and cyanosis and pulmonary hypostasis are almost invariable.

Mild typhus.

BRILL'S DISEASE (*Attenuated Typhus*).—This is a form of sudden onset, profound prostration, severe general pain, violent headaches, and febrile movement, associated usually with an erythematous sometimes misleadingly, "rose-spot" like, maculopapular rash of wide distribution and variable profusion appearing on the fifth or sixth day, reaching its acme in twenty-four hours and persisting until the end of the illness which is attained either by crisis or rapid lysis in from twelve to fourteen days.

The rise of temperature is abrupt and it terminates in a rapid descent covering but 24 to 36 hours.

Anderson and Goldberger have apparently proven this to be a mild typhus of relatively slight transmissibility, identical with the typhus of Europe and Mexico.*

MALTA FEVER

(*Mediterranean Fever, Undulant Fever, "Rock Fever," Neapolitan Fever, Gibraltar Fever*).

Definition.—*An endemic tropical and subtropical septic infectious fever due to the "micrococcus melitensis" (Bruce)* and *characterized by long-continued undulatory pyrexial periods and repeated relapses.*

Etiology.—The micrococcus melitensis, discovered in 1887, is a small oval or round coccus (0.33μ), occurring singly or in pairs, staining readily though not by Gram's method, easily cultivated in *faintly acid* media at 37 to 38°C. It is of slow growth and shows an agglutination reaction similar to that of typhoid fever. The milk of the Maltese goat has been proven the chief agent in the spread of disease in Malta and Gibraltar and resulting preventive methods have enormously reduced the incidence of the disease in its home. It is essentially a disease of summer, respects no age, is uninfluenced by sex and confers prolonged immunity. The disease has been almost wiped out

* Dr. N. R. Brill has reported 225 cases with no mortality.

in the garrison at Malta and in the Mediterranean fleet by stopping the use of raw goats' milk. Nearly 15 per cent. of these, the most numerous of the domestic animals of Malta, were found to carry the germ in their milk.

The disease is also said to be endemic in certain regions of Texas where goats are extensively bred.

Morbid Anatomy.—The pathological changes merely reflect the toxemia and high fever; splenic tumefaction and congested lung bases are constant factors. There is moderate anemia, and often a relative leucocytosis, the spleen contains the specific germ in quantities and shows an increase of lymphoid tissue. It may be found also in liver, kidney, urine, blood and in the lymphatic and salivary glands.

History.—Unrecognized or undifferentiated, this disease has existed from time immemorial along the Mediterranean shore, and ancient writers are supposed to have referred to it under the head of malaria. It would seem to be widely distributed, cases having been reported from the Mediterranean Islands, Italy, Greece, Turkey, Arabia, Palestine, China, South America, the Philippines, East and West Indies, Central America, and even in this country in isolated instances.*

The period of incubation is about fourteen days.

Repeated relapses.

Symptoms.—The onset closely resembles typhoid, the fever running in waves of three weeks' duration, repeated once, twice, or many times with intervening periods of normal temperature. It may last for from two or three weeks to two years, its average duration being about three months. *Severe joint pain* and swelling is present at the onset in 50 per cent. of the cases and neuritis and orchitis are frequent complications. As a rule, the first relapse is more severe than the original attack, being associated with chills, high intermittent fever and a diarrhea which replaces an antecedent constipation.

The spleen may be enlarged moderately or attain large proportions. *Blood cultures* are obtainable from the second day to the fortieth week, during the maximum febrile periods.

Differential Diagnosis.—From *malaria* it is differentiated by the absence of the specific hemameba of that disease, from *typhoid* by the joint pain,

Specific test.

absence of rose spots, the failure of the agglutination test of Widal, from either by its own complete *agglutination test* in dilutions of at least 1–30 obtainable as early as the end of the first week. Aside from these procedures one must depend simply upon the differences in the general symptomatology, the temperature chart and the tendency to distinct relapses.

Low mortality.

Prognosis.—Oddly enough, the disease furnishes but 2 to 3 per cent. mortality, the loss of time, the discomfort and interruption of affairs being the chief inflictions.

* Bruce states that nearly all cases occur in Malta and that the annual incidence among the people averages 30 per 10,000 and in the garrison, formerly, 370 per 10,000.

ROCKY MOUNTAIN SPOTTED FEVER

The cause of this curious disease is unknown. It is prevalent in small districts of western Montana, Idaho, Wyoming, Utah, Oregon, Washington, California, South Dakota, Colorado and Nevada and particularly in a small area from 4 to 10 miles wide and 50 miles long on the western side of the Bitter Root River. It prevails only from the middle of March to the middle of July and seems to affect only those persons who have been bitten by the tick known as "*Dermacentor Andersoni.*" The presence and activity of this tick corresponds accurately to the time of disease prevalence. Dr. Chowning states that these were found in every case seen by him but might easily be overlooked because at times only present in the hair about the genitals.

Probable intermediate host

FIG. 508.—Rocky Mountain spotted fever.—Exanthem. (*After J. F. Anderson*).

The disease has been known to the physicians of Montana for over thirty years and was first reported formally by Maxey in 1899.

Long known

In 1902 in response to a request from the Montana State Board of Health, Drs. Chowning and L. B. Wilson were sent from the University of Minnesota to investigate the disease. They reported the finding of an intra-, and extra-corpuscular hyaline organism to which they gave the name of *piroplasma hominis.*

In 1906 King reported *Dermacentor venustus* (*Andersoni*) to be the carrier and transmitter and Ricketts succeeded in conveying the infection to a monkey by the bite of this tick.

The finding of *piroplasma hominis* by Wilson and Chowning has failed of confirmation. Wolbach reports an uncultivable spirochete.

Causal Agent.—*Dermacentroxenus rickettsi* is a minute, intracellular, bacterium-like microörganism which does not stain by Gram's method and is best demonstrated with the Giemsa stain. It appears usually as lanceolates in pairs, about one micron in length. The organism is non-filtrable, is readily killed by most disinfectants as well as by heat and drying. It is uncultivable.

Symptoms.—After an incubation period of from three to ten days, headache, nausea, muscular soreness, a chill or chilliness, and nose-bleed, are followed by a rapidly rising fever and an accelerated pulse and respiration.

Rash

The rash appears from the third to the seventh day, first on the forehead and extremities, later on the chest, abdomen, and back. It is macular, bright red, and becomes petechial in severe cases. It is profuse everywhere except on the abdomen and desquamation may follow. The exanthem appears first on the ankles, wrists and back, the abdomen being least affected. Later

Profound asthenia.

the visible mucous membranes may be involved and even the palms of the hands and soles of the feet.

With the establishment of the hemorrhagic character of the rash, areas of confluence may occur and necrotic spots may develop in the dependent areas. Desquamation follows but usually is not extensive.

The character of the exanthem and the tendency to necrosis directly corresponds to the changes found at autopsy in the peripheral blood vessels. In fatal cases rapid anemia appears with sustained high temperature and a rapid weak pulse. Albuminuria is constant. The spleen is enlarged.

Differential Diagnosis.—No confusion can arise if the salient points are recalled. The history of exposure, the tick bites, the discovery of the tick itself, the sporadic nature of the disease, its distribution, the character and time of appearance of the rash, are sufficiently distinctive.

Prognosis.—In favorable cases termination by lysis is initiated by the twelfth day. The mortality averages 70 per cent. in Montana and pernicious cases of rapidly fatal course are seen but it is said to be very low in other states (1 to 3 per cent.).* One attack seems to confer lifelong immunity.

Contrast in mortality.

TULAREMIA.—The relatively rare occurrence of this peculiar and relatively severe and disabling febrile infection has been recognized for several years within a very circumscribed area in Utah, during the months of June, July and August, under the name of "deer-fly fever."

Its etiology appears to have been established by Dr. Edward Francis of the United States Public Health Service who, in 1919, established the *Bacterium tularense* in cases investigated in the field, repeating his observations on six cases in 1920.

Dr. Francis asserts that in Utah the disease is conveyed from infected jack-rabbits to man by the blood-sucking fly *Chrysops discalis*. Cases of tularemia have been observed also in ground squirrels in Utah and California and the cotton-tail rabbit in Indiana.

Symptoms and Course.—Following the bite of a deer-fly carrier the adjacent *lymphatic glands* become swollen and tender and on the third day the site of infection presents a black necrosing centre which sloughs and leaves a "punched out" ulcer, the healing of which leaves a characteristic permanent scar. The glands usually suppurate and demand incision and drainage.

The *fever* is of the intermittent type and seldom exceeds 102° F. as a daily maximum.

Chills may occur even in the early prodromal period and may be severe during the active stage of the disease.

Profound *prostration* is present during the active stage which endures about four weeks. Recovery of strength is seldom attained until one or two months thereafter.

Prognosis.—Death occasionally results but the mortality is light and apparently recovery after a tedious convalescence is the rule.

* The Bitter Root Valley cases furnish mortality figures of 90 per cent. In Idaho the death rate is only 3 per cent.

MILK FEVER (*Milk Sickness*).—This is a rare and frequently fatal disease supposedly caused by the *bacillus lactomorbi* of Johnson and Harris, limited to the southern and western part of the United States, and apparently results from the ingestion of the uncooked flesh or milk of cattle suffering from "trembles."

Symptoms.—A short period of malaise is followed by vomiting and epigastric pain, constipation and *muscular tremors*, with little or no fever. The symptoms closely resemble ordinary ptomain poisoning, and there may be active or typhoidal delirium, coma, or convulsions.

MOUNTAIN FEVER.—There is no single disease to which this term may be properly applied. Most of the cases so denominated by the earlier writers were unquestionably unusual forms of typhoid fever.

EPIDEMIC DROPSY.—Definition.—An extraordinary epidemic "deficiency" disease of the Orient, of unknown origin, characterized by the abrupt appearance of dropsy of the cardiac type, usually preceded by fever, vomiting, and diarrhea, and often accompanied by facial erythema and a rubeolar exanthem on the limbs and trunk. It occurs in individuals whose diet is largely composed of "dal" and polished rice.

Whether this disease is distinct from beri-beri is not yet decided. Nervous symptoms such as prickling, burning, or itching often precede the occurrence of the dropsical condition, but, according to Manson, the disease in the epidemics is pure and unmixed with cases of frank beri-beri, a fact strongly adverse to the opinion held by many, that epidemic dropsy is neither more nor less than beri-beri with predominance of dropsy and slight nerve symptoms.

Extreme cardiac weakness is frankly displayed in all cases.

The mortality varies from 2 to 40 per cent. but fortunately the epidemics are rare, widely separated chronologically and limited in distribution.

EPIDEMIC GANGRENOUS RECTITIS.—This terrible ailment attacks children and the lower animals chiefly, and its cause is unknown.

Rectal prolapse occurs and the clinical picture is one of gangrenous dysentery.

It occurs only in Central America, Northern South America and some of the islands of the South Pacific, so far as is known at present.

MILIARY FEVER (*Sweating Sickness*).—In ancient times this disease was extremely prevalent and fatal throughout all Europe, but is now seen only in limited districts of Italy, France, Austria, Asia Minor, Germany, and Switzerland.

With or without prodromata, the onset is marked by drenching sweats, persistent and recurrent, with from seven to ten days of high fever, attacks of paroxysmal tachycardia, subjective laryngeal and epigastric constriction, and on the third day an eruption consisting of erythema, miliary red papules, and tiny vesicles. Chantmesse believes that field mice carry the mysterious contagium, fleas serving as intermediary hosts.

FOOT-AND-MOUTH DISEASE.—*This is a severe epidemic form of febrile aphthous stomatitis affecting the buccal mucous membrane and even the pharynx.*

It is due to the fitrable virus of Loeffler and Frosch, introduced usually by eating the flesh or uncooked milk of infected cattle and is extremely rare, but shows a considerable mortality ratio (10 per cent.).

Children are attacked chiefly and the hands and feet may be affected.

FLOOD FEVER (*Japanese River Fever*).—This is a rare disease limited to a small area of, and afflicting only workers on inundated land, among whom it spares neither age nor sex. Its exact cause is unknown and it has no specific morbid anatomy.

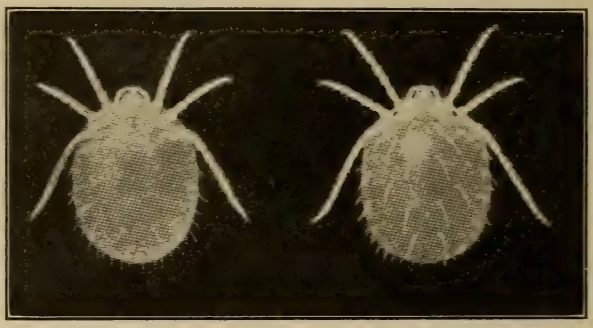


FIG. 509.—Japanese Flood (or River) Fever. ("Tsutsugamushi.") The "Kedani mite" (*Trombidium akamushi*), the carrier of this disease. "A minute orange-red arachnoid," common in the ears of field mice and quite harmless save in the particular inundated district affected. (*Stitt.*) (From "Endemic Diseases of Japan," Imperial Institute for Infectious Diseases, 1911).

It is transmitted only by the bite of a minute, orange-red larval mite, *Trombidium akamushi*.

Symptoms.—Ulcers in the axilla, groin, or neck are associated with repeated chills, continued high temperature, moderate lymphatic involvement and profound toxemia. A coarse papular eruption appears on the sixth day and disappears promptly after several days.

Prognosis.—The duration of the disease is from two to three weeks and the mortality varies greatly, running from 10 to 80 per cent. in different epidemics.

GLANDULAR FEVER (*Contagious Cervical Adenitis*).—An acute cervical adenitis involving especially the glands lying behind the upper portions of the sternocleidomastoid muscle is occasionally encountered in children, chiefly between the ages of one and ten.

The disease is mild but *contagious*, comes on suddenly with fever, sore throat, and decided constipation or diarrhea.

The glands are hard and tender, but seldom fuse or suppurate. Occasionally relapses occur and other groups of glands may become involved.

Enlargement of the liver and spleen is sometimes observed and acute nephritis is not uncommon. The febrile period is brief and the glandular enlargement is of but two or three weeks' duration.

PAPPATACI FEVER (*Phlebotomus Fever*).—**Definition.**—An epidemic three-day fever caused by the filtrable virus of Doerr and Russ, prevailing chiefly along the shores of the Adriatic, transmitted by the bite of a gnat or

moth-midge (*phlebotomus pappataci*) and resembling clinically abortive typhoid fever, or, oftener, an irregular dengue.

The virus may be recovered from the blood during the first twenty-four hours only.

Bradycardia constitutes one of the most important points in differentiation and neither liver nor spleen are enlarged.

It is probably identical with the East Indian "Sand-fly Fever."

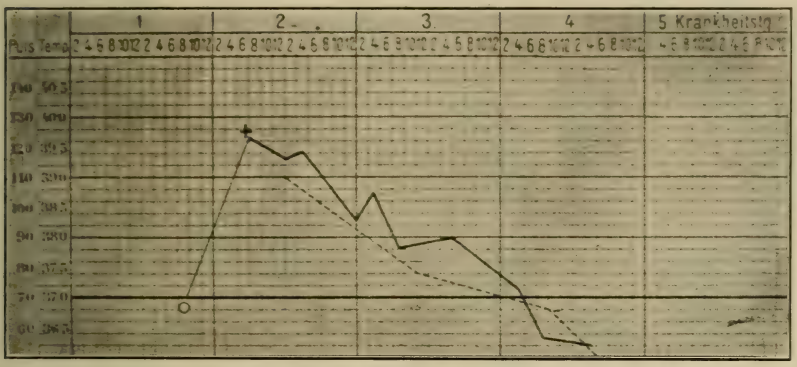


FIG. 510.—Pappataci fever. Fever chart. (*After Franz Doerr and Taussig.*)

SIX-DAY FEVER.—This dengue-like fever is differentiated from that disease chiefly by its continuity and the accompanying splenic enlargement.

Deeks reported cases from the Panama Canal Zone.

SEVEN-DAY FEVER.—This, reported by Rogers, is probably an irregular dengue of the type occasionally, or often, encountered in the Philippines, in which the febrile intermission is so slight or insignificant as scarcely to interrupt the febrile period (Stitt).

LEISHMANIASIS

(*"Kala Azar," "Tropical Splenomegaly," "Dumdum Fever," "Black Fever"*)

Historical Note.—This curiously obscure and deadly disease has for some years prevailed in a limited district of Assam, having commenced its onward march at Rungpore in 1870.* It extends along lines of communication at the slow rate of 14 miles a year, its visitation lasting for five years and the natives believe that one year is required to render safe any house in which it has appeared. It spares no age and neither sex, and, what is more peculiar, attacks the acclimated quite as readily and violently as the stranger. Its mortality is frightful, being put at 90 per cent. by Manson, and its victims may linger on for a year or more. It seldom attacks Europeans.

* The first case reported is said by V. Schilling to have occurred at Dum-dum near Calcutta. In 1869 British administrative officers in Assam had to deal with a severe epidemic in the Garo Hills. It was for many years regarded as a peculiarly severe and obscure form of malarial fever.

Etiology.—Peculiar oat-shaped, circular or oval protozoan bodies (*"the "Leishman-Donovan bodies"*) 2 to 4μ in size, and first discovered by Leishman and Donovan in 1903, are found in most cases if the spleen or liver be punctured, or they may appear in the leucocytes of circulating blood, or, as

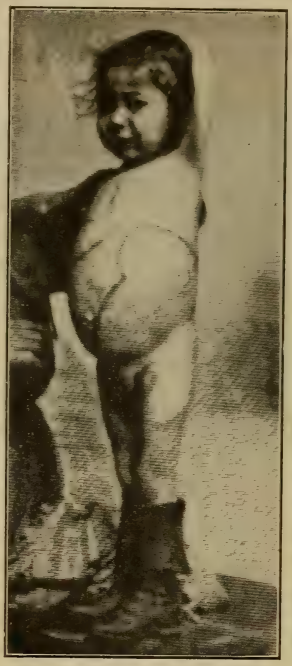


FIG. 511. FIG. 512. FIG. 513.

FIGS. 511, 512, 513.—Liver and spleen in child suffering from leishmaniasis. (*After Nicolle.*)

apparently free bodies, if the leucocyte host be damaged in making the smear preparation. On appropriate culture media they develop flagellæ.

In the blood they are most frequently carried by the large mononuclear leucocytes. They are abundant in the spleen, liver and bone marrow.

The readiest and most certain method of recovering the specific organism is by smears of spleen juice obtained by the somewhat hazardous splenic-puncture.

The mode of conveyance of Kala azar is not yet established but it is probable that in India bedbugs are the carriers and that along the Mediterranean, fleas may transmit the disease.

Symptoms.—The high fever, often preceded by vomiting or chill, may assume either a remittent or intermittent type, lasting one, two, four, or six weeks; then follows an apyrexial period, then an exacerbation, and so on for months, when the fever becomes persistent, though remittent, and profuse sweats characterize the remissions. Emaciation is extreme, the liver and spleen enlarge early, profound anemia supervenes, and a dirty gray pallor and brittle dry or falling hair add to the striking clinical picture.

One of the most characteristic features of the disease is the double febrile paroxysm in every twenty-four hours.

If the organism is present in the blood smears the diagnosis is simple and direct. This failing, puncture of the liver is much safer than spleen puncture.

In either case a preliminary coagulation test of the patient's blood is demanded, inasmuch as fatal hemorrhage has resulted in some instances.

One suggestive symptom is the decided leucopenia, which chiefly affects the polymorphnuclear cells.

The ratio of white to red cells may be but 1–2000 or 1–4000. The normal ratio is about 1–600.

Termination.—The patient dies ordinarily from an intercurrent pneumonia, dysentery, or exhaustion within twelve to eighteen months.

Under modern treatment about 30 per cent. may recover.

FIG. 514.—"Tropical sore." (*After Cardamatis and Melissides.*)

INFANTILE KALA-AZAR is another form of Leishmaniasis due to infection with the form known as *l. infantum.*

It attacks infants exclusively and has long been known as an infantile splenic anemia along the shores of the Mediterranean.

TROPICAL SORE (*"Leishmaniasis Cutanea," Aleppo Boil, Nile Sore, etc.*).—This disease, also a Leishmaniasis, is caused by *l. furunculosus sive tropica* and affects exposed parts of the body almost exclusively.

It begins usually as an itching, red papule on the site of some insect bite or slight wound or abrasion and during weeks or months gradually increases in size and assumes the appearance of a sluggish furuncle.

This stage is succeeded by one of persistent, painless, sluggish ulceration.

The boil may be solitary or there may be a score or even more. Healing usually takes place only after the lapse of six to eight months.

This condition masquerades under a host of names, in addition to those given above, *e.g.*, "Oriental sore," "Bagdad sore," "Bouton d'Orient," "Uta" or "Espundia" (Peru), Bouba (Brazil).

RAT BITE FEVER.—This curious infection is believed to be caused by the *Streptothrix S. muris ratti.*

Nature of the Ailment.—Following rat bite, in a certain proportion of cases local inflammation of unusual severity is associated with enlargement of the tributary glands, chills, high fever, and pronounced toxemia.

The initial attack terminates by crisis on or about the fourth day, only to be renewed in most instances after the lapse of a few days.

Not infrequently multiple relapses occur.

The mortality is about 10 per cent. (Hata) and death occurs in delirium or coma.

It is chiefly a disease of Japan and China, but recent reports would indicate a wide distribution, extending in fact all over the world.

A somewhat morbilliform purplish rash may accompany the relapses.

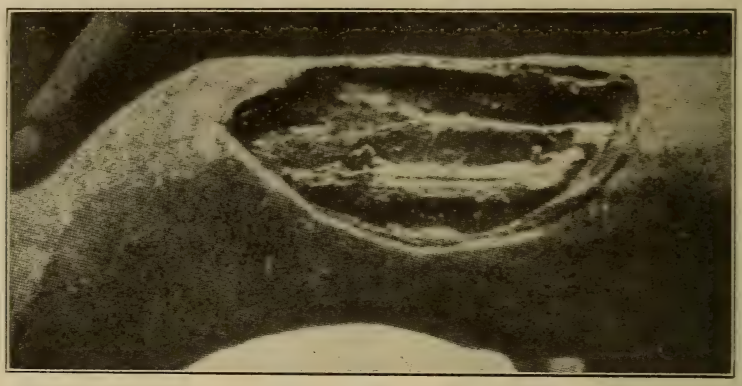


FIG. 515.—Tropical sore. (*After Brault.*)

VERRUCA PERUANA.—This disease was formerly regarded as a part of the ailment now described as "Oroya fever" but has been proven to be a distinct yaws-like disease (infectious granuloma) prevalent in the same districts of Peru.

Unlike yaws it is found to affect the viscera to some degree.

OROYA FEVER.—Definition.—This extraordinary acute, infectious, febrile, hemolytic, disease is characterized by a period of malaise, followed by irregular remittent fever, excruciating bone and joint pain and tenderness and an incredibly rapid development of an anemia of the extreme pernicious type.

Remarkable Hemolysis.—Within a period of four or five days the red blood cells may drop from 5,000,000 to 500,000, and the blood picture is that of Addisonian anemia, especially in the fulminant cases.

To Strong and his colleagues of the Harvard Expedition we owe the separation of this disease from *Verruca Peruana*, with which it was formerly confounded.

Limited range.

The two ailments occur exclusively in certain narrow hot Peruvian valleys lying from 3000 to 9000 feet above sea level.

Possible cause.

Strong confirmed the report of Barton (1909) with respect to the presence of rod-like bodies in the red cells, especially at the period of febrile onset. These he regards as intermediate between bacteria and protozoa and proposes the name *Bartonia bacilliformis*.

The mortality of this disease is frightful, death occurring within two or three weeks in from 20 to 40 per cent. of the cases.

The disease is wholly resistant to treatment so far as is known at the present time.

HILL DIARRHEA (*"Simla Trot"*).—**Definition.**—A morning diarrhea of unknown origin, specially affecting Europeans visiting the higher altitudes after a residence in damp lowlands.

It is widely distributed, not confined to India as was supposed formerly, seems to be especially frequent during the rainy season in the tropics and may assume an epidemic form. Its resemblance to sprue, with which it was long confounded, seems to lie wholly in the character of the stools, which

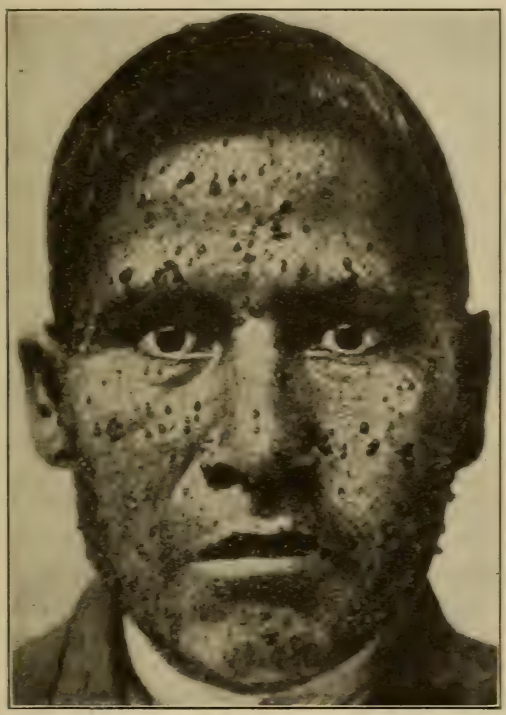


FIG. 516.—Verruca. Miliary form. (*After Odriozola.*)

are pale, even white, very copious, and frothy, together with the associated flatulent dyspepsia which the two ailments possess in common.

The diarrhea may be very active and urgent between the hours of 4 and 11 a.m., after which time the patient is free for the remainder of the day.

The ailment may or may not subside spontaneously if high altitude residence is maintained, but disappears at once upon the return of the victim to the lowlands (Manson).

SPRUE (*"Psilosis," "Ceylon Sore Mouth," "Cochin China Diarrhea"*).—**Definition.**—A tropical disease caused by *Monilia psilosis* (Ashford) characterized by an afebrile, but excessively *painful* stomatitis of the recurrent ulcerative type, vague digestive disturbances, chronic diarrhea, decided secondary anemia, and atrophy of the liver and the intestinal mucosa.

Afebrile chronic stomatitis.

The Causative Organism.—Monilia psilosis is thus described by Ashford: "Monilia psilosis appears in fresh glucose bouillon cultures as a large, clear-

cut yeast with a large, brilliant nucleus. While the greatest variety of size and shape is seen in this protean organism, it may be said that it has few granules and tends to be round and from four to seven micra in diameter. In time in all, and especially in unfavorable media such as lactose, hyphæ develop. Budding takes place from the side of the article, without sterigmata, as well as from the end of the terminal article, a succession of buds forming a chain of yeasts and giving rise to the name of the genus, Monilia. The best differential medium for primary isolation is Sabouraud agar, 4 per cent. glucose, plus two acidity."

Distribution.—It chiefly attacks Europeans dwelling in the tropics and is especially frequent in Java, Cochin China, Ceylon and Porto Rico.

In the country last named it is on the increase, is much dreaded by American residents and among them is even more fatal than tuberculosis. A few cases have been reported in the United States (Ashford).

Symptoms.—*Stomatitis.*—The disease begins with buccal hyperesthesia which is followed after a considerable period by inflammatory redness and an extremely painful recurring superficial, disseminated ulcerative process along the sides, frenum and margins of the tongue and, later, on the buccal mucosa. Very characteristic ulcers appear opposite the posterior molars (Crombie's ulcers), but the tongue remains clean and late in the disease becomes smooth, glazed, centrally "cracked" and fissured, though red and oftentimes showing punctate or dotted ulceration along its anterior surface and margins.

A distressing condition.

Salivation is usually profuse and troublesome during the active periods.

Dyspepsia.—This is distressing, yet indeterminate in type, flatulence being decided or extreme, and an apparently causeless nausea and vomiting an occasional occurrence.

Diarrhea.—This, in its typical form is chronic, at first matutinal, represented by only one or two stools daily, but extremely exhausting and debilitating. Accessions of the diarrhea follow closely the exacerbations and recurrences of the stomatitis.

Curiously characteristic.

Stools.—The stools are characteristically and extraordinarily copious, pale, pasty, acid, "fermenting," "*foamy*" "mawkish" and "evil smelling." No pain attends their discharge.

Early in the disease attacks of evanescent acute diarrhea may occur and the stools are dark and bilious.

Many cases of an irregular type are seen and in these for a time at least, either the buccal, gastric, or intestinal symptoms may dominate or wholly fill the clinical picture. The complement fixation test is positive in this disease.

Progressive inanition.

Course and Termination.—Very gradually increasing anemia, debility, and emaciation terminates in chronic invalidism excessive exhausting diarrhea, sallow, earthy pallor, or decided darkening of the skin appears, the patient becomes bedfast, gravity edema is present, and he dies of exhaustion or intercurrent disease.

According to P. Manson, the outlook for recent cases is good under absolute rest and the milk cure. For ambulant cases such a diet is impracticable.

TRENCH FEVER.—This ailment of trench warfare which appears at present to be a definite clinical entity is separable into two clinical types, viz.: 1. A brief abortive form in which the primary febrile period terminates usually within the week and is followed after an afebrile stage of a few days duration by a short relapse. 2. A form characterized by a more prolonged primary febrile period and multiple, periodic relapses. Two forms.

Aside from the fever, the chief clinical symptom is pain in the back and legs, and a remarkably and decided punctate basophilia.*

Although a very large amount of work has been done with reference to the etiology of trench fever, its specific cause still remains in doubt and its diagnosis yet lacks the clarifying element of a pathognomonic sign or symptom or a clean-cut syndrome.

Trench fever appears to be transmitted usually by the body louse, *pediculus corporis*, and it is not improbable that the cause may prove to be a species of *rickettsia*.

The *incubation period* seems to vary between ten and thirty days and during this period prodromal symptoms of the type usually encountered in infectious diseases may be present.

Apparently trench fever is a specific form of septicemia giving rise to a wide diversity of symptoms leading to frequent errors in diagnosis.

A sudden onset is almost invariably present accompanied by vertigo, headache, pain at the back of the eyeballs, injection of the conjunctivæ, and enlargement of the spleen. When the eyes are turned sharply to the right or left, nystagmus usually occurs and in a large proportion of the cases small erythematous spots appear over the chest, abdomen and back. These resemble in appearance and behavior the typhoid fever exanthem and are transient, enduring only a few hours.

The urine usually contains a trace of albumin, but true nephritis is not observed.

The blood shows no characteristic changes. A leucocytosis may be present but there is no constancy in its appearance.

Comment.—This extraordinary ailment was of very great importance during the Great War on account of the number of cases occurring, though these were for the greater part mild and of short duration. It has been said that from one-fifth to one-third of all cases of illness in the British armies and about one-fifth of those in the German and Austrian armies might be classified as trench fever. The American troops in France were but slightly affected because of the fact that at the time of their appearance at the front, the mode of transmission of this disease was understood and every effort made to eradicate the body lice which proved the responsible agents.

Inasmuch as the disease is readily transmitted by the whole blood but not by the plasma or by filtered blood containing its corpuscles in solution, it would appear that the virus is usually, but not invariably, filtrable.

* See paper of J. W. McKee, A. Renshau and E. H. Brunt, Brit. Med. Jour., Feb. 12, 1916, pp. 225–260.

MEASLES

(*Morbilli*)

Definition.—An acute, highly contagious febrile exanthem of childhood of unknown causation, conferring immunity, and characterized by marked catarrhal symptoms of the respiratory tract and a peculiar eruption.

Etiology.—No organism has been proven as the causative agent. Hektoen, Anderson and Goldberger have proven that the filtered blood serum produces typical symptoms when injected into the monkey (Rheous) and indeed its inoculability has long been known. Continued prevalence seems to confer a partial racial immunity, as the disease is terribly fatal when introduced into countries previously exempt.

Extremely contagious.

Contagiousness.—*Practically every exposure of children under ten results in an attack, and, as it is contagious from its earliest appearance even before Koplik's spots appear, spreads by proximity, contact, and fomites and ceases to be contagious only with established convalescence, few children escape infection.*

The production of *artificial immunity* is being given much attention at the present time and there is reason to believe that definite results may be obtained.

Mortality,

The *mortality* is terrific among the poorer tenement classes and in badly nourished infants and young children, but in private practice it should not exceed 3 per cent. among well-nourished youngsters. The chief mortality falls upon the first half decade of life and especially the period lying between

Effect of age.

the sixth and the twenty-fourth month. Children over five months old and under twelve years of age are remarkably susceptible, and the winter and spring are its chief seasons. Adults suffer very severe attacks but these usually lack the more serious and fatal complications.

Comment.—Our experience during the period of mobilization for the Great War demonstrated the very great importance of measles as an army disease and this ailment was especially active following the entrance into the camps of the first draft during the fall of 1917. In this earlier epidemic pulmonary complications were astonishingly frequent and this was especially true of empyema.

Morbid Anatomy.—Merely that of the complications and a catarrhal inflammation of the respiratory tract.

Significant signs.

SYMPTOMS.—*The presence of a marked* **coryza, injected conjunctivæ, photophobia,** *and indications of slight fever may be the only signs noted at the onset and by their intensity the first,* **second** *and third always suggest the true cause in the case of children. Chill or decided chilliness is not common.*

Important fever-curve.

Fever.—The fever rises steadily, reaching its maximum (103 to 105°F.) with the *appearance of the exanthem* (*fourth day*). Its subsidence begins as the rash commences to fade, and the normal is attained, either by crisis or through a lysis occupying from two to four days.

Earliest positive sign.

The Rash.—*Koplik's spots* appear early in 90 per cent., and the mucous membrane shows the eruption before the skin is affected.

PLATE VII.

MEASLES. While the rash is not very obvious on the face, the expression and eyes of the patient are fairly characteristic. (*From "Infectious Diseases," C. B. Ker.*)

These may be present from two to four days before the appearance of the exanthem and appear first opposite the molar teeth as pearly gray (not white or yellow) *spots, smaller than the head of a common pin, each lying usually but not always within a red aureola.*

Later they may extend to the inner surface of the lips, occur more widely distributed on the surface of the buccal mucous membrane and even on the soft palate. When present in large numbers, the whole surface involved is reddened and the discrete appearance of the spots is lost. They have then become a multitude of gray dots on a red congested background. *They disappear as the body rash develops.*

Character and distribution.

The appearance of the true spots should be carefully studied inasmuch as somewhat similar lesions have been reported in other contagious diseases, and furthermore because of their great importance in *early* diagnosis.

At the end of the third or during the fourth day, and at the height of the fever, the general rash becomes manifest as a dusky mottling, becoming more distinctly red though still dusky, and, at the end of twenty-four hours (fifth day usually) palpably papular and "shotty."

Usually it appears first at the margins of the hair and in the region of the ears and the forehead and gradually spreads over the forehead, neck, face, chest, trunk, and lower extremities, often assuming a crescentic arrangement and reaching its acme and fading in its location before reaching its maximum in the later ones. Hence cases are likely to present rashes of varying stages of development in the different portions of the body. Desquamation, fine and branny, follows and lasts for about one week.

Desquamation.

Duration of Stages.—*Period of incubation, one to two and one-half weeks. Period of invasion, three to four days. Period of developed rash, one week. Period of desquamation, one week.*

Important data.

Complications.—Broncho-pneumonia is the most common and serious one, causing death in 30 to 40 per cent. of such cases in certain of the public services, and even in private practice furnishing a heavy mortality. The disregard of this disease manifested by the laity is deadly in its effect, as children are exposed to pneumonia and other complications from sheer ignorance of its high mortality. Broncho-pneumonia is a deadly complication in sucklings and especially so during the year succeeding the first six months of life. Severe conjunctivitis and keratitis occur especially often in those poorly nourished and badly cared for, and the same may be said of severe gastro-intestinal disturbance, stomatitis or even gangrenous processes. The eyes may remain sensitive for a long period. Pertussis is frequently associated with measles, tuberculosis is a not infrequent sequel, and the milder degrees of otitis media and affections of the nasal structures are not rare.

An underrated ailment.

Sucklings.

The eyes.

The Urine.—Transient albuminuria is not uncommon, and Ehrlich's diazo-reaction is invariably present even in high dilution (1:100).

Diazo-reaction.

Blood.—Leukopenia or a normal count is the rule during the period of active infection, a definite and sometimes a decided leucocytosis (30,000+) being present during the stage of incubation.

Diagnosis.—*The combination of a febrile period of three full days associated with marked coryza, Koplik's spots and the peculiar eruption serve to distinguish the disease from drug and serum rashes and rubella.*

Look out for small pox.

The temporary measles-like rash of the preliminary stage of smallpox must always be borne in mind. Great variations in the disease exist; it may be trivial or virulent, febrile or afebrile, *with a very slight or absent exanthem.** The eruption may be hemorrhagic (black measles) and cases may, from the start, show predominance of nervous or pulmonary symptoms, and the lungs should be examined carefully at every visit. *A case of persistent measle-like rash in the adult may prove a syphilis.*

Syphilis.

MUMPS

(*Parotitis, Epidemic Parotitis*)

Definition.—*An acute epidemic, infectious, immunity conferring, highly contagious but usually trivial disease of childhood, of unknown causation, but due probably to the action of a filtrable virus, characterized by inflammation of the parotid gland and mild systemic symptoms.*

Highly contagious.

Etiology.—Children under four and adults are but slightly susceptible. The disease is transmissible in any stage by contact, contiguity, or, possibly, to some slight degree, by fomites, but still lacks a specific causative agent.

Filtrable virus.

Martha Wallaston has described a filtrable virus obtained from the sputum, of great potency when passed through a series of susceptible animals, reproducing in them all the leading clinical and pathologic changes of the disease.†

Incubation Period.—Forty-eight hours to three weeks. *Duration.* Active symptoms about one week, followed by gradual subsidence.

Morbid Anatomy.—An acute parenchymatous inflammation of the parotid gland.

Strikingly objective.

Symptoms.—*Pain and swelling in the parotid region, in front of or below the ear, usually bilateral, but often single.*‡ *Fever usually mild* (102°F.) *and of short duration. Dry mouth and intolerance of acids.*

The appearance of pain and swelling may be preceded by tenderness just posterior to the angle of the jaw, and a peculiar mottling of the skin of the chest may also be an antecedent symptom lasting for several days but preceding the parotid swelling by only twenty-four or thirty-six hours.

The involvement of the surrounding tissues may be so great as to wholly disfigure the victim.

* A hot wet pack or full bath is sometimes necessary to develop the rash.

† E. C. Rosenau (1915) reports that he obtained, by catheterization of the stenonian duct, pure cultures of streptococci which, when injected into dogs intravenously, produced with absolute constancy a parotitis and also pancreatitis or appendicitis in some instances. This streptococcus strain is apparently identical with that reported by Isabella Herb in 1909. These findings do not appear to have been confirmed.

‡ Cases are occasionally encountered in which the submaxillary gland is primarily or alone affected.

PLATE VIII.

A scarlet fever rash showing blotching and papulation on the forearm, and presenting a somewhat measly appearance. The color (especially on the fingers), however, is more that of scarlet fever. (*From "Infectious Diseases," C. B. Ker.*)

PLATE IX.

Scarlet Fever. Note the pallor round the mouth and the fine punctation of the rash. The neck is still bright scarlet, but the chest rash is faded and there is an intense abdominal efflorescence. The deep color about the axillæ is noticeable and the face is somewhat more involved than is usual. (*From "Infectious Diseases," C. B. Ker.*)

The cant of the head to the affected side, or its erect rigidity in bilateral involvement together with the distressed shapeless countenance makes a "figure of fun" and is likely to excite laughter rather than sympathy. The ailment is actually extremely painful and harassing.

Complications.—In the comparatively rare cases in adults unilateral or rarely bilateral orchitis is a frequent complication (20 to 30 per cent.), though rare in children. Suppuration of the gland by infection through Stenon's duct is a rare development. Rarely, in the female, ovarian congestion or inflammation or painful swelling of the breasts may be found. Recently several observers have reported a metastatic pancreatitis as occurring with some frequency in certain epidemics. *The duration is about one week.*

Orchitis.

Ovaritis.

Mastitis.

Orchitis is followed by atrophy in 40 to 50 per cent. of cases presenting this complication.

Testicular atrophy.

Inasmuch as this may mean sterility in bilateral cases, the patient should be kept absolutely in bed until full subsidence of the infection is attained.

SCARLET FEVER

(*Scarlatina*)

Definition.—*An acute infectious and highly contagious fever of children, of unknown causation, conferring immunity and characterized by a sudden onset, the prompt appearance (second day) of a punctate bright scarlet exanthem terminating in desquamation, and showing a marked tendency to inflammatory involvement of the kidney, middle ear, throat, and cervical glands.*

Etiology.—The disease is conveyed chiefly by *direct contact and fomites*, less often by proximity, the secretions being, according to late investigations, more potent than the desquamated scales. In hospitals severe and complicated cases should be kept away from the mild and uncomplicated.

The incubation period is *relatively short, seldom or never exceeding seven days* (*Ker*).

It attacks chiefly children over one and under sixteen years, but adults enjoy only a relative immunity until the age of twenty-five is passed. It is rare in persons over forty years of age (eight of 1400 cases reported by Rolly from the Leipzig Clinic).

Mallory described protozoan bodies (*Cyclosterion scarlatinæ*) apparently characteristic of, and peculiar to, the disease, but no causative organism is proven. The "leucocytic inclusions" of Döhle are not peculiar to this disease and both are now regarded as products of degeneration.

Specific cause unknown.

Very recently (1915) Mallory and Medlar have reported a gram-positive microörganism (*bacillus scarlatinæ*) as present constantly upon the tonsils, faucial pillars, soft palate and uvula during the first forty-eight hours.

The streptococcus theory lacks any substantial support despite the common findings in scarlet fever, inasmuch as streptococcic infections with any known strain do not with any constancy produce the phenomena of scarlet fever nor do they establish the type of immunity so marked in that disease.

Prevalence.—It prevails throughout the year, but chiefly in the autumn and early winter months and covers nearly the whole globe.

Age, physique and climate.

Mortality.—The general mortality varies from 10 to 15 per cent., and in children under six years of age it may reach 25 or 30 per cent. As in other diseases of this group, the mortality varies greatly in private as compared with public-service practice, and in weaklings as compared with the sturdy. A damp cold climate seems to increase the severity and frequency of complications, and hence the mortality.

Abrupt onset.

SYMPTOMS.—*With or without antecedent malaise there is* (*a*) *an abrupt onset, usually marked by vomiting with or without nausea, and commonly by sore throat; indeed the latter symptom may be dominant.*

The uvula and anterior pillars of the pharynx are almost invariably fiery red and the angina tonsils are swollen and often enlarged greatly.

A follicular exudate may be present dotting the tonsils, or the exudate may be yellowish or yellowish gray.

High initial fever.

(*b*) **Fever,** 100 *to* 105°*F. at onset, diminishing by lysis with the complete development of the rash and reaching normal in a week or ten days, unless complications occur.*

Early rash.

(*c*) **The Rash.**—*This appears in from twelve to twenty-four hours, first on the chest, and spreads with great rapidity. It is really punctate, the color being deepest about the hair follicle, but it appears diffuse and is commonly described as a boiled-lobster-like redness which spreads over the whole body in a few hours. As in every other eruptive fever, it varies greatly in intensity, in some cases escaping superficial observation unless intensified by a hot wet pack or bath. It should be remembered that it is most intense about the flexor aspect of the joints, the groin, and inner aspect of the thighs, and on the anterior surface of the upper chest.*

Points of election.

Peculiar facies.

In marked cases it is unmistakable and the fact that about the upper lip and upper chin it is often almost absent gives a charcateristic facies.

It appears first usually on the sides of the neck and upper chest and moves downward fading gradually above as it becomes more intense below.

It fades in from three or four to seven or eight days and a *remarkable desquamation* follows, quite unlike that of measles. *The skin peels and may come away in large sheets.* Exfoliation may be complete in a few days or last for three or four weeks, and the scales are infectious, though probably less so than has been believed. The order of desquamation is usually: chest and neck, fourth to seventh day; hands, twelfth to fourteenth; feet, fourteenth to twenty-first.

Lamellar peeling.

Diagnostically suggestive.

(*d*) **Rapid Pulse.**—Excessive rapidity may be present *before the appearance of the rash* and persist throughout the active stage.

(*e*) **Strawberry tongue,** light white or grayish furring punctuated by red and prominent papillæ is a somewhat prominent feature after the first two or three days.

The Blood.—A decided polymorphonuclear leucocytosis is present usually and may reach 50,000 or even 60,000 cells per cubic millimeter in severe cases.

Complications.—*The danger in scarlet fever lies in its complications and*

PLATE X.

Early desquamation showing as white points on the skin of a patient whose rash has not as yet entirely faded. Here and there small circular pin-holes may be seen. (*From "Infectious Diseases," C. B. Ker.*)

of these nephritis is most to be dreaded. This when present takes the form of an *acute nephritis*, developing usually at the end of a week. Mere febrile albuminuria is common and sharp congestion may occur and promptly subside. Nephritis.

The blood-pressure readings should be taken daily by the physician and any definite rise in systolic pressure should direct attention to the kidneys, the urine should be measured and a specimen examined at least once in twenty-four hours, and the patient should be carefully guarded from exposure to cold and wet for some time after the disappearance of all active symptoms of scarlatina.* Safeguards.

Suppurative otitis media is another dangerous complication, resulting oftentimes in mastoid abscess, septic meningitis, general sepsis, and permanent deafness. The throat should be kept as clean by sprays and gargles as the circumstances permit, and the ears should be watched carefully, especially in infants. *Swelling of the cervical glands* is common and may result in extensive infiltration and suppuration. A cause of deafness and meningitis.

Pharyngitis and Tonsillitis.—These may be extreme and a true or pseudo-diphtheritic membrane may be present. The occasional coincidence of scarlatina and diphtheria is not to be forgotten. Diphtheria.

Endocarditis, pericarditis, myocarditis, and even pancarditis frequently result and the heart should be carefully watched. *Parotitis, septic arthritis, broncho-pneumonia, lobar pneumonia, pleuritis*, and *empyema* are rare complications, but *active delirium* and even *convulsions* may be present. Other complications.

Patients must not be pushed in convalescence for persistent weakness is likely to be an indication of myocardial inadequacy.† A common error.

Diagnosis.—*Sudden onset, vomiting, fever, sore throat, a typical rash, and a rapid pulse are unmistakable*, but many cases arise which tax the resources of the cleverest clinician. *Doubtful rashes may be made plain sometimes by the hot wet pack or hot bath. Excessively rapid pulse is* important in connection with any other suggestive symptom. The *hard pulse suggests nephritis* but renal involvement may be present without hypertension. *A secondary rise in temperature always suggests a complication.* Hard pulse.

Cases occur with no discoverable rash and no fever, and these may transmit the disease in its typical form to other children.

There can be little doubt that the severity of scarlet fever has been materially modified during late years. And, furthermore, it must be noted that the tendency to complications and the mortality alike are influenced markedly by climatic conditions, varying more or less strikingly in different portions of our country.

Another factor greatly influencing mortality is the virulence of the individual epidemic. A considerable number of cases observed by the writer during the prevalence of an epidemic in the northern section of

* The early administration of urotropin and the free use of Vichy and cream should diminish the frequency of this dreaded complication.

† The author encounters each year cases in which irreparable damage to the heart has resulted from the almost universal failure to guard the heart in convalescence from acute prostrating diseases.

North Dakota over a decade ago showed an appalling mortality, many cases dying before any rash developed, and renal complications being extremely common.

Age is a factor always to be considered in prognosis; the younger the affected child, the greater being the danger.

Sex plays a smaller part although it is held that the mortality in the male exceeds that in the female.

The utmost caution should be observed with regard to the mild cases lest an under-estimation of the possibility of severe complications should lead to neglect of those precautions which always should be observed.

Spurious Rashes.—Unfortunately, roseolas are common and often due to trivial causes. In differentiation one must bear in mind that the scarlet-fever rash appears first on the chest, is seldom unassociated with sore throat, rapid pulse, enlarged lymphatics, and decided fever and desquamation, following the order given in a preceding paragraph. Rubella should never be mistaken for it as the eruption appears first on the face and is macular. *Indigestion.* Scarlatiniform rashes, evanescent and incomplete, are not uncommon in children as a result of dietary indiscretion but such rashes lack the other essential features of scarlet fever. *Drug rashes* may be traced to their cause and lack cardinal signs. *Septic rashes* are known by their association with other signs of sepsis, onset, etc. *Syphilis.* Scarlatina-like rash is rare in syphilis, and the other concurrent signs make the diagnosis. *Febrile Roseola.* Simple (non-epidemic). Roseola exactly simulates mild scarlatina, but its benign character and the history of repeated attacks may serve to distinguish it. All suspicious cases should be isolated, and no chances taken. It is a bastard syndrome. Duke's "fourth disease" is discussed separately.

Location and associated symptoms.

RUBELLA

(*Rötheln, German Measles*)

Definition.—*A trivial acute contagious exanthematous disease of unknown causation conferring immunity and chiefly affecting children.*

Trivial symptoms.

Symptoms.—*After an incubation period of from fourteen to twenty-three days, the disease begins with slight malaise, mild catarrhal symptoms, occasionally a slight angina, trifling fever, and on the second or even the first day the rash appears on the face, scalp and throat, spreading rapidly over the body, the old areas usually fading as it extends.*

Evanescent rash.

Polymorphic rash.

Either the measles or the scarlatina rash may be closely simulated, *but neither type is constantly displayed* over the entire body and the color is but a "*faded red.*" The disease protects from neither of these ailments and the triviality of its symptoms and the multiformity of the rash will usually readily distinguish it.

Neither catarrhal symptoms nor sore throat are marked, but a stiffness of the neck from swelling of the post-cervical glands is common and may precede the eruption for several days.

There is practically no mortality or serious complication and the diazo-reaction is usually absent.

May be miscalled.

Differential Diagnosis.—Doubtless many cases of rubella of the scarlatiniform type or rather, such of these as are seen first during the second day of eruption, are miscalled "scarlet fever."

Important differential points.

The primary rash is less brilliant and coarser than that of scarlet fever and yet finer, more definitely circular and less blotchy than that of measles.

On the second day it has assumed a decidedly more scarlet-fever-like appearance, *but it lacks the fine punctate spotting and on the inner aspect of the thighs and groin may be distinctly morbilliform. On the foot it may still retain its primary discrete circular arrangement. The color is, as previously stated, "faded red," not scarlet.*

The catarrh is too decided for scarlet fever and usually too slight for measles and both the strawberry tongue of the former and Koplik's spots of the latter are absent. An evanescent crop of discrete red spots may appear on the soft palate or even the buccal mucous membrane but these lack the pearly gray points of "Koplik's spots" so characteristic of measles.

Still *sub judice.*

FOURTH DISEASE (*"Filator-Duke's Disease"*).—This transient febrile exanthem of unknown causation may prove to be a distinct disease if it can be clearly shown that neither scarlet fever nor rubeola confers immunity against it, and that it is not a mere indigestion rash, but it is very like certain cases of rubeola or mild scarlatina.

Incubation.—Its incubation period is said to vary from nine to twenty days and is usually symptomless.

Onset.—The scarlatina-like rash appears abruptly and in two hours may cover the entire body though the abdomen and thighs are likely to be already affected. *It is usually a lighter and less vivid red than that of scarlet fever, affects the body more than the face, or, exclusively, and tends to avoid the area about the mouth as in that disease.* The rash endures usually but a few hours, lasting but for a day or two at the longest and is followed sometimes, according to Duke, by a desquamative period lasting one or two weeks.

A dubious entity.

The author has seen a number of cases which might be called the "fourth disease," but in nearly every instance a dose of castor oil has wrought a magic disappearance of symptoms. Fever is slight and usually lasts only during the active eruptive stage. *Injected conjunctivæ* may be noted. No adequate proof of the existence of a "fourth disease" has yet been adduced.

A "Fifth Disease" has also been described but like the "fourth" has so far not achieved and is not likely to attain an independent status.

ESCHERICH'S INFECTIOUS ERYTHEMA.—This harmless, slightly contagious ailment is unreported in this country and offers practically no constitutional symptoms of consequence.

An erysipelas-like flush appears first on the face and spreads to the body where it affects chiefly the exposed lateral and extensor surfaces of the limbs producing there a map-like configuration. Upon the body the exanthem

assumes the form of a more or less closely packed agglomeration of discrete crescentic patches. It is of slight clinical interest.

DIPHTHERIA.—(*Cynanche maligne, putrid sore throat, suffocative angina, membranous croup.*)

Definition.—*An acute highly infectious, contagious and profoundly toxic disease, due primarily to the Klebs-Loeffler bacillus, but often a mixed infection, characterized by the development of a nasal, pharyngeal or laryngeal false membrane and especially affecting young children.*

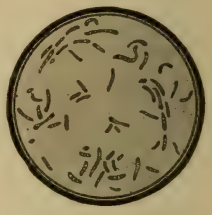


FIG. 517.—Diphtheria bacillus (bacillus diphtheriæ) of Loeffler. Pleomorphic, non-motile, non-sporogenous, non-flagellate, non-liquefying, non-chromogenic, aërobic, readily cultivated and stained by all methods. F. F. Wesbrook has described (1) a virulent type with clubbed extremities and polar granules, (2) granular type, (3) barred type, (4) solid type.

All channels available.

Dissemination and Distribution.—The disease is well-nigh universal and is readily disseminated by contact, contiguity (cough dissemination), clothing, drinking utensils, pencils, school books, and intermediaries, such as parents, nurses, physicians, and certain persons who undoubtedly carry the germ, but are themselves unaffected. Such may carry the germ for months or years and some of them are culturally positive yet non-virulent as to the strains carried. So also foodstuffs, pet cats and dogs, room or street dust may convey it.

Carriers.

Individual Susceptibility.—A decided natural immunity to diphtheria exists in the great majority of individuals. This is clearly indicated by the results obtained by the application of "Schick's intracutaneous test."

It would appear that 75 to 80 per cent. of newborn babes, 40 to 50 per cent. of children between one and five years of age, 75 per cent. of those between the ages of ten and fifteen, and 90 per cent. of adults are thus protected. (See "Schick's Test.")

The introduction of the newer methods for the production of artificial immunity have made it possible to protect individuals for periods of several years' duration.

Organs chiefly affected.

Morbid Anatomy.—Aside from the false membrane itself, the interest centers about the heart, nervous system, and kidneys which are the chief sufferers from the diphtheria toxin. The most important conditions are myocarditis and paralysis of the vasomotor center (the two most frequent complications causing sudden death), laryngeal stenosis and peripheral neuritis. Anterior poliomyelitis or even hemorrhages into the spinal cord are common and the cranial nerves are quite frequently affected. Congestion of the kidneys is almost invariable and acute nephritis not uncommon. The spleen is hyperemic, the lungs not infrequently congested or the seat of bronchopneumonia.

The throat.

Symptoms.—*No disease is more variable in its symptoms and the modern physician has learned to expect diphtheria without marked fever or malaise and even in the absence of marked throat symptoms or the presence of an apparently frank and relatively innocent tonsillitis, while on the other hand what*

appears to be a true membrane upon the tonsils and pharynx may prove non-diphtheritic.

Ordinarily there is *chilliness* and *general malaise*, the *fever rising to* 102° to 103° during the first day, and there is *nearly always sore throat*, and *what is far more important as an early sign* and in dubious or larval cases, *enlargement of the glands at the angle of the jaw.* If the child has been sick for some days its appearance and evident weakness may be out of proportion to the temperature and local signs, yet here again exceptions occur and extensive membrane and marked glandular involvement may co-exist with slight systemic disturbance. *The pulse is usually rapid* and not infrequently irregular and irritable from the onset, but its more significant variations appear, as a rule, later in the disease, when it may be *very rapid, very slow, or show decided arrhythmia,* even to heart block. It is, moreover, *weak and compressible* in any established case *unless there be some decided renal involvement.*

A valuable sign.

Heart symptoms.

The appearance of the throat is variable and it may be that of a mere congestion or simple follicular tonsillitis with a readily detachable pultaceous exudate, but more often shows a definite patch or extensive membrane spreading to the faucial pillars and perhaps to the uvula, naso-pharynx or even the larynx. It is gray or drab (mouse color), dirty and usually shaggy and if removed leaves a raw bleeding surface which it quickly re-covers.

Diphtheria without membrane.

The membrane.

The spread of the membrane is sometimes astonishingly rapid and even on the physician's first visit it may have reached an extreme development. The nervous symptoms of the early stage may be *nil* or consist of restlessness or delirium. In the later stages a state of *profound nervous depression* may be evident, with or without low delirium, stupor, or terminal coma.

Nervous system.

Attention should be paid to the nose and larynx no less than to the throat in every case suggesting diphtheria, as the membrane may be limited to these regions, and the utmost care and caution should be exercised in allowing the child to assume the sitting posture and in getting it out of bed during convalescence. So also the urine should be frequently examined and the heart auscultated and percussed at each visit.

Nose and larynx.

The wise physician will use antitoxin promptly in dubious cases without waiting for a laboratory report, though these are now furnished with remarkable promptness. Nasal diphtheria may at times only be detected by a rhinoscopic examination, laryngeal cases by the laryngoscope, but the evidence of related stenosis in either case, together with the symptoms of profound exhaustion which almost invariably accompany them will point the way. In the former an irritating nasal discharge is common.

Antitoxin.

Membranous croup has properly been shelved by modern methods of diagnosis *and replaced by laryngeal diphtheria* which in 99 per cent. of such cases is the proper descriptive term. The symptoms of *dyspnea* and *suffocation from laryngeal membrane* do not need description, but no more terrible picture is seen in medicine and, *if urgent,* it of course demands *immediate* recourse to intubation, and that failing, to tracheotomy. *Both the nasal and laryngeal forms yield a higher mortality,* in the latter not only because of the obstruction,

"Membranous croup."

Stenotic symptoms.

but more because of the higher degree of toxemia associated with these two varieties.

Sudden deaths.

Complications.—*Myocarditis* and acute dilatation is the most common one and, together with acute vaso-dilatation, accounts for many cases of sudden death. In controllable children efficiently treated with large doses of antitoxin it should occur less frequently than it does now. The uncontrollable ones practically *commit suicide.* *Endocarditis* is not uncommon, and *in any event a weak, rapid, excessively slow, irritable, or irregular pulse demands the utmost care and caution not only during the attack, but long after it.*

Suicides.

Even mild cases of diphtheria must be safeguarded carefully during convalescence inasmuch as serious after-effects or complications may arise in them after all active symptoms have disappeared.

The kidneys may also become seriously involved and the urine should be watched carefully throughout the illness and tested even after convalescence. The daily examination of the heart and determination of arterial tension are imperatively demanded.

Complicating Paralyses.—These may be extreme and severe, or localized and relatively slight. They most commonly affect the palate, occur during convalescence and *almost invariably recover completely.* The complication may be expected in between 10 and 15 per cent. of all cases even though treated with antitoxin. *Diaphragmatic paralysis* is a rare event, but may cause death.

All recover.

A most misleading and readily overlooked paralysis is that of accommodation, which may if disregarded involve serious trouble in school work.

Profound hemorrhages are unusual and nephritis may, but seldom does, prove persistent. *Anemia* should receive attention as soon as the membrane has disappeared and not be postponed until late in convalescence.

Differential Diagnosis.—*The symptoms described enable anyone to make a reasonably sure diagnosis in most cases, but this is always to be controlled by cultural methods. The most important points are the membrane, its distribution, its common tendency to spread rapidly and involve adjoining surfaces, decided evidence of toxemia, glandular swelling, and in certain cases nasal or laryngeal stenosis.*

Culture, the only certain means.

In taking a culture due regard must be had for the situation of the membrane and an attempt made to reach the larynx or nasal passage with the swab if the pharynx is free from exudate.

Follicular Tonsillitis.—The onset is often more severe than is usual in diphtheria, the primary exudate is likely to be purely follicular (spots) and to show less tendency to rapid spread, is lighter, clearer, and less adherent, does not, as a rule, extensively involve surrounding structures and the glandular involvement is less marked. *If operations have been recently performed the wounded surface may be covered with a membrane closely resembling that of diphtheria and lead to error.**

Wound membranes.

* Unfortunately true diphtheria also develops readily upon an operation wound and occasionally upon the vulva or within the vagina.

Prognosis.—There are differences in epidemics and the surroundings, previous health, and resisting power of the individual child cut a considerable figure, *but, practically, the whole outcome depends upon the promptness with which antitoxin is administered and the adequacy of the dose.* It is impossible to get accurate statistics, but we know that without antitoxin the mortality reaches from 50 to 60 per cent., while with antitoxin in private practice where cases are promptly seen and children well nourished it may not exceed or reach 5 per cent.

Act promptly.

VINCENT'S ANGINA.—To Prof. Vincent we owe our knowledge of an angina which, ordinarily assuming the form of an ulcerative or ordinary tonsillitis or pharyngitis, may closely simulate diphtheria. Two organisms are described, one a fusiform bacillus (*Bacillus fusiformis*), the other a spirochæte (*Spirochæta macrodenis*).

The microörganisms responsible for this disease have assumed great prominence of late because of their apparent identity with the microörganisms responsible for so-called "trench mouth" so prevalent in the armies during the Great War. It would seem from investigations made during and following the war period that the usual primary site of infection is the gum margins and that many individuals in civil life carry these without showing the graver symptoms of the disease.

"Trench mouth."

Amongst soldiers the readiness of conveyance from one to another may be brought about through the handing about of pipes and cigarettes, the common use of drinking utensils, and like causes, and the number of individuals carrying the pathogenic organisms is very much larger than is the case in civil life.

Obviously, in every case of Vincent's disease involving the tonsil, pharynx or buccal mucous membrane, the gingival surface should be thought of as the primary source of the infection.

It is also evident that in all cases of apparent diphtheritic infection proving negative to culture, Vincent's angina should be thought of and proper laboratory tests resorted to promptly.

MENINGEAL INFECTIONS

The characteristic individual symptoms of meningitis are very similar in all its acute forms, differing chiefly in degree. The lesion is in every case essentially an inflammation of the pia mater and arachnoid, localized or general, and, in its different forms, shows a more or less well-defined predilection for certain regions.

Various Forms.—*We distinguish*: (1) **Epidemic cerebro-spinal meningitis** *due to the meningococcus of Weichselbaum.** (2) **The simple or sporadic form,** *which is probably due to the same germ.* (3) **Tuberculous meningitis,** *due to direct tuberculous invasion and associated in* 80 *per cent. of all cases with a preëxisting tuberculous focus in other portions of the body, which may be evi-*

* Recently it has been found that various strains exist and that one or the other of these may be the active factor in the individual case. It is necessary therefore to identify the strain present if potent antisera are to be obtained.

*dent only upon a painstaking post-mortem investigation.** (4) **Syphilitic meningitis.** (5) **Secondary meningitis** *due to septic infection or the toxins of acute infectious diseases.* (6) **Serous meningitis,** (*a*) *diffuse,* (*b*) *circumscribed.*

With the exception of the syphilitic and secondary forms the disease shows a special tendency to develop in infancy and early childhood. The syphilitic form almost invariably appears within the three years following the primary infection.

MORBID ANATOMY.—*In the epidemic form* the entire pia mater of both brain and spinal cord is involved, but to the ordinary symptoms of meningeal inflammation are added those indicative of an acute febrile toxemia.

In the septic and secondary forms, the pia of the convexity is chiefly affected, often in limited areas and the base may entirely escape, this being especially true of cases due to direct injury or extension of disease from the mastoid cells or nasal structures.

In tuberculous meningitis the involvement is so often limited to the base as to give it the name of "basilar meningitis," though it would seem that syphilitic meningitis was quite as well entitled to the term.

In cerebro-spinal meningitis the general inflammation of the meninges of the brain and cord is associated with a fibrinopurulent or seropurulent exudate, most abundant at the base of the brain and posterior surface of the cord though, in malignant, rapidly fatal, cases, nothing may be found but an intense congestion. In those of long duration there is thickening of the meninges and yellow areas mark the seat of past exudation.

The ventricles contain turbid fluid, and, in chronic cases, may be greatly distended. Foci of purulent infiltration and of hemorrhage may be found, and cranial and spinal nerves are of course enveloped by the exudate which constantly contains the meningococcus.† In some cases the central canal is dilated. In purulent meningitis there is a tendency to the formation of multiple abscesses and the exudate will yield cultures of the associated germs.

In chronic inflammation of the pia there is an increase in connective tissue producing linear surface projections and a marked tendency to adhesions. By compression, cerebral nerves may be destroyed, ventricular openings obliterated and hydrocephalus produced.

Usual Appearance of Exudate in Various Meningeal Inflammations.—With respect to the macroscopic appearances of the exudate, *actinomycosis* is indicated by yellowish granules; *pyogenic cocci* by a distinctly purulent exudate; *meningococcus cases* by a fibrinopurulent or seropurulent exudate;

* This finding means rather less than is usually believed, unless an *active* focus is present. About 80 per cent. of our adults probably carry old lesions, and older children would probably run to 40 per cent. at least. (See Tuberculin.) Infants and little children show a much lower percentage, however.

† This germ described by Weichselbaum in 1887 is aërobic, non-motile, occurs in pairs, fours, or short chains, and unlike the pneumococcus does not grow on gelatine, but on blood serum, and shows small circular smooth colonies.

whereas it is thick and grayish yellow or yellowish green and gelatinous in *tuberculosis*.

Tuberculous Meningitis.—The development of miliary tubercles is superadded to an inflammation with a more or less characteristic exudate. The tiny gray miliary tubercles appear chiefly in the choroid plexus, along the Sylvian artery, on the inner surface of the dura, on the membranes of the cord, and even in the retina. The usual changes, *i.e.*, hyaline and caseous degeneration, occur and tubercle bacilli are ordinarily readily found in the exudate.

Syphilis is indicated by the presence of soft grayish-red areas, most common at the base, tending to undergo caseous degeneration, absorption, or cicatricial contraction. Both the dura and the cortex may be involved.

Hydrocephalus

Hydrocephalus may result from an arrest of the flow of the cerebro-spinal fluid secreted by the choroid plexuses of the lateral ventricles by blocking, compression or adhesion of the "aqueduct of Sylvius" or the "foramen of Monro." In many cases of tuberculosis and practically all cases of syphilis the exudate is non-purulent.

EPIDEMIC CEREBRO-SPINAL MENINGITIS

(*"Petechial fever," "Spotted fever," "Malignant purpuric fever," "Brain fever"*)

Definition.—*An acute, contagious leptomeningitis, strikingly, though not wholly, epidemic in type,* caused by the *Meningococcus intracellularis meningitidis, and transmitted by contact, contiguity and healthy intermediaries carrying the meningococcus in the naso-pharynx.*

Fig. 518.—Epidemic Spinal Meningitis. Meningococcus in centrifugated cerebro-spinal fluid. Stained with Loeffler's Methylene Blue.

The incubation period varies from a few hours to a week or ten days, and *the onset is usually sudden,* though headache, anorexia and pain in the back may precede it.

It affects individuals of both sexes equally, most commonly attacks those between ten and twenty-five years of age and seldom occurs in adults who have passed the age of forty-five.

Symptoms.—In some instances a preliminary stage occurs which is marked by decided and often severe nasopharyngeal symptoms. This is followed after several days by the more decided evidences of established infection. *There are* chills, causeless projectile or regurgitant vomiting, headache of increasing severity, associated in little children with the plaintive, wailing cry representing its exacerbations, severe backache, usually painful cervical and dorsal rigidity and moderate fever (101° to 102° F.). Auditory and visual hyperesthesia coexist with restlessness, irritability and muscular tremor. The head is retracted (*boring occiput*), *opisthotonos or, more commonly, orthotonos may occur and, in children, general convulsions.*

Irritative symptoms.

Facial spasm, strabismus and ptosis are common, pain in the head, back and extremities is decided and progressive and, less frequently, the trunk muscles may be paralyzed.

Hyperesthesia of spinal or general localization is often noted and marked irritability may give place to delirium, stupor and finally coma. Albumin, sugar, and in malignant cases, blood, may be present in the urine.

The fever curve *is extremely variable, some cases showing hyperpyrexia, others almost no fever.*

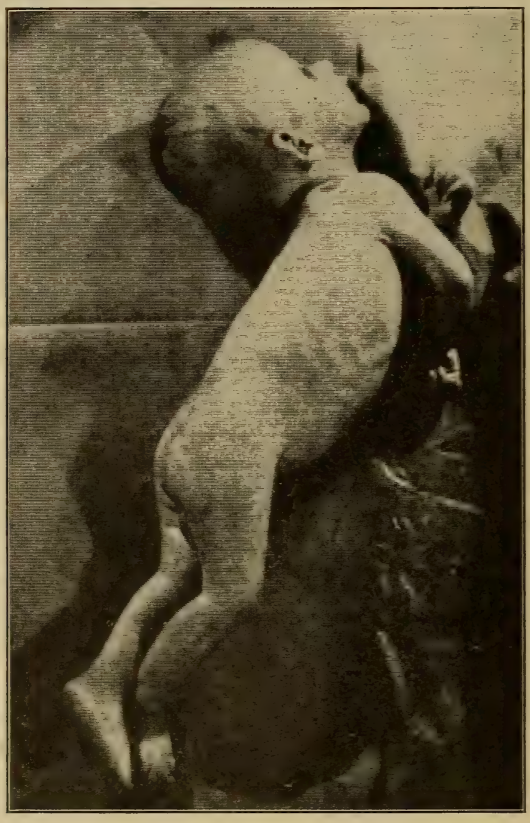


FIG. 519.—Marked cervical retraction. (*After Knopfelmacher.*)

Respiration is not markedly increased. The pulse is sometimes remarkably slowed and almost always weak, but in children may be much accelerated.

Blood.

Leucocytosis is present in all cases (20,000 to 40,000).* The bowels are usually constipated, the spleen moderately enlarged, *excessive* vomiting, without nausea and of the projectile type, is an unusual but troublesome complication.

Variable.

Skin Rashes.—*The cutaneous symptoms vary greatly, herpes is almost constant and purpuric spots occur in from two-thirds to three-fourths of the cases.*

* Leucocytosis is sometimes present in tuberculous cases though not so constantly.

Dusky mottling, erythema, rose spots, urticaria and various eruptions may be present.

Malignant cases may kill so promptly that the brain shows at autopsy only an acute congestion. They are sometimes *apyretic* and are almost invariably associated with purpuric rash and feeble pulse, somnolence and profound asthenia. Rapidly fatal.

Intermittent Form.—This is characterized by a febrile curve of a distinctly pyemic type. A chronic form may succeed the acute.

Abortive Form.—This "type" is characterized by a severe onset, an extremely short duration and a sudden termination, followed by rapid convalescence. Certain other mild types present symptoms hardly more than suggestive which promptly subside and in each of these varieties there is abundant opportunity for damaging erroneous conclusions. Errors common.

Complications.—These are, chiefly, pneumonia, often of a true meningococcus type, septic arthritis, peri- and endo-carditis and parotitis. Convalescence may be greatly retarded by persistence of headache, nervous irritability, mental impairment, or long-persisting cranial nerve paralysis. Optic neuritis occasionally occurs as do purulent conjunctivitis, choroiditis and iritis. Permanent deafness and persistent ocular palsies are very common and the frequency of nasal catarrh suggests the nasal secretion as a possible chief vehicle of infection. Convalescence tedious. Residual lesions.

Kernig's Sign.—*Flex the thigh sharply upon the abdomen, the leg upon the thigh; if meningitis is present, extension of the leg is then prevented by contraction of the hamstrings.* Exact procedure necessary.

Fig. 520.—Kernig's sign. Improper method (*After Sahli-Wiener.*)

This sign is present in 90 per cent. of the cases, but also is to be found in sciatica, diseases of the hip and knee-joint and lesions of the cerebellum or upper motor neurons and to a less degree in old age or after prolonged fixed recumbency. Present in other conditions.

Brudzinski's Phenomena.—These may sometimes be helpful in diagnosis and are referred to elsewhere under "Synkinesias."

Diagnosis.—*The presence of an epidemic, and the characteristic symptoms of cerebro-spinal irritation and compression ordinarily make the diagnosis easy, yet the cerebral symptoms present in other acute febrile conditions are often misleading. Lumbar puncture is the decisive test.* Chief features.

The rash, if present, is of great assistance in diagnosis. Herpes, leucocytosis and diminished urinary chlorides assist in the differentiation from typhoid of the meningeal type; the physical signs, from pneumonia, though either disease may co-exist with meningitis (see also "Tuberculous Meningitis," "Differential Diagnosis").

Prognosis.—Over 50 per cent. of the deaths occur in the first week and the mortality under Simon Flexner's serum treatment ranges from 20 to 50 per cent. according to the early or late administration of the remedy and the type of patients, being higher in children than in adults and most Importance of early treatment.

excessive in those children under one year of age. Hyperpyrexia, coma, repeated convulsions and sudden fall of fever are unfavorable signs.

Technic.

LUMBAR PUNCTURE.—*Place the patient on the side, the body so bent as to get the maximum dorsal curve. Carefully count the vertebræ and introduce the sterile hollow needle slightly* (0.5 *cm.*) *to one side of the median line between the third and fourth lumbar vertebræ,* or, better still, between the fourth and fifth at the level of the iliac crests, the needle point being directed toward the center of the canal and slightly upward.*

Point of election.

Sterilization of the skin with iodin is necessary and sterile graduated tubes should be at hand to receive and measure fluid.

FIG. 521.—Kernig's sign. Proper method, *i.e.*, preliminary flexion of thighs on abdomen followed by attempted extension of leg on thigh.

Children vs. Adults.

In children, a point midway between two spinous processes should be chosen; in adults it should pass just below the lower margin of the upper process of the chosen space. A long needle, (9 to 10 cm. in length and 0.6–1.2 mm. calibre) carrying a stylet, is needed, the distance traversed varying from 2 cm. in children to from 4 to 6 cm. in the adult.

Depth of puncture.

Pressure indications.

Pressure is indicated by the force of the flow, and normally represents 5 to 7 mm. of mercury, which may be increased to from 15 to 60 mm. in meningitis and tumors (Sahli). It is easily measured by attaching a small (1 mm. caliber) mercury manometer. Any admixture of blood interferes seriously with cytodiagnosis. It often appears temporarily only, and in such cases a good specimen may be secured. If persistent a repetition of the procedure at another sitting may be necessary.

Spinal fluid.

Normally the fluid is clear and limpid, specific gravity 1006–1013, and but slightly albuminous (0.02 to 0.05 per cent.). Marked cloudiness and the presence of leucocytes points to septic meningitis, the fluid being relatively clear in tuberculous meningitis and cerebral abscess. High albumin content is also significant though occasionally encountered in tumor and apoplexy.

Globulin Content.—This is far more important than the test for albumin in that only the faintest trace is present in normal spinal fluid.

In most cases of cerebrospinal syphilis and paretic dementia it is decidedly increased.

Globulin Test.—Rose & Jones' modification of the Nonne-Apelt method is simple and definite.

Test.—To 2 c.c. of a saturated solution of Merck's ammonium sulphate add 1 c.c. of cerebrospinal fluid by running it slowly down the inside of the inclined test tube.

A delicate grayish white contact ring should appear at about three minutes.

The differential value of the interesting but complex "colloidal gold-test" is not yet determined fully.

* To avoid the spinal cord Sahli prefers the interval between the fifth lumbar and the sacrum.

Caution.—Lumbar puncture is a procedure of importance and some risk and must not be made a matter of hasty routine procedure, nor performed under adverse conditions save in emergency.

Lateral recumbency for the patient, the withdrawal of only small amounts of fluid in all save meningeal cases, and absolute rest in bed for 24 or 48 hours in the dorsal recumbent posture, following exploration are all matters of importance.

Headache, often severe and persisting over several days, may follow lumbar puncture in a considerable portion of patients and need excite no serious alarm.

In the case of cerebral tumors, puncture is attended by considerable danger if large amounts of fluid are withdrawn.

ACUTE TUBERCULOUS MENINGITIS.—The symptoms of this ailment are *in general* exactly the same as in other forms of meningitis, but, as assisting differential diagnosis, there is frequently a *history of preëxisting active tuberculosis* in other parts of the body, or the disease may be a part of an acute miliary process. *Furthermore, there are usually marked prodromal symptoms such as a general failure or impairment of health extending over several weeks, loss of weight and appetite, irritability, headache and restlessness.*

Preexisting disease.

Prodromata marked.

First Stage.—The actual onset is occasionally somewhat abrupt in children and there is beginning cervical rigidity, the curious whining "*hydrocephalic cry,*"* apparently due to abrupt exacerbations of the headache. *The pulse, at first rapid, becomes slow and irregular,* the respirations irregular and sighing and the pupils are generally contracted (stage of irritation). Projectile vomiting may or may not occur.

Peculiar cry.

Second Stage.—In the second stage *there* is obstinate constipation, marked emaciation, retraction of the abdomen (*scaphoid belly*), boring occiput, stupor and irregular or dilated pupils, often associated with strabismus. The temperature is usually moderate in both stages (101° to 103°F.).

Scaphoid belly.

Third Stage.—The third stage (*paralysis, coma*). Paralysis of the ocular muscles is *common, optic neuritis may be present,* diarrhea may replace constipation *and the victim lies* in a "typhoid state," *with partially open eyes and dilated pupils.*

Temperature may at this stage be subnormal, the pulse rapid and there may be ante-mortem hyperpyrexia.

The Duration.—This varies from a few days to a month, from two to three weeks being the common period. Occasionally instances of a chronic course are encountered some of which lack the pronounced initial symptoms.

Kernig's sign is of course present in this form and the spinal fluid usually contains tubercle bacilli and shows mononuclear cell predominance.†

Spinal fluid.

The tubercle bacillus usually may be determined readily in the spinal fluid but a proper procedure involves the preservation of a carefully drawn specimen for 12 hours in the ice box.

* Occasionally screaming is marked and almost constant.

† Animal inoculation is sometimes required to prove the presence of tubercle bacilli.

The clot which forms is then withdrawn and direct staining of smears is undertaken.

Inoculation of guinea pigs should be a *routine* procedure when facilities are available.

General Comment.—The greatest diversity of symptomatology must be expected in a lesion involving the brain stem and perhaps the cortex in an inflammation associated with the outpouring of an exudate. The pupillary symptoms are quite constant as regards miosis of the early stage and the irregularity and dilatation of the later periods. Not only projectile vomiting, but convulsive seizures or even tetanic contractions or catalepsy may be encountered in the earlier periods, and ocular palsies may exist alone or be associated with extensive cranial involvement.

Symptom diversity a feature of meningitis.

Prognosis.—*Practically all cases die.*

Differential Diagnosis.—Tuberculous meningitis, in contrast to the epidemic form, is almost invariably of gradual onset; shows less fever and that often of the inverse type; rarely shows any leucocytosis, in sharp contrast to the epidemic form, in which leucocytosis is constant and decided; only in exceptional cases shows herpes; pursues a slow course; presents fever, spinal symptoms and, on lumbar puncture, yields an exudate showing no meningococci, mononuclear cells in predominance and tubercle bacilli.

Contrast decided.

Meningococcus absent.

Rigidity and retraction of the neck is extreme in the epidemic form, less marked and, in rare instances, absent in the tuberculous form.

A family history of tuberculosis or active pulmonary or glandular foci may be demonstrated by physical signs or *radiography* and retinal tubercles are sometimes revealed by the ophthalmoscope.

SUPPURATIVE MENINGITIS.—This differs from the tuberculous and the epidemic form alike in the distinctly septic fever curve, often with pronounced and repeated chills and sweats, and in its usual freedom from purpuric manifestations and herpes alike.

Septic type.

Lumbar puncture reveals a predominance of polymorphonuclear leucocytes, as in the epidemic form, but the meningococcus is lacking by cultural tests and other pyogenic cocci are found in abundance.

Meningococcus absent.

Caution.—It should be remembered that redness and swelling of joints may occur in the course of epidemic or septic meningitis, *but not at the onset* as in acute rheumatism. One need but know the fact of their possible appearance to avoid confusion.

SYPHILITIC MENINGITIS.—This combines symptoms of brain tumor with those of meningitis; is essentially chronic in type and peculiarly variable in its meningeal manifestations. It cannot well be confused with the acute forms. The slowly developing and progressing symptoms are those of brain tumor, headache being pronounced and usually increased at night, and irritability, change in disposition and lack of appetite are *prominent features.*

Brain tumor symptoms.

Causeless vomiting and vertigo may be present and eye symptoms or facial paralysis may indicate involvement of the cranial nerves. *In fact nearly all such persistent cranial nerve involvements are of syphilitic origin.*

Cerebellar ataxia and polyuria may occur and, if the Sylvian fissure be

involved, there may be hemiplegia or aphasia. To name all of the symptoms would be to review the whole subject of brain symptomatology.

Wide range of symptoms.

The essential feature is the distinctly slow, halting, indeterminate, onset and progress, interrupted by the intervals of betterment in many instances and the ready response of the condition to treatment.

Slow onset and therapeutic test the features.

The diagnosis is usually assisted by a history of syphilis within three years or visible evidence of previous infection and positive Wassermann and luetin tests. Without antisyphilitic treatment an absolute diagnosis between syphilitic meningitis and brain tumor can hardly be made.

ALCOHOLIC MENINGITIS.—The form of meningitis known as wet brain deserves special mention because of its occasional occurrence in alcoholics and certain peculiarities in its course.

History of alcoholism.

It usually terminates a long debauch and is frequently a terminal event in delirium tremens. Its characteristics are stupor or coma with low muttering delirium, hallucinations and delusions. There is general muscular tenderness, cutaneous hyperesthesia, contraction of the pupils, and after a short time muscular rigidity, occipital retraction, loss of control of the sphincters, exaggerated reflexes and scaphoid belly. The temperature is variable, often being normal or slightly raised throughout the disease, occasionally higher, especially at the close. The final stage may never be reached, the case terminating in recovery. The diagnosis is based largely upon the peculiar delirium and the history of alcoholism and is sometimes assisted by a knowledge of the occupation.

Hallucinations marked.

Occupation may be suggestive.

It must be regarded as an expression of low grade infection by any one of several pathogenic microörganisms.

SECONDARY MENINGITIS.—These cases present merely the symptoms of meningitis as already described, usually in the presence of a suggestive, antecedent acute or chronic disease, or the history of traumatism, accidental or operative.

In acute diseases error is likely to occur from two sources: one, the fact that certain acute febrile ailments such as pneumonia may present marked meningeal symptoms without true meningitis; the other, the liability that true meningeal symptoms may be masked by those of the disease which it complicates. These difficulties apply of course only to the *stage of irritation* and the doubtful diagnosis may almost always be made certain by lumbar puncture (see "Diagnosis" of "Epidemic" and "Tuberculous" forms).

Lumbar puncture in doubtful cases.

"Circumscribed Serous Meningitis."—This probably does not deserve separate consideration, representing as it does a condition whose symptoms are determined merely by its more or less fortuitous maximal localization in the cerebellarpontine angle.

Nevertheless, the condition is one which might give rise to serious diagnostic error by reason of the peculiarly *otic* character of its symptoms.

These are: *deafness, tinnitus aurium, pain behind the mastoid process, suboccipital headache, facial nerve involvement, and high intracranial pressure.*

It may complicate almost any form of meningitis and is often confounded with brain tumor. One of the most interesting and characteristic symptoms

is the so-called "pointing-error" a modification of the older "touch-test,"* in ataxia.

The preliminary induction of so-called "caloric" horizontal nystagmus is prerequisite to the performance of the test. The attempts to carry out touch-movements show a constant deviation error in the direction opposite that of the induced nystagmus.

INFANTILE MENINGITIS.—A word is necessary concerning the special symptoms present in cases of simple meningitis affecting babes. The period of maximum susceptibility seems to lie between the third and sixth month.

The symptoms are usually sudden in onset, and frank in their character; pain in early stage being indicated by pulling the hair and constant crying or screaming. Emaciation is peculiarly rapid, and *hydrocephalus* is usually evident by the second week, the head enlarging and assuming the typical outline, the fontanels bulging, and changes in the eyes, such as undue prominence, strabismus, and the usual pupillary symptoms, being evident. The tendency in the simple cases is to chronicity, and recovery occurs in perhaps 20 per cent. Many serious and permanent impairments persist, these being chiefly deafness, mental defects and blindness.

CHRONIC REMITTENT MENINGITIS.—The symptoms of this ailment are essentially those of chronic syphilitic or tubercular disease of the meninges and therefore consist almost wholly of pressure phenomena. Any form of meningitis may, however, become chronic.

Whether we should recognize as a distinct ailment the so-called "serous meningitis" of this type would seem to be doubtful.

Under the names of "*Primary Idiopathic Hydrocephalus*," or "*Quincke's Disease*," we have been giving special recognition probably to various wet meningitides of diverse bacillary or toxic origin.

It would seem to be sufficient at present to regard these as chronic subinfections for the greater part, but hold clearly in the mind the fact of their occasional occurrence.

The forms with an abrupt onset are peculiarly misleading, the picture being often that of brain tumor (vomiting, headache, optic neuritis and cerebellar ataxia) or that of *acute hydrocephalus*.

Pressure symptoms.

The essential points lie in the fact that in such cases we deal with evidences of high intracranial pressure, a limpid, clear, spinal fluid containing few cells, with remissions and with chronicity.

LETHARGIC ENCEPHALITIS

During the winter of 1918–19 there appeared in the United States what was apparently, so far as this country was concerned, a new epidemic involving the central nervous system and representing an encephalitis with distinct lethargic symptoms. It is probable that the first cases arose in Vienna

*The elaborate mechanism and the technic of the complex and difficult "pointing error" tests is described fully in Dr. L. F. Barker's admirable work, "Monographic Medicine," Vol. IV, pp. 169-176, 1916.

during the winter of 1916. It is also probable that the disease itself is not a new one and may conform to what in 1890 was termed "nona" in Europe where cases seem to have been reported in Austria, Italy and Switzerland. It is also suggested that the disease may be identical with what was called "sleeping sickness," an epidemic of which occurred in Germany in 1712.

It has been thought by many that lethargic encephalitis was one of the sequels of influenza but this would seem to be improbable in view of the fact that the first cases reported abroad occurred before the date of the influenza epidemic of 1918. Nevertheless there seems to have been a close relationship in the past between the epidemic occurrence of the two diseases.*

Etiology.—It is evident that the disease is communicable and in some instances institutional outbreaks have occurred. On the other hand there are relatively few examples of multiple cases in the same household.

Many efforts have been made to discover the specific organism responsible for this curious disease and several observers have recovered and cultivated successfully microörganisms which they believe causative. Nothing so far is proven, however, and the etiologic factor remains a mystery.

Pathology.—Those portions of the central nervous system especially affected are *cortical* and represent chiefly the tissues in the neighborhood of the third ventricle, the aqueduct of Sylvius, the lateral ventricles, the optic thalamus, the pons and medulla. The lesions chiefly take the form of *cellular infiltrations* in the nerve tissues themselves, cellular aggregations about the blood vessels, edema, and small, often microscopic, perivascular hemorrhages; these changes occurring both in diffuse and circumscribed (nodular) forms. The *spinal cord* is affected relatively late, but may show changes in the cervical portion.

Tissues involved.

Chief lesions.

Symptomatology. *Onset.*—Nothing is known definitely of the incubation period and there seem to be few or no prodromal signs. The onset is strikingly sudden in many cases, but usually there is a prodromal period varying from a few days to a month or more. During this time the patient may experience unusual somnolence, headache, the varying degrees of asthenia, defective memory and disturbances of vision.

Initial Symptoms.—Associated with an inflammation of the upper respiratory tract, there are chills, chilliness, general malaise, anorexia, nausea, headache often severe, and more or less generalized pain.

Fever.—This may be present at the outset or be delayed in its appearance, and in most instances does not reach high figures, the usual case showing a reading of from 101°F. to 102°F. Exceptionally the range is higher and may reach or even exceed 104°F. In many instances the temperature drops to subnormal after a few days and shows thereafter only an occasional rise, usually of moderate range.

Later Symptoms.—The dominant and outstanding feature of the disease is the *drowsiness* and *lethargy*. The patient appears dull and apathetic, prolongs unduly the natural sleeping hours, and can be aroused only with

Drowsiness.

*There would appear to be no substantial reason for assuming any direct connection between *encephalitis lethargica* and acute poliomyelitis.

Usually develops gradually.

some difficulty. This symptom may be present at the outset of the disease but is usually developed gradually and progressively. It resembles in most cases the profound sleep encountered in children and is associated with the same difficulty in arousing the individual to consciousness. Variants occur, representing on the other hand a mere deepening of this condition into actual stupor, the passage into coma, or, in a small number of cases, the occurrence of delirium.

Clearness of mind.

One of the most striking things in connection with the somnolence in many instances is the *clearness of mind* shown by the patient when aroused with difficulty from deep stupor.

Other signs.

Blurred vision, diplopia, vertigo, tinnitus aurium, photophobia, muscle twitchings and tremors may develop and be associated with restlessness, irritability, or more or less profound mental depression prior to the onset of the deeper lethargic stage.

Paralysis.

Third nerve paralysis and facial paralysis may occur, and symptoms of meningeal irritation are present usually in some degree and at times so markedly as to suggest an actual meningitis.

Rigidity of muscles.

Rigidity or spasticity of the muscles of the extremities is a relatively common symptom and this may involve the face and produce a mask-like countenance.

Erythema.

Early in this disease cutaneous manifestations may be present in the form of diffuse erythema or a morbilliform rash.

Duration and Course of the Lethargic State.—The lethargy may continue throughout weeks or even months, even in cases making a full and complete recovery, and the recovery of mental function ordinarily is gradual. Indeed the same statement applies to other symptoms of the disease. More rarely, a relatively rapid recovery occurs not only from the somnolence but from the paralyses as well.

The Spinal Fluid.—The spinal fluid in these cases is quite unlike that in meningitis, being clear, showing but a slight increase in the number of cells per cubic millimeter, and no pathologic globulin content. The cells present are both mononuclear and polymorphonuclear.

Occasional Kernig's sign.

A factor leading to errors in diagnosis is the occasional appearance of Kernig's sign in this disease.

Diagnosis one of exclusion.

Diagnosis.—Obviously, in this disease the diagnosis is one of exclusion. Meningitis, brain tumor, typhoid or paratyphoid fever are the three conditions most likely to cause confusion. The application of the usual test for typhoid and paratyphoid would serve to exclude that disease; the history and the examination of the fundus oculi would usually suffice to exclude brain tumor; and the normal spinal fluid content would exclude meningitis.

High death rate.

Prognosis.—The death rate is not as yet definitely established, but is high, the reports varying from 20 to 40 per cent.

Microörganism causing epidemic poliomyelitis. (*Flexner and Noguchi.*)

Fig. 1.—Culture in ascitic fluid tissue medium of Noguchi, showing opalescence. The translucent zone above is paraffine.

Fig. 2.—Solid culture showing colonies and line of demarcation, between the opalescent colony-filled medium below and the unaffected upper level. (*From the Journal of Experimental Medicine*, Vol. XVIII, 1913.)

ACUTE INFECTIOUS POLIOMYELITIS

(*Acute Anterior Poliomyelitis, "Infantile Paralysis"*)

Through the brilliant work of Simon Flexner and his colleague, Noguchi, the cause of this immunity-conferring, but deadly and disabling, scourge of children and young adults is proven to be an extremely minute, filtrable cultivable, non-motile, virus, for which Barker has proposed the name, *Flexneria noguchii.*

The resistance of this virus is extremely great both to *cold and drying*, though it is readily destroyed by heating, and, fortunately, by sunlight.

It has been grown successfully by Flexner and Noguchi on ascites-fluid-agar to which a sterile bit of rabbit's kidney is added. Air is excluded from the culture by a layer of paraffin oil.

The specific virus occurs singly, or, in pairs, chains, or groups, which are Gram-positive and with the Giemsa stain appear as minute globular or globoid bodies varying in diameter between 0.15 and 0.3μ.*†

It retains its virulence even when grown for 20 generations on an artificial medium and shows a most striking elective affinity for the spinal cord and brain.

The disease is readily inoculable into monkeys, is easily passed on through a series in experimental work, is constantly present in the nose, throat and central nervous system, whatever the type or degree of severity of the infection, and persists for weeks or months in the naso-pharynx after all active symptoms have subsided.

Flexner seems to feel convinced that all channels of infection are wholly subordinate in importance to direct contact or contiguity.

The fly is one of its carriers through food substances and it is disseminated also by dust but fortunately its actual *contagiousness* is slight.

Natural immunity is decided and a comparatively small proportion of exposed individuals contract the disease.

About 90 per cent. of all cases occur in the first decade of life, 60 per cent. under the age of three years, and nearly all arise during the summer months.‡

It is distinctly an epidemic disease, though sporadic cases occur and its incubation period is from two to fourteen days.

A fleeting passive immunity only has been attained in monkeys by the use of immune serum, but on the individual infected the disease confers absolute immunity.

*Flexner and Noguchi: Jour. Exper. Med., Vol. X, 108, 1913.

† Various observers have reported what they have believed to be the causative microörganisms, notably Geirsvold who reported a diplococcus and, very recently, E. C. Rosenow who reports a streptococcus. At the present time, the virus of Flexner and Noguchi only is generally accepted as capable of producing the typical cord changes.

‡ Severe winter epidemics have been reported but are most unusual.

The virus may remain in the nasopharynx for at least five months after recovery, but it is seldom infective after six weeks.

Morbid Anatomy.—Contrary to our older conceptions it is actually a polio-myelo-meningo-encephalitis, with *predominant* destructive lesions in the ganglion cells of the anterior horns of the spinal cord which results in varying degrees of degeneration of the components of the lower motor

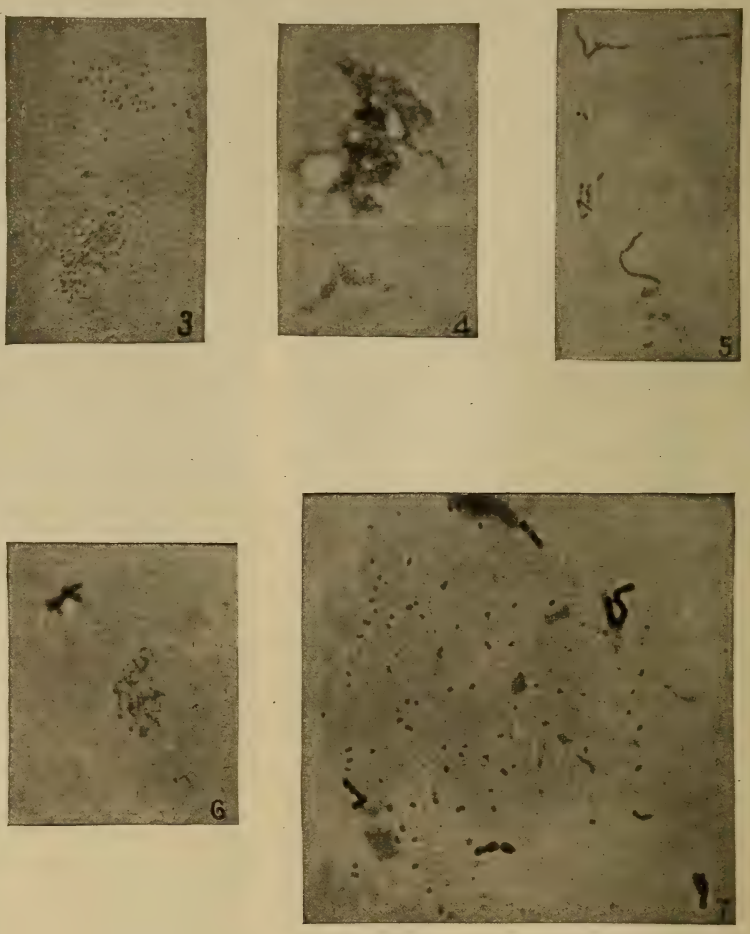
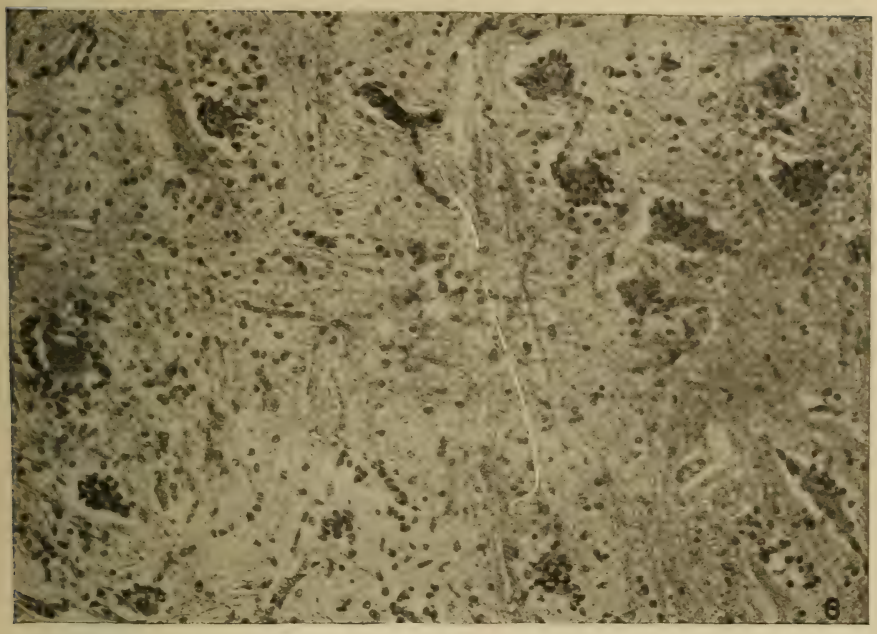


FIG. 522.—(3) Separate globoid bodies. × 1000. (4) Aggregated masses of globoid bodies. × 1000. (5) Chains and pairs of globoid bodies. × 1000. (6) Chains of globoid bodies compared with streptococcus pyogenes. (7) Agar fragment showing pairs of globoid bodies compared with streptococcus pyogenes. × 1000. (*Flexner and Noguchi, Journal of Experimental Medicine*, Vol. XVIII, 1913.)

neurons affected, axis cylinders, nerve roots, peripheral nerves and the muscles activated by them. The chief pathologic change is one of perivascular lymphangitic inflammation and round-cell infiltration about the central arteries each of which supplies a segment of the cord about 5 cm. in length, which corresponds to the focal nature of the changes noted in the disease.



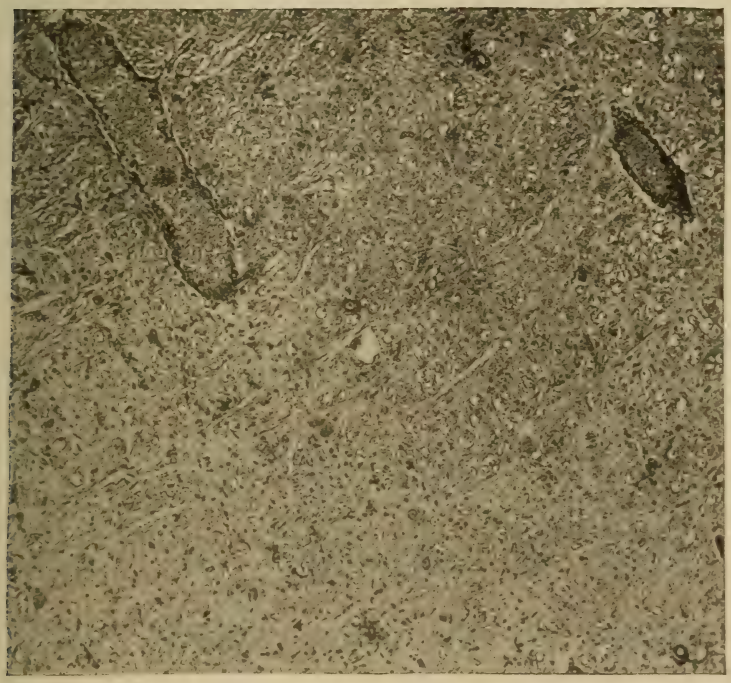


FIG. 523.—(8) Spinal cord showing degeneration of nerve cells and invasion of leucocytes (neurophagocytosis). × 144. (9) Perivascular and interstitial mononuclear cell infiltration of medulla. (*Flexner and Noguchi, Journal of Experimental Medicine*, Vol. XVIII, 1913.)

Round-cell infiltration of the pia-arachnoid is present and chiefly marked on the anterior surface of the cervical, sacral, and lumbar enlargements. Extensive foci of infiltration and edema are evident in the white substance of the cord which accounts for the fleeting nature of some of the symptoms observed.

In extreme or fatal cases, like changes are found in the pons, medulla, centrum ovale and cortex but the ganglion cells seldom or never undergo such destruction as is a constant finding in the cord segments chiefly affected.

This disease, like epidemic cerebrospinal meningitis, is a general infection showing a peculiar elective affinity for the brain and cord and exerting its pathogenic effects predominantly upon the cells of the anterior horns.*

Symptomatology.—The onset is variable; in certain cases the paralysis is unconnected with any definite febrile attack; more commonly (80 per cent.) there is slight or moderate fever lasting about twenty-four hours, and malaise, while rarely, in young children or infants high fever, diarrhea, vomiting, delirium, convulsions and coma may be present.

The more severe the primary attack, the more rapid is the onset of paralysis.

Pain and tenderness in the affected limbs may first attract attention but are usually absent or unimportant and *the paralysis may be complete in from twenty-four to forty-eight hours* after the onset. It may involve all four extremities, rarely one, oftener both legs and especially the peroneal and tibial groups, and tends to recede in certain muscle groups and become marked and permanent in others.

Whenever a large muscle tract is involved, great inequality of damage is evident and certain muscles such as the forearm extensors are almost immune. Secondary contractures occur late in the disease.

It should be remembered that the onset of this disease is sudden, the paralysis appears within from twenty-four to forty-eight hours, the lesions soon become fixed, the paralysis is abrupt, flaccid, complete, atrophy rapid and extreme, reflexes and normal electrical reactions lost, sensation unaffected, and the sphincters and facial muscles rarely involved.

Cardinal Early Symptoms.—1. *An extraordinary pain response to every movement, passive or active.* 2. *Marked hyperesthesia of short duration.* 3. *Profuse sweating.*

In a large proportion of the cases these symptoms are present before paralysis supervenes.

Chief Clinical Types of the Disease.—*There are three chief types.*

1. *The Respiratory Type.*—In this the physician or parents may be misled by the appearance of what seems to be a simple coryza, bronchitis or, more frequently, a sore throat.

2. *The Gastrointestinal Type.*—In this form there may be nausea, vomiting, diarrhea, or obstinate constipation. Vomiting may be present only on

* The characteristic changes were admirably summarized by H. E. Robertson and A. J. Chesley in 1910. ("Pathology and Bacteriology of Acute Anterior Poliomyelitis," Archives of Internal Medicine, Vol. VI, p. 233-269).

the first day or persist for several days. Diarrhea is very common (two-thirds of cases) in some epidemics. Appetite is lacking and the tongue heavily coated in many instances. Such cases resemble a so-called bilious attack and are usually so treated.

3. *The Meningeal Type.*—Headache, vertigo, tremor, twitching, unsteady gait and even convulsions may be encountered. In this form or the others spontaneous pain along the spine, with marked tenderness, may be observed. Slight rigidity of the neck and spine may be present.

Misleading Factors in Diagnosis.—*There can be no doubt that a large number of cases escape diagnosis and act as unconscious carriers of virulent organisms for weeks, while mixing freely with their kind. It is evident that the onset even in the usual case may be of the most misleading character, simulating influenza, neuritis, rheumatism, meningitis, gastroenteritis, scarlatina, tonsillitis, herpes zoster, a simple "bilious attack," or even (in the mother's mind) "growing pains."*

Save during a severe epidemic, the greater number of cases have no medical attendance until paralysis has declared itself.

It is now certain that a large number of cases are abortive and do not show paralysis, although they are as highly infective as those of the most fatal type.

These facts emphasize the necessity of determining the character of the deep reflexes and seeking for the cardinal symptoms of the early stage in all sick children during any epidemic, and importance should be ascribed, not only to diminished reflexes, the usual finding, but also to any abnormalities in response.

It must also be remembered that suggestive weakness may be present even though decided paresis or actual paralysis is lacking.

Prognosis.—The disease is a terrible one by reason of the strong tendency to persistence of the paralysis. In certain cases, brisk and persistent eliminative treatment may result in complete or partial recovery.

The actual mortality varies in different epidemics but runs between 2 and 20 per cent. Death occurs usually before the end of the fourth day in fatal cases.

As to recovery from paralysis much depends upon the severity and degree of the original attack and the institution and *persistence* in a proper line of treatment.

Many cases show betterment under treatment extending over a period of two years, and probably more than 20 per cent. make a complete functional recovery.

SMALLPOX

(*Variola*) (A. S. *pock*, bag or pocket)

Definition.—*An acute infectious and highly contagious disease, conferring immunity and characterized by a definite mode of invasion and a peculiar eruption.*

Historic Note.—Smallpox was so called originally in contradistinction to great pox (syphilis). It has existed for thousands of years in Eastern

Smallpox vs. Great pox.

Formerly inevitable.

countries, was introduced into Europe in the eighth century, and by the tenth had become so general throughout Europe that prior to the day of Jenner, few persons failed to show on the face its characteristic pits.* It was first brought to the American continent by the Spaniards in the sixteenth century, and to Massachusetts in 1633. The aborigines and negroes seem to be especially susceptible and the disease flourishes in its most virulent form on virgin soil. Once so general and fatal, it has in our day become a comparatively

A medical hero.

rare disease with a low mortality. For this we have to thank that great and courageous physician, Edward Jenner, who, being convinced of the identity of cowpox and smallpox, in 1796 vaccinated, and six weeks later inoculated with smallpox virus, a boy of eight, reporting the success of his experiment in June, 1798. To appreciate his courage one has only to read the journals of

Inoculation.

that period in which he was most heartily denounced and abused. Prior to this time, Lady Wortley Montagu had become convinced of the value and efficacy of inoculation as practised in the far East and succeeded in her efforts to introduce this procedure into England.† Unfortunately, the extreme care characteristic of Eastern nations in choosing a proper age, season, and state of health was not observed by their English imitators, with the result that the good accomplished was overbalanced by the extreme mortality and disease incidence engendered. The Asiatic races had for centuries successfully practised inoculation upon a most rational basis; nevertheless, the disease is still a scourge in India, where effective vaccination is rendered extremely difficult by the attitude of certain of the people, who believe smallpox to be a form of purification sent by the gods and who object to the use of cows for the production of vaccine.

Etiology.—The cause of variola is not yet fully proven, though it is evident that its virus is contained in the contents of its vesicles and pustules

Organism unproven.

and in the scabs themselves, even though dried and old. It is improbable that the general body secretions and excretions contain it, save as a result of contamination by the products of its exanthem and its presence in the blood is doubtful. The virus does not pass through colloid filters, but is otherwise filtrable. Prowazek has utilized this fact for the removal of ordinary bacteria by filtration.

Prowazek's bodies.

A second filtration using a colloid filter leaves a residue which should contain the virus. This he has found to consist of very minute, coccoid bodies which multiply by subdivision and when injected into the cornea of a rabbit produce *"Guarnieri's corpuscles"* (*Cytorrhyctes variolæ*) within which the "initial bodies" develop, subdivide and form the small elementary corpuscles (*chlamydozoan bodies*) which he believes to be the cause of the disease. Whether this be true or not, so far as is now known, they occur only in variola and vaccinia, never in varicella.

Age and Sex.—No age and neither sex is exempt, and rarely intrauterine disease may exist if the mother be affected.

Unvaccinated pregnant women apparently are peculiarly susceptible.

* An old writer has said, "From smallpox and love few escape."

† Coincidently Cotton Mather and Dr. Boylston braved adverse public opinion and mob violence in Boston in the same cause.

Morbid Anatomy.—The condition of the patient who has died of smallpox is sufficiently indicated by the symptomatology. The lesions of the viscera are essentially those of profound toxemia and high fever.

Contagiousness.—*The infectious element exists in the secretions, excretions, pustules, and probably in exhalations from the lungs*, and the skin conveys it through the medium of the dried scales which are readily converted into dust. Though contagious at all stages, the frequent exposures without infection indicate that its early periods are *comparatively* innocuous. The contagium is difficult to destroy and remains active for long periods, hence the radical measures employed in the disinfection of rooms in infected houses. The question of conduction through the air is still *sub judice*, but has much to support it. It may be conveyed not only by the clothing, but by room dust, by rats, mice, flies, or vermin, *and the patient ceases to be an infective agent only when the last bit of desquamating skin has been removed. The severity of the disease bears no relation to that of the case from which it is derived.*

Remarkably transmissible.

Early stages slight.

Fomites and carriers.

Individual Susceptibility.—This varies greatly, a few being apparently immune, even though unvaccinated, and those sufficiently vaccinated being either wholly immune or subject only to mild attacks.

Natural and acquired immunity.

Varieties of Smallpox.—There are six distinct forms, varying greatly in severity, namely, *varioloid, discrete, confluent, hemorrhagic, malignant, and verrucose.* Since the Spanish-American war a mild form of smallpox has prevailed in this country and spread widely, especially in country districts, because of its close resemblance to chickenpox, and further because the prolonged immunity resulted in careless disregard of the necessity for vaccination. According to some observers, the light form is gradually becoming more virulent.

Six forms.

Period of Incubation.—From seven to fifteen days.

Characteristic Symptoms.—(*a*) *Sudden onset*, often with chills or, in children, convulsions. (*b*) *Headache*, intense and frontal; this corresponds to the congestion of meninges found at autopsy. (*c*) *Severe backache* (congestion of the membranes of the spinal cord). (*d*) *Vomiting.* (*e*) *Severe pain in the limbs.* (*f*) *High initial temperature* (103° to 105°F.), *followed by a marked recession and a high secondary rise associated with suppuration of the vesicles.*

Tearing backache.

Peculiar fever.

Note.—*The pains are more severe than in any other definitely eruptive disease* and in the presence of an epidemic these symptoms must be given great weight, though they are in no sense indicative of the severity of a given case.

Eruption.—Rarely the disease occurs without any true eruption or a tardy one, and in from 10 to 20 per cent. of the cases misleading initial rashes occur during the first two days. *These assume one of six forms:* (1) *Erythematous.* (2) *Urticarial (very rare).* (3) *Morbilliform.* (4) *Scarlatiniform.* (5) *Purpuric.* (6) *Astacoid (lobster-like).*

Important initial rashes.

The morbilliform and scarlatiniform initial rashes may be extremely misleading and it is said that the sixth variety indicates a fatal issue.

The scarlatiniform eruption is usually distinctly localized and limited to (*a*) *the inner surface of the thighs,* (*b*) *the lateral thoracic areas,* (*c*) *the lower abdomen,* (*d*) *the axilla. It may persist until the appearance of the true rash.*

Localization.

The morbilliform (measles-like) rash usually appears early (second day), lacks the dark, central, raised portion of the true measles macule, the skin feeling smooth, and is fleeting, seldom lasting more than a few hours. It chiefly affects the face and extensor surfaces of the limbs, though occasionally most manifest in the area represented by the triangle (Semon's) formed by two lines passing from between the knees to the respective anterior superior iliac spines and connected by a transverse base line.

Macules, papules, and vesicules (3d-4th day).

The Typical Eruption.—*On the third day of the disease bright red macules appear*, readily blanched by pressure, these rapidly becoming hard elevated "shot-like" papules. *At the end of twenty-four hours* these show *vesicles* and the *vesicular stage reaches its full development* usually by *the fourth day of the eruption.* The vesicles rarely exceed ¼ inch in diameter, contain a milky fluid, and are surrounded by distinct though narrow areolæ. *By the*

FIG. 524.—Confluent smallpox in the unvaccinated adult. (*After Ricketts and Byles.*)

fifth or sixth day depression of the apices of the vesicles is noticeable. This *"umbilication" is usually general* by the end of the first week of the disease (seventh or eighth day). *Almost coincidentally the vesicles become distinctly purulent (pustulation being general from the eighth to the tenth day and associated with a secondary temperature rise to* 102°—104+° *F.). The whole process is completed usually by the end of the twelfth day. By the fourteenth, desquamation should commence and be completed at the end of three or four weeks.*

Umbilication (5th-6th day of rash).

Pustulation (6th-12th day of rash).

Secondary fever.

Parts Affected.—*The eruption chooses by preference the portions exposed to light, chafing, irritation, and injury, and upon them reaches its highest development. Externally it usually first appears on the face and near the border of the hair, yet the mucous membrane may be still earlier involved, the rash appearing on the buccal mucous membrane, tongue, soft palate, pharynx, and even upon the*

First on mucous membranes; next, the face.

stomach and rectum in the virulent cases. In none do the mucous membranes escape. The wrists are also affected early, and, like syphilis but unlike varicella, *it involves the palms of the hands and the soles of the feet*, a point of considerable importance in differential diagnosis. The involvement of the larynx may cause distressing cough and even fatal edema.

Palms affected.

Confluent Smallpox.—In this form all symptoms are intensified and in the stage of pustulation the patient presents a frightful appearance and a foul and *characteristic odor.* The face is a swollen and unrecognizable mass of fetid sores, though on the body and legs the eruption often remains discrete.

Odor.

Hemorrhagic Form (*Purpura variolosa, Black Smallpox, Variola hemorrhagica*).—As indicated by the name, the eruption is distinctly hemorrhagic in type, the patient presenting a frightful appearance. In some cases hemorrhage occurs from any or all mucous membranes and *it has proven fatal in from three to four days after the onset of hemorrhagic symptoms*, though in others of milder type the hemorrhages are limited to the vesicles and pustules.

FIG. 525.—Smallpox. Confluent case in unvaccinated child. (*Courtesy of Mr. Dames.*)

Malignant Smallpox.—This is characterized by *low fever, scant or absent eruption, profound asthenia, and a fatal issue* with or without hemorrhage, death occurring between the third and seventh days.

Variola Verrucosa.—This varies from the other forms only in leaving a warty growth after desiccation and hardly deserves separate classification.

Varioloid.—*This is an attenuated smallpox*, modified by vaccination, natural immunity, or a previous attack. It differs from true variola in the following particulars: (*a*) *Short duration.* (*b*) *Irregular or incomplete eruption which runs a rapid course.* (*c*) *The absence of any marked secondary temperature rise.* (*d*) *The early completion of desquamation.*

Mild symptoms.

The *initial symptoms* are usually *but not necessarily mild* and the eruption may or may not be limited to the face and hands.

It need seldom cause error if one will remember that the genesis, development, and structure of the pustule wherever found is usually typical.

Complications.—These are easily inferred from the symptoms and pathology of the disease, being chiefly connected with the suppuration and pro-

found toxemia. It suffices to name them as follows: edema glottidis, necrosis of cartilages, broncho-pneumonia (sometimes initial), lobar pneumonia (rare), pleurisy (not uncommon), pseudo-angina, diarrhea (especially in children), albuminuria (true nephritis is rare), orchitis, ovaritis, parotitis, abortion, fatal delirium, myelitis, myocarditis, neuritis, pyemic abscess, general tuberculosis, local gangrene, arthritis (suppurative or not), bone necrosis, otitis, iritis, keratitis, purulent conjunctivitis, phlebitis, etc.

The heart muscle suffers severely from the toxemia during the attack, but endocarditis and pericarditis alike are rarely encountered.

FIG. 526.—Smallpox. Soles of the feet in stage of desiccation. (*After Ricketts and Byles.*)

Prognosis.—Absolute figures are out of the question. Assuming a *severe epidemic*, from 25 to 50 per cent. of the unvaccinated will die; among the vaccinated (varioloid) the mortality will vary according to the efficacy of the primary vaccination and its proper repetition (see "Vaccination"), and with the age and physical condition of the person attacked.

In unvaccinated, great mortality.

In vaccinated, negligible.

Disease type, age and physique.

The mortality in all unvaccinated cases is exceptionally great in infants and young children. Efficiently vaccinated persons are practically immune and in

those attacked the mortality should not exceed from five-tenths of 1 *per cent. to* 1 *per cent.*

Of the hemorrhagic and malignant cases, practically all die within the first week, and the same is true of unvaccinated children under one year of age. Dissipation and preëxisting disease increase the virulence of the primary attack and its mortality, and *the more profuse or confluent the facial eruption, the greater is the death rate.* Pitting is unusual nowadays in the ordinary forms. Pitting.

Diagnosis.—*In the presence of an epidemic, all cases presenting extremely severe pain in the head, back and limbs, and vomiting, must at once be under suspicion.* Suspects.

Indeed, lacking an epidemic, they are still important and the greatest care should be observed when such severe symptoms are associated with the suggestive and *peculiarly located initial rashes.* Initial rashes.

The typical eruption is unmistakable and the course of the fever especially significant, viz., appearing on the third day, primarily high and dropping to normal, or, in severe cases, to 100°*F. or* 100.5°*F., only to reappear with pustulation.* Fever.

The greatest stress must be laid upon certain features aiding the recognition of the eruption of smallpox, viz.:

1. *It appears primarily on the face and wrists.*
2. *The face, forearms and wrists are always predominantly affected.*
3. *The trunk is least affected.*
4. *Profuse lesions on the palms of the hands and soles of the feet strongly counts for smallpox as against chickenpox.*
5. *The shape of the smallpox vesicle is rounded or conical and it appears deeply and firmly seated.*
6. *The vesicles do not collapse when pricked.*
7. *True widespread umbilication is characteristic of smallpox.*
8. *The uniformity of the lesions as to stage of development over any given area is of great significance.* (For further differential diagnosis, see "Chickenpox" and "Syphilis.")

Cardinal points in differentiation.

VACCINATION.—The origin of vaccination with cowpox virus as the supplanter of inoculation from smallpox pustules is, as before stated, directly traceable to the keen observation and rare courage of Dr. Edward Jenner, who first proved its efficacy on May 14, 1796. As a result of his brilliant work millions of lives have been saved, and smallpox has lost its terrors in every intelligent and civilized country. Few remain who deny the efficacy of vaccination, but they are of the type who would insist that the sun moves around a stationary earth. *Wherever vaccination is required and* **enforced by law,** *smallpox is a negligible factor.* A boon.

Extent and Duration of the Protective Influence.—It should be clearly understood that to be effective and efficient vaccination must be repeated at certain intervals. *First performed in infancy, it should be repeated at the age of puberty, and always in the presence of any especial liability to exposure.**

* In the old days the greatest mortality occurred in infants and young children, but owing to the general practice of early vaccination they are now the best protected of the age groups.

FIG. 527.—Varicella ("Chicken pox").—The great variation stages of development of the lesions contrasts strongly with the tendency to uniformity in variola. (*After T. F. Ricketts and J. B. Byles.*)

Immunity attained.

The experience of every physician shows *almost absolute immunity* to be the result of intelligent and repeated vaccination however direct the exposure.

Revaccination necessary.

Furthermore, *the disease if contracted by a vaccinated person is almost invariably the mildest possible* (varioloid).

Antivaccinationists.

The false idea that one vaccination in infancy or childhood is absolutely or approximately protective throughout life is altogether too widespread among the laity and much of the prejudice against vaccination, aside from that depending upon pure ignorance, obstinacy, or more or less ingenious misrepresentation, is the result of the vaccination of the earlier days when the virus was usually taken from the vaccinated human being, and the operation performed with scant regard for the danger of septic infection.

The maligned heifer.

The antivaccinationists still talk of the *transmission of syphilis* as if an innocent heifer could acquire and transmit a disease of this peculiar nature.

Vaccination after exposure.

It should be remembered that even though a person has been actually exposed to smallpox, vaccination will ordinarily either confer complete immunity or greatly modify the disease, it being understood that the time elapsing between exposure and development shall have been sufficient to allow of some action on the part of the vaccine. As between cases *of even doubtful vaccination* and the unvaccinated, the ratio of mortality is as one to four.

Preparation of Vaccine.—The material for vaccination is now invariably obtained from the heifer, and the so-called humanized lymph should not be used, except in emergency.

Humanized lymph passe.

Various reliable firms that prepare this substance maintain farms and conduct their inoculations and the recovery of the virus under the most perfect sanitary conditions.

Bovine lymph.

The source of the lymph is the vesicle produced by the inoculation of the udder of the cow. This is allowed to dry on sterilized ivory points or quills, or is treated with glycerine and collected in sterilized glass tubes, so contrived as to permit their use without danger of contamination.

Points and glycerinated lymph.

Method.—The region of the deltoid insertion of the arm or the outer surface of the thigh or calf should be thoroughly cleansed with alcohol (cologne, bay rum) and washed off with boiled water, then lightly but rapidly and multiply *incised*, not scarified, with a very sharp blade (the blades of "safety" razors serve admirably) in such a manner as will carry the cut into the cutis and yet produce no free bleeding, which *must* be avoided.

Vaccination technic.

The procedure is practically painless if properly performed. The vaccine is placed upon and gently rubbed into the prepared area, allowed to dry thoroughly and afterward the affected portion is covered with one of the numerous forms of "shields" readily obtained at any drug store.*

Shields.

A very neat, but less certain procedure, consists in the *intradermic* introduction of glycerinated lymph with a sterile hypodermic needle.

All vaccinated persons should be warned of the danger of conveying virus from the point of vaccination to other portions of the body.

Sequence of Events in Vaccination.—A *reddish papule should make its appearance in from three to five days, promptly becoming vesicular, filled with*

* According to the author's experience, these, if properly constructed and firmly anchored by accessory *short* strips of adhesive plaster, are far better than a gauze dressing.

clear lymph and later umbilicated. Its maximum development is reached at the end of seven or ten days, when its contents become purulent in appearance.

A brilliant areola surrounds the pustule, underlain by a tender and more or less firm or brawny area. During the succeeding three or four days pain and tenderness become localized along the region of the lymphatics and their tributary glands may be slightly or decidedly swollen and inflamed, with fever and malaise.* The itching is intense, and scratching frequently causes secondary infection, hence in infants the movement of the arms must often be restrained. *Occasionally a general eruption closely resembling chickenpox may appear or there may be a distinct roseola.* In from *ten days to two weeks* these symptoms disappear, *desiccation being complete, as a rule, by the end of the fifteenth* or *sixteenth* day, *the scab falling about a week later.* A depressed pitted scar is left behind varying in size with the severity of the process. All sorts of variations may be met with, such as long periods of incubation, entire absence of constitutional symptoms, or, in some instances, a more prolonged and severe stage of suppuration. *A thoroughly good vaccination* usually *leaves a scar which persists throughout the whole life of the individual.* In an unvaccinated person one should never rest content with a single vaccination, and in the presence of direct opportunity for infection or during an epidemic it is wise to repeat every vaccination if the first fails. It would appear certain, moreover, that cases presenting unusually severe symptoms, owe these rather to secondary infection than to the true virus, so that some severe "takes" are less protective than the thoroughly typical and milder form.

Duration of scar.

Pseudo "takes."

The Previous Health.—Common sense should determine the propriety of vaccination in exceptionally delicate and diseased children, and a proper period chosen. As to the site of the vaccination, the female infant or adult should, as a rule, be vaccinated on the leg, the male on the arm, the resulting scar being of no moment to the latter. One should always inquire as to the right- or left-handedness of the male candidate, and vaccinate him on the less useful arm to ameliorate so far as possible the discomfort and inconvenience of any temporary disability.

Site of vaccination

VARICELLA

(*Chickenpox*)

Definition.—*A trifling infection of unknown causation, characterized by transient fever and a vesicular eruption, but absolutely unrelated to smallpox.*

Etiology.—Children are peculiarly susceptible between the ages of one and eight years, adults remarkably resistant. It prevails chiefly in the spring and autumn, spreads rapidly, and one attack usually protects for life.

* The signs of inflammation and glandular abscess are often far more alarming than the actual result merits, but, rarely, septic conditions are encountered. In an experience covering nearly two decades and including two years of municipal service the author has never had or seen one case of death, deformity, paralysis, or invalidism, rightly attributable to modern vaccination, and is convinced that the few bad results reported are almost invariably due to previously existing syphilitic or tuberculous taint, or lymphatism.

Symptoms.—In from one to two weeks, rarely three or even four, after exposure the symptoms of mild infection appear.

Trivial symptoms.

Malaise, chilliness, pain in the legs and back are associated with a mild fever, rarely exceeding 101° *to* 102°*F. Some children show no fever or signs of illness save the eruption which appears within twenty-four hours in the form of minute, irregularly placed papules changing in a few hours to vesicles.*

Vesicular eruption (2nd day).

Sites of election.

The chest and back are first attacked, and it then spreads to the face, scalp, neck and extremities. It is especially well marked on the back, which usually shows the oldest lesions, and is attended by a troublesome itching. *The forearms, wrists and hands are usually but slightly involved.* Roseolar forms of brief duration and a pemphigoid type are seen rarely.

Differential Points.—*The following points serve to distinguish varicella from smallpox or varioloid*: (*a*) *In chickenpox but few or no vesicles in a given case umbilicate.* (*b*) *The contents are serous, not purulent, though a few may suppurate and leave pits.* (*c*) *The unilocular vesicles are emptied by a single puncture.* (*d*) *They rarely have an areola.* (*e*) *They come in successive crops lasting two or three days and form superficial crusts which desquamate in a week or ten days.* (*f*) **The vesicles, therefore, are seen in all stages, i.e., the eruption is not uniform.** (*g*) *The spots may be few in number, are scattered widely and do not tend to become confluent.* (*h*) *The secondary fever of suppuration is lacking.* (*i*) *Vesicles rarely appear in any considerable number upon the palms or soles.* (*j*) *Varicella is relatively rare in the adult.* (*k*) *But two or three hours are required for the passage from papule to vesicle.* (*l*) *Guarnieri's bodies are lacking.** (*m*) *The order of involvement and its extent are the exact opposites of smallpox* (see also "Smallpox").

Duration.—A week to two weeks.

Prognosis.—Almost invariably good.

Complications.—Ordinarily lacking and seldom serious. The eruption occasionally appears in the mouth and hard palate, but rarely or never involves the conjunctivæ, larynx, or trachea. The vesicles always variable in size, in rare instances become bullæ, and in any case the otherwise harmless vesicles may be infected by scratching. *Cachectic children in this as in other infections may present a peculiarly virulent and profuse eruption.*

WHOOPING COUGH

(*Pertussis*)

Definition.—An infectious disease of childhood caused, probably, by the Bordet-Gengou bacillus, self-limited and immunity conferring, characterized by peculiarly violent paroxysms of cough, *followed by a "whooping sound."*

Contagious in all stages.

The disease is highly contagious, especially before the appearance of the characteristic paroxysms,† but adults enjoy comparative immunity. Infants

* Inoculation of the rabbit's cornea is required for their production in variola or vaccinia

† It is *comparatively* innocuous later, but still contagious, and great individual variations in this respect are found to exist.

Age.

and very young children (six months to five years of age) are peculiarly susceptible, and at all ages female predominance is evident. It is a disease chiefly of early spring.

Still undetermined.

Etiology.—An influenza-like bacillus is believed by Bordet and Gengou to be the causative agent. It is Gram-negative, is grown with some difficulty on potato blood agar and its colonies possess a peculiarly tenacious, mucoid character.

Unlike the influenza bacillus which it strongly resembles it is never intracellular. Its discoverers reported that it gave the complement fixation test and Klimenko and Fränkel have produced the disease in monkeys by inoculation. On the other hand these reports are not completely confirmed and the organism is not yet fully accepted as the causative agent.

Mallory, Homer and Henderson believe that the characteristic lesion consists of masses of the bacilli lying between and adhering to the cilia of the trachea and bronchi.

Incubation.—Two days to two weeks.

Duration.—Eight to ten weeks.

Prognosis.—Good in robust young children, bad in weaklings, nurslings, and the aged.

Morbid Anatomy.—Practically none save that of any complicating diffuse bronchitis, broncho-pneumonia, emphysema, or cardiac overstrain. Death by inanition, starvation, and inhalation or deglutition broncho-pneumonias are rare phenomena.

Peculiar cough.

SYMPTOMS.—*A coryza or mild bronchitis* is associated with a series of short barking *coughs, progressively urgent and explosive* in character, the protracted expiratory efforts resulting in glottis spasm and a terminal long-drawn stridulous inspiration, the "whoop." The seizures vary in severity and frequency, and *absence of the whoop may obscure diagnosis, but the cough is peculiarly explosive and paroxysmal. Attacks may be almost constant or occur but once or twice a day,* the average being 15 to 20.

"Whoop" may be absent.

Paroxysms easily excited.

Any emotion, pleasurable or otherwise, food, dust, overheating, or a cold draught may provoke the dreaded paroxysm. The child may vomit, the urine or feces may be passed involuntarily, hernia may be produced, rectal prolapse may appear and hemorrhages may occur from the nose, ears, or under the skin or conjunctiva.

Glands.—A general though moderate enlargement of the lymph nodes is present, especially marked in the peri-bronchial and cervical regions.

Lymphocytosis.

The blood shows a decided primary leucocytosis,* later a lymphocytosis and finally an eosinophilia (Barach).

The lymphocytosis begins before, and reaches its height during, the paroxysmal stage. The primary increase is in the small mononuclears, later the large mononuclears show a decided or proportionate increase.

* Counts of 85,000 are reported for the primary stage but the average is 20,000+ and some observers give much lower figures. In the acute stage with pneumonic complications counts of nearly 200,000 are reported. A blood count may or may not aid diagnosis, but should always be made in doubtful cases.

The urine may contain a trace of albumin and *fever* may be present or absent.

Diagnosis.—The presence of an epidemic, decided leucocytosis or lymphocytosis, a persistent bronchitis, and the paroxysm of urgent, short, barking, rapidly repeated coughs with or without the whoop is sufficient usually for diagnosis.

In the case of a child any cough which persists and shows a tendency to nocturnal urgency of a spasmodic character, should excite suspicion.

In any case, the question of an existing "*spasmophilia*" must be considered for two reasons, viz., first, the ailment may be merely an intensified cough of non-specific origin and, second, the spasmophilic state may greatly increase the frequency of seizures in true pertussis. (See "Spasmophilia.")

Complications.—There are many. It is frequently associated with measles, with subcutaneous, interstitial and true pulmonary emphysema, pneumothorax, persistent vomiting leading to inanition, *cardiac dilatation*, enlargement of the bronchial glands, general convulsions, cerebral hemorrhage, *broncho-pneumonia*, and lobar pneumonia, or even pulmonary tuberculosis. *Adenoids* greatly increase its dangers and add to the severity of the paroxysms.

Many and grave.

Adenoids.

Comment.—*The laity regard pertussis too lightly. It is a disease of high mortality through its pneumonic complications, often induced by a foolish disregard of ordinary precautions. Of children under one year, 25 per cent. die. Between one and two years, 15 per cent. Above these ages the mortality is slight, but it will be seen that the disease is formidable and deadly if lightly regarded. Among the ignorant and poor attending the public services of great cities it stands second to pneumonia as a cause of death in infants.*

A formidable disease.

Infant mortality high.

SYPHILIS

("*Lues venerea*," "*Pox*")

Etiology.—*The Treponema pallidum* of Schaudinn and Hoffman (1905) is now fully accepted as the proven causative organism on the basis of Noguchi's brilliant work fulfilling the requirements of Koch's law, and has been found in practically all lesions from the primary sore to the gumma, as well as in the circulating blood. The germs abound in congenital syphilis and are in general most abundant in the lesions of greatest virulence, though radical antisyphilitic treatment usually causes their prompt diminution or apparent disappearance.

Smear Preparations.—With dark field illumination (using the ultracondenser) and in salt solution the germ and its motility may be studied without staining, but usually (*a*) the smear is dried in the air and placed in absolute alcohol for one hour. (*b*) To each drop of a modified Giemsa stain* 1 c.c. of water is added, and in this staining fluid the specimens remain

* Giemsa-Lösung für die Romanowsky Färbung, put up by, Grübler.

from one-half to twenty-four hours, the longer period being more reliable. In a properly stained specimen the organism is violet and the nuclei of any leucocytes present a blackish-red. In making smears from a primary sore, vigorous scrubbing with soap and water should precede a slight curettement.

The Spirocheta refringens is often encountered in large numbers in all superficial lesions, in contrast to the *Treponema pallidum*,* which seldom exceeds two or three to the field. The former is highly refractile, broader, readily takes a deep stain, and its spirals are not typically corkscrew-shaped, but undulant.

FIG. 528.—Treponema Pallidum (in center) and the Spirocheta refringens.

Readily transmitted.

Mode of Conveyance.—Syphilis in an active form may be, and usually is acquired, less often inherited. Dilute inherited syphilitic infection is of course well-nigh universal, but is for the greater part symptomless.

Protean.

It is both contagious and infectious, confers almost absolute life-long immunity, and furthermore is capable of affecting each and every structure of the human body so diversely as to make it the chief of protean diseases.

Essentially venereal.

Given an abraded skin or mucous membrane in a non-immune and contact with the secretions or blood of a syphilitic, infection almost certainly follows. Less frequently, prolonged contact or retained virus alone may produce it on a *sound mucous membrane*. The disease is essentially venereal, sexual intercourse being the means of transmission and sexual organs the usual seat of primary lesions. Yet owing to the irregular and unusual forms of intercourse and the communicability of the disease through certain secondary lesions and by means of intermediate substances, we find labial, lingual, tonsillar, mammillary, and rectal infections as well as the digital sore not infrequent in physicians, dentists, laundresses, rag pickers, etc. It follows also that syphilis may be innocently acquired with or without sexual congress. Husbands infect innocent wives and less often a guilty wife transfers the disease of her lover to an unsuspecting husband. The innocent girl carrying an oral chancre for which the mucous patches of her fiancé are responsible is no rare client and may infect other family members. *The individual syphilitic is a menace to all about him during the active stages, as the secretions of his lesions, his blood and his lymph, are alike virulent.* Fortunately breast milk, saliva, and usually the semen are not infectious unless contaminated through local sources. The tertiary lesions are seldom sources of infection unless relatively early or co-existent with late secondary lesions. High

Extra-genital infection.

The "syphilitic" a menace.

Tertiary lesions not often contagious.

* The "treponema" of syphilis was first called *Spirocheta pallida*, but it was found that this name had previously been applied to another organism by Ehrenberg.

degrees of heat or cold seem to destroy the virulence of the secretion, otherwise active even in a state of desiccation.

Modifying Influences.—As to its later course no correct inferences can be drawn from the primary symptoms or the site of the lesion, *though the general rule governing all infections which leads us to expect the severer manifestations in persons of bad habits, poor physique or a vitality impaired by antecedent or co-existent chronic disease holds good in this one.* The ravages of *acquired* syphilis in children are exceptionally violent and widespread.

Many factors.

Acquired lues in children.

The Three Stages.—*The primary stage of syphilis comprises the time elapsing between the recognition of a local lesion at the site of infection and the appearance of the constitutional symptoms and eruption of the secondary period. It is represented by the development of the chancre and coincident swelling of the adjacent lymph glands (syphilitic buboes).*

The third stage or tertiary period marked by soft nodular growths of peculiar structure (gummata) may be entirely lacking in efficiently treated cases or may appear despite the most active and prolonged specific medication.

Extending beyond these circumscribed and classified periods is an *insidious larval activity of indefinite or life-long duration* which makes the individual liable to develop degenerative diseases of an apparently non-syphilitic nature. Such are locomotor ataxia, paretic dementia, arteriosclerosis, aortitis, apoplexies, aneurysm, and the nephritides.

After effects.

This fact has led life-insurance companies to exact a long period of immunity and evidence of radical and efficient treatment as prerequisite to acceptance as "first-class risks," yet the actuarial report of the "combined investigation" committee shows the futility of the requirement by the enormously excessive death rate experienced. (See "Cardiovascular Syphilis.")

Insurance experience.

The Initial Lesion or Chancre and the Bullet Bubo.—After an incubation period of from ten days to six weeks the first characteristic symptom is apparent in a *single papule* often a part of an *herpetic eruption* at the *point of infection,* whether genital or remote, which increases in size, becomes *indurated,* and may remain as a dry plaque or show an eroded surface with scant secretion.* The feel is that of a split pea, save that in certain cases, especially in the female, the induration may be lacking. Its usual location is the vulva or the prepuce, but it may appear upon any portion of the genitals or in neighboring or distant parts *and always represents the actual site of infection.* The glands representing the lymph drainage of the affected part undergo a painless enlargement, known, when in the groin, as the *bullet bubo.*

Incubation.

Papule or erosion.

Induration.

Bubo.

The Secondary Stage—*Within from six to eight weeks after infection systemic infection is manifested by general glandular swelling, mental depression or irritability, insomnia, malaise, pallor, pain on pressure over or near the sternochondral joints, neuralgia, nocturnal headache, joint pains and exertion-dyspnea, fever being rare or, if present, trivial. As these subside the exanthem develops.*

Constitutional symptoms.

* Multiple lesions are not uncommon in women, but are relatively rare in men, and sometimes, "satellite" indurations of later formation may be observed.

Exanthem.

Exanthemata of the Secondary Period.—Their characteristics must be thoroughly understood because of their great importance in diagnosis. These are: (*a*) *Their polymorphous tendency.* This may merely represent different stages in the development of a certain type or show a true mixed eruption. *Such polymorphism is the rule in syphilis.* (*b*) *Symmetry and tendency to general distribution.* (*c*) *The tendency to follow skin lines in arrangement.* As is seen especially in profuse eruptions and particularly in the oblique parallel lines of eruption from the back downward obliquely to the front. (*d*) *The absence or trivial degree of itching.* (*e*) *The tendency to oval elliptical or circular forms.* (*f*) *The prompt response to mercurial inunctions or more active treatment.* (*g*) *The tendency to shift their pigment to the periphery of the lesion** is characteristic especially in the case of disappearing or past lesions. In the former a central faint red may be surrounded by a lighter area, in its turn encircled by a deeply pigmented border. In the latter the whole inner area is white (syphilitic leukoplasia).

Peculiarities must be known.

Early eruptions.

Varieties of Syphilitic Exanthemata.—*The earlier syphilitic eruptions show a marked tendency to predominant involvement of the abdomen, front of chest, and anterior surface of arms.*

Earliest form often overlooked.

Roseola.—*This, the earliest form, chiefly affects the trunk and is so evanescent* and otherwise symptomless as, in many instances, to escape the patient's notice.† The *brownish-red pea-sized macules* are seldom raised above skin level and leave no trace.

Readily recognized.

Large Macular Syphilide.—Unlike roseola this is due to actual round-cell infiltration, *is usually slightly raised* and *frequently, by central fading or border coalescence, forms the varieties known as "gyrate," "circinate," "annular," etc.* The *color* is *livid* in the lower extremities, *copper color* in the upper, and its pigmentation may persist for considerable periods or give place to the white areas known as cutaneous leukoplasia.

Commonest type.

The Papular Syphilide.—*This may be pure or mixed with the macular or pustular form and is the commonest syphilide and the most diverse in size and orm.* It may be distinctly papular, nodular, or flattened, and in size varies rom a pinhead to 2 cm. or more. *Its distribution may be general or in later types limited to the genitals, palms, soles, or other areas.*‡ Resembling psoriasis in some respects, it lacks the silvery scales and peculiar localization of that disease and seldom involves large areas, occurring usually in small patches.

Distribution.

Lenticular.

The lenticular form is a rash of brief (seven to fourteen days) duration followed by desquamation and leaving no permanent markings. Its nodules are grouped and especially involve the extremities and trunk.

Lichenoid.

The flat, glistening form involves especially the face and is *sharply defined, flattened, and centrally depressed. It also leaves no permanent marks. The lichenoid papules are grouped, not generally distributed, persistent, become crusted*

* The *coppery or ham-colored eruptions*, though extremely suggestive, are neither invariable in nor are they peculiar to syphilis.

† Dashing ether upon the surface of the body will often bring out a phantom-like, but clearly specific roseola.

‡ Any papular or pustular form may be hemorrhagic in grave or complicated cases.

and leave tiny pits. Among the later forms are the *orbicular papules* which show a *ring-shaped border and central depression*, and chiefly affect the genital region. The *tubercular form*, very *late* and *persistent and closely allied to tertiary eruptions*, is *grouped, scaly or crusted, richly pigmented, and may terminate in ulceration which leaves permanent scars.* Tubercular.

The Pustular Syphilides.—These are *rarely early but may co-exist with the papular form and are associated with marked general symptoms* (fever, malaise, pallor, etc.). *The exanthem may closely resemble acne or even smallpox* at the outset, and *may form extensive crusts. Ulceration, superficial and spreading (echthyma syphilitica) or deeper*, may occur and the crusts may so arrange themselves as to be termed syphilitic rupia. *They leave peripherally pigmented, smooth, shiny, loose scars. Hemorrhagic forms of* syphilides occur and mean a serious case as in other exanthematous diseases. Suggests variola. Hemorrhagic types.

Palmar Syphilides.—Both the palms and soles may be involved in the late secondary papular syphilides and *this eruption is of almost pathognomonic significance.** If these papules form between the fingers or toes the result is maceration and oftentimes decided inflammation, and if the matrix of the nail or its margin be attacked *syphilitic onychia* is produced, the nail becoming dead and being finally cast off. There is frequently a *perionychia* due to suppuration at the nail margin. Onychia and perionychia.

Indurative Edema.—On and near the genitals the secondary processes may assume a most severe type of *excoriation, inflammation, and even indurative edema*, and *these lesions are highly contagious* and probably the most frequent source of infection. *Fissures, ulcers*, and *venereal papillomata* are associated with this condition. Excoriations. Fissures, ulcers and warts.

The Hair.—Syphilis of the scalp commonly takes the form of *pustules* which by coalescence may produce *more or less irregular or scattered spots of baldness*, sometimes replaced by gray hair, but usually, under efficient treatment, promptly taking on a normal new growth. A less common form is the *seborrheic, which leads to diffuse baldness.* Baldness.

The Buccal Cavity.—This region is one of the most important in connection with the diagnosis of syphilis in its secondary stage. The earliest change in the tongue consists of *prominence of the papillæ with small spots of whitish exudate; these areas later becoming raw and glistening, and, at the edge, converted into ulcers.* Many variations occur in the form and distribution of such lesions. Tongue. "Mucous patches."

The faucial pillars, tonsils, and soft palate should always be inspected for such "mucous patches," as well as the lips and inner aspect of cheeks where fissures and ulcers may be detected. Indeed the kidney-shaped, gray-bordered ulcer of the tonsil is one of the earliest secondary manifestations, and may be painless and so escape the patient's notice. Important regions.

Tertiary Syphilis.—*This is essentially the gummatous stage and should never appear in a marked form in those of good constitution who have received* May never appear.

*The late Mr. Berkeley Hill used to say that he always taught his *students* that any eruption in the palm *was syphilis*, and the exceptions are so few, aside from smallpox, that the dogmatic statement was justifiable.

adequate treatment, though in those less fortunate it assumes the most terrible types of destructive lesions, *affecting the osseous as well as the soft tissues* and leading to frightful suffering and humiliating deformity. *Large pustules* may appear in this stage (pustula major) with rapid necrosis and coalescence, foul discharge and severe pain, or the various *gummatous neoplasms* may develop in various parts of the body, *internal and external*.

May cause destructive lesions.

Cutaneous.

The *cutaneous gumma varies in size from a buckshot to a mandarin orange. It is somewhat soft in consistency and at first spares the true skin; the tendency, however, is to disintegration which may or may not involve the skin according to the deep or superficial situation of the growth.* In certain instances the peculiar *serpigenous ulceration* so characteristic of the disease may appear and leave its characteristic *sinuous, reniform* or *semicircular scars*. The gummata may involve the *bone, periosteum, muscles, joints, or tendons, the painful swellings of which are common in the earlier stages*, and if the inflammation be severe may result in fibrous ankylosis.

Serpigenous ulceration.

Osseous, myositic and arthritic forms

Syphilis of the stomach has already been referred to, *the soft palate is often affected* at its junction with the bony portion, the process tending to *a pathognomonic perforation*; or the *hard palate* itself may be involved. Indeed, *space will not permit a description of the gummatous changes, inasmuch as they may involve any region of the body and are dealt with in other sections.*

Soft and hard palate.

Syphilis of the Respiratory Tract.—The discovery of the causative organisms of syphilis and the studies bearing upon its persistence and localization have greatly broadened our knowledge of its etiologic possibilities.

Formerly syphilis of the respiratory tract was a somewhat dubious entity when one went below the larynx.

In this region the congestion attending the secondary eruption, the occasional occurrence of mucous patches, and the occurrence of gummata were quite readily recognized and accepted.

Involvement of the trachea alone is one of the rarest of syphilitic manifestations. In its tertiary form it may result in partial or even complete tracheal stenosis, and the same change may occur in the large bronchi when the seat of gummatous invasion, ulceration and healing with resulting scar contraction.

In the lungs, luetic involvement is rare, though less so than was believed formerly.

Aside from the so-called "white pneumonia" of the luetic fetus or infant little is known of the lung changes due to the congenital form.

Acquired syphilis of the lung may be gummatous, take the common form of a fibroid induration or show catarrhal changes in association with induration.

"Syphilitic phthisis" is the term applied to those rare cases of pulmonary syphilis in which gummata have broken down and cavities resulted.

There are practically no distinctive signs or symptoms in any way characteristic of syphilitic involvement of the respiratory tract, but one should bear in mind its possibilities in confusing the clinician and leading

him to wrong diagnostic conclusions as to the specific etiologic factor present in a given case.

The Wassermann test is helpful and the old therapeutic test invaluable.

The Lymph Glands.—*A general glandular hyperplasia characterizes the secondary stage* and may be exaggerated in any group of glands which drains an ulcerating surface. *The post-cervical, inguinal, axillary, epicondylar, and posterior mastoid glands* are especially affected and are easily palpated. Occasionally a similar glandular swelling occurs in a tertiary period, not infrequently ending in necrosis. *The tongue* may be the seat of tertiary as well as secondary changes in the form of *leukoplasia or gummata*, the latter of which may produce painful or permanent *fissures and deforming scars*, as the result of ulceration. The so-called *syphilitic psoriasis* as affecting either the tongue or buccal mucous membrane is recognized by the presence of *whitish patches which are hard and horny* to the touch. *These are easily eroded and constitute an intractable and painful tertiary lesion.*

Glands most accessible.

Tongue.

Buccal membrane.

Hereditary Syphilis.—Little need be said concerning the appearances in this condition beyond that contained in the first section of the book. The unfortunate inheritors suffer especially from the following lesions: (*a*) *Syphilitic hemorrhage of the new-born* leading to death shortly after delivery. (*b*) *Ulceration and hemorrhage from the navel*, usually uncontrollable and resulting in death. (*c*) *Snuffles.* The nose is blocked by syphilitic inflammation and there is usually an *excoriating secretion* and a *specific periostitis which may produce saddlenose.* (*d*) *Excoriations, due to maceration of papular and pustular* eruptions *about the nates.* (*e*) *Marked nutritive disturbances* which give to the child a *wizened, senile appearance*, the skin being dull, inelastic, and often hanging in folds. (*f*) *Pemphigoid lesions, which occur early in infancy* (*the first few days*) *and usually justify a fatal prognosis.*

Pathognomonic signs

"Snuffles."

"Sore bottom."

Senile physiognomy.

In addition one finds enlargement of the liver and spleen, epiphyseal inflammation and even separation, iritis, keratitis, conjunctivitis, etc.

Syphilis Hereditaria Tarda.—*This term is applied to those forms of hereditary syphilis which appear about the fifth year or sometimes as late as the twelfth and tend to last until the age of twenty or twenty-one. The Hutchinsonian syndrome is:* a hazy cornea or actual interstitial keratitis, increasing deafness, and crescentically notched, prominent upper incisors. To these Mracek would add a flat or depressed nose, fine scars at the angle of the mouth, on the upper lip, or on the mucous membrane of the lips, *radiating* from the nares, and prominent frontal bosses, together with a manifest arrest of development.

Jonathan Hutchinson's syndrome.

Prognosis.—Recent developments, especially such as relate to the frequency of occurrence of living specific syphilitic organisms in many chronic diseases both of the nervous and the cardiovascular systems, show clearly that complete cure is relatively rare.

Undoubtedly the most effective therapy is that which is both early and intensive, preferably instituted before "secondaries" develop.

It should be noted that there is a growing belief that mercury remains the most potent and reliable of single remedies. This does not preclude the use of arsphenamin which has certain advantages associated with its rapid action, and no doubt both may be employed with advantage, and that one supplements the other.

In any event, treatment must be continued until the Wassermann reaction is negative and the patient's blood must be tested at regular intervals over a period of not less than two years. Its reappearance calls for the immediate resumption of therapeutic measures.

Therapeutic test.

Specific tests.

Comment.—*The student must remember that syphilis is the most protean of diseases and frequently assumes forms unrecognizable by any save the Wassermann, luetin, and therapeutic, tests. Whether the patient be infant or adult, male or female, cleric or convict, as a possibility, it always exists, for the innocent suffer even as the guilty, though less often, and the physical results of the errors of youth cannot always be obliterated.*

Important rule.

In this connection it may be said that when one encounters conditions peculiarly baffling and indeterminate in their symptomatology, syphilis, disturbances of the internal secretions, or drug addiction should always be considered.

ULCERATING VENEREAL GRANULOMA

Definition.—A superficial extremely chronic ulceration, occurring chiefly in the tropics, almost invariably genital in site, transmitted, probably by sexual intercourse, and frequently associated with the presence in the lesions of a spirochete closely resembling *Treponema pallidum.*

FIG. 529.—*Granuloma venereum.* (*After Wenditandt.*)

Symptoms.—The appearance of a papule on the penis or labia minora, unattended by constitutional symptoms, is followed by superficial painless erosion, exposing a sluggish granulating surface, which yields a profuse, foul smelling discharge.

Cicatrization of old areas may attend the extension of the process.

The superficial painless erosion or ulceration may or may not extend to the groins and inner surface of the thigh and, rarely, is destructive.

It shows a decided tendency to excessive chronicity and extensive scarring, affecting the general health but little or not at all, and shows no tendency to glandular changes. It does not respond to anti-syphilitic treatment and the Wassermann test is negative.

YAWS (*"Frambesia"*).—A tropical disease due to *Treponema pertenue*

FIG. 530.—Treponema pertenue. The cause of yaws (*Frambesia*). (*After Mühlens.*)

FIG. 531.—Yaws.—Initial lesion on breast of mother. General infection (secondary stage) in child. (*After Henggeler.*)

Three stages.

(Castellani) and characterized by papular, tubercular and ulcerative skin lesions, appearing in three stages like syphilis, the tertiary phase, only, producing the deeper ulcerative lesions.

Incubation.

Initial Lesion.—After a period of incubation averaging three weeks, nocturnal headache, dyspeptic symptoms, joint pains and an irregular fever usher in the primary effect in the form of single or multiple papules at the site of inoculation. Superficial fissuring ulcerations follow, exposing a raspberry-like base of sluggish fungoid granulation (the "mother yaw") exuding a seropurulent discharge.

The "Mother" yaw.

Lymph nodes.

Tenderness and enlargement of the lymph nodes may accompany or shortly precede the appearance of the primary lesion which is almost invariably extragenital.

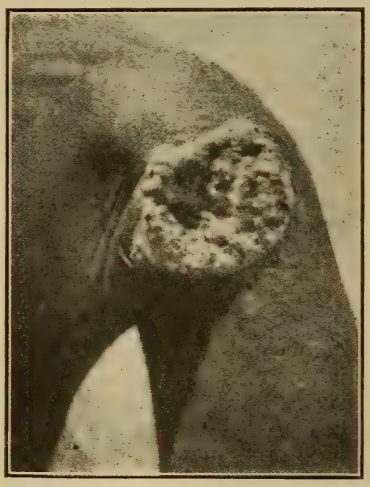


Fig. 532.—Yaws. Tertiary ulcer. (*After Bärmann.*)

Fig. 533.—Yaws. Note tubercles in face and around mouth. (*Stitt's "Tropical Diseases."*)

The "mother yaw" may desiccate and disappear or, more commonly, persist as a fungous mass of exuberant granulation tissue.

Fever and eruption.

Secondary Stage.—In from six to twelve weeks after the coming of the initial lesion, another attack of fever and malaise appears, and a generalized eruption of papules *similar both in character and course to the primary lesion* becomes manifest. This involves by preference the junctional tissue connecting the skin and the mucous membranes (*Stitt*), the papules and tubercles appearing chiefly around the nose, forehead, mouth and anus, plentifully over the neck, anus, legs and buttocks, sparsely over the trunk and scalp.

Preferential sites.

This secondary stage lasts for several months or years, the tubercles appearing in successive crops.

Tertiary Stage.—This is not constant in appearance but represents the one period of yaws characterized by the formation of gummatous nodules and *deep* ulceration.

In no stage are the viscera affected.

Mortality.—There is almost no mortality, ½ per cent., and the disease chiefly attacks children and confers immunity.

FIG. 534.—Yaws. Note somewhat unusual trunk involvement. (*Stitt.*)

Differential Diagnosis.—The following points have seemed to disprove the apparently reasonable assumption that yaws is syphilis.

1. An individual suffering from yaws may contract syphilis.

2. A person suffering from "syphilis" may contract and pass through typical yaws.

3. The primary and secondary eruptions are identical in yaws, not so in syphilis.

4. Certain distinct morphological differences obtain as between *Treponema pallida* and *Treponema pertenue.*

5. Unlike syphilis yaws does not affect the viscera or the mucous membranes.

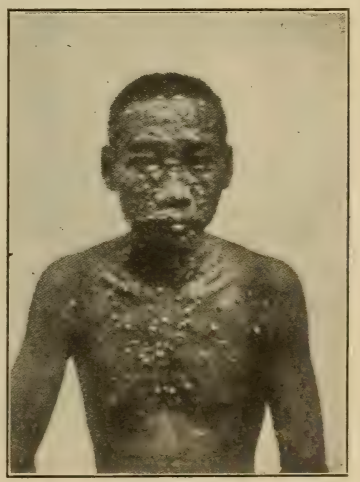


FIG. 535.—Yaws.—Secondary lesions. (*After Henggeler.*)

6. Unlike syphilis yaws does not affect the central nervous systems.

7. The primary lesion of yaws is *almost invariably* extragenital.

Reasons for believing yaws an attenuated syphilis:

1. *Monkeys* rendered immune to syphilis are immune to yaws, but yaws infected monkeys are not immune to syphilis (*Levaditi* and *Larrier*).

2. Salvarsan is more specific for yaws than for syphilis.

3. Gangosa, probably a tertiary yaws, gives nearly 70 per cent. positive luetin reactions and yaws yields a higher percentage of positive Wassermann tests than does syphilis.

FIG. 536.—Gangosa. Cases from Island of Guam (U. S. Naval Bulletin). (*Stitt.*)

Despite the authoritative opinion held against the identity of the two diseases, it would seem possible that with better knowledge the future may reverse this decision.

GANGOSA (*"Muffled Voice"*).—This disease is probably merely a tertiary form of yaws, nearly all patients showing scars of that disease or admitting past infection.

In this ailment or stage the mucous membranes are viciously attacked.

Preferentially the destructive process first attacks the soft palate, invades the floor of the nasal passages, the nasal septum and the fleshy and cartilaginous structures of the nose itself, often destroying the eye.

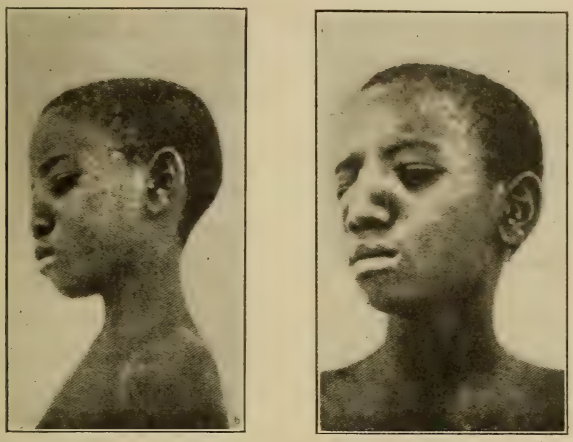


FIG. 537.—*Goundu.* A curious disease of the Tropics called "*big nose*" in the districts harboring it. Apparently it is nothing more than an osteoplastic periostitis due perhaps to yaws. The affection is painful at the onset and attended by purulent nasal discharge. After six or eight months this ceases and painless tumors result which usually attain the size of half a hen's egg, but may become so large as to interfere with, or even destroy the eyes. (*Manson.*)

FIG. 538.—*Ainhum.* This tropical disease of unknown origin begins as a constricting fissure, around one or more of the toes, which increases its pressure gradually until actual amputation results, with or without attending ulceration.

During the course of the destructive ulceration the breath is foul and repulsive.

Gangosa is extraordinarily responsive to anti-syphilitic medication whether this be salvarsan or mercury, and about 70 per cent. react positively to a luetin test.

ERYSIPELAS

Definition.—*An acute infectious and contagious disease due to a strain of the streptococcus pyogenes* and characterized by lymph tract extension and a spreading dermatitis associated with marked toxemia.*

Syphilitics.

Etiology.—The disease is particularly common and easily induced in women after delivery, in cases of surgical operation and in elderly patients or those debilitated by chronic disease. Imperfectly treated syphilitics are especially liable and may suffer excessively.† In no disease is recurrence more commonly observed.

Symptoms.—*After an incubation period* varying from one day to a week and with or without *slight malaise, tingling or burning* of the affected surface, there is *chilliness or an actual rigor and a high, somewhat irregular and remittent fever* (103° to 105°F.) associated with *marked leucocytosis.*

Pathognomonic signs.

The affected skin is dull crimson, swollen, tense, presents a raised margin at the line of advance and later may become covered with tiny or large blebs.

It tends to spread most readily over the regions covered loosely by integument, and although the face is its favorite seat, it but seldom extends to the scalp. *Per contra*, the eyelids and ears may become so enormously swollen and edematous as to produce a superficial necrosis and the same is true of the scrotal tissues. Gangrene is not an uncommon complication in old people.

In favorable cases it remains but a few days, gradually fades and is followed by desquamation, the fever ending usually by crisis. The neighboring lymph glands are moderately enlarged during the attack. *Relapses are common* and in unfavorable cases the process may shift its seat and a typhoid state may develop.

Differential Diagnosis.—The author knows of no other disease that

Usually simple.

presents these symptoms save primary phlegmon of a mucous membrane, which may so closely resemble erysipelatous infection as to make primary differentiation difficult or impossible.

The rare and curious ailment described by Rosenbach under the name of "*Erysipeloid*"‡ presents the same outward appearance but lacks febrile manifestations and is extremely sluggish in its extension. It should be re-

Obscure cases.

membered that the reddening may be misleadingly slight and pale in profoundly cachectic patients and afebrile cases doubtless occur. In an advancing process the older areas fade gradually after one or two days.

The characteristic raised border soon declares itself in almost every instance. The diffuse redness of ordinary erythema or of phlegmon, therefore, stand apart clearly.

* Fehlheisen's *streptococcus erysipelatis* has been conclusively proven to be identical with the S. pyogenes.

† In cases of imperfectly treated syphilis in the tertiary stage the onset of erysipelas may be attended by *immediate* periostitis and necrosis of the facial bones and base of the skull which may produce tremendous deformity and death within a few days. They seem to melt away.

‡ Also called "crab-bite." Three hundred and twenty-nine cases of this type, 323 of which arose from this cause, were reported by Gilchrist in 1903.

The vesicles and bullæ so commonly encountered on the surface are also helpful points in diagnosis.

PYEMIA AND SEPTICEMIA.—These are essentially surgical conditions and need no extended description in this volume.

Diagnosis.—This depends upon a *septic temperature or one which may resemble at first that of typhoid fever* or malaria, but is associated with *marked leucocytosis* in any resistant patient, *lacks the Widal test of typhoid and hemameba of malaria* and is oftentimes associated with scarlatina-like rashes or toxemic jaundice. *Pyemia* must be traced to its *focus* and *is distinctly septic from the start* having a widely *remittent or intermittent* temperature associated with *recurring chills and sweats*. Aside from the surgical conditions, these two diseases are likely to be confused only with typhoid fever in the former case and malaria in the latter, or with both. Ordinarily, care and a study of the symptoms is always sufficient to make a differential diagnosis. It is also necessary, however, to have in mind *malignant endocarditis* as a possibility, and the heart should be examined in every case where either septicemic or pyemic symptoms are present. Advanced tuberculosis is a septicemia in part, but offers few difficulties in its pulmonary form, and as an acute miliary process lacks leucocytosis.

Blood cultures.

The blood cultures are of great service in these cases.

LEPROSY

Prevalence.—*This, the most ancient of known diseases, is caused by the bacillus lepræ* and still prevails throughout the world, though to a very limited and rapidly diminishing degree in Europe and North America, and, indeed, in all countries where modern methods of control through segregation have been attempted.

A vanishing disease.

The Bacillus Lepræ.—(Armauer Hansen, 1871).—The bacillus is sometimes morphologically indistinguishable from the tubercle bacillus and resists decolorization in the same manner. It cannot be readily cultivated, stains more easily, and does not produce tuberculosis in inoculated guinea-pigs.

Duval and Wellman have successfully employed specially treated human placental tissue as the basis of a culture medium. The Bordet and Gengou reaction has so far proved non-specific for the lepra bacillus but specific agglutinins have been obtained. Deycke asserts that in preparations stained by the Ziehl-Neelsen method the following points of differentiation often may be observed: (1) Pointed ends. (2) Great numbers found. (3) Tendency to occur in groups. (4) Frequency of intracellular organism. Much has shown that some lepra bacilli are not acid fast and distinguishes two groups.

Mode of Conveyance.—Much difference of opinion still exists concerning the direct and hereditary conveyance of this disease. As regards the latter it is probable that leprosy, like tuberculosis, may, *but rarely does*, exist in the fetus, and that in nearly all cases it is acquired, the young children of leprous parents being especially exposed to the bacilli which may be found in nearly all of the excretions and secretions of affected cases.* Most

Slightly infectious.

* Statistics show that 93 per cent. of the children of lepers escape infection.

intimate contact seems indispensable to infection, inasmuch as nurses and doctors in leprous settlements are rarely attacked, and it is probable that the nasal passages represent the usual channels of infection though it is said to occur even through the skin and to be conveyed through clothing as in the case of washerwomen especially. Inoculation experiments as applied to man have proved unsuccessful save in one case of Arning's who inoculated an Hawaiian criminal who later developed the disease.

The solitary instance is not sufficient to establish the communicability of the disease by artificial means.

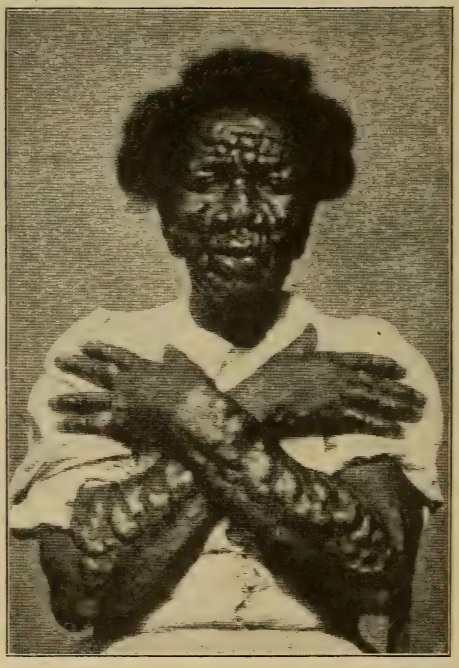


FIG. 539.—Tumor-like leprosy nodules. (*Deycke.*)

The anesthetic form is but slightly communicable, the tubercular type most dangerous, although the necessity for some intermediate change in the virus antecedent to transfer of infection is suggested strongly by its vagaries of transmission. No intermediary host is known.

Incubation Period.—From two to ten or even twenty years may elapse between known exposure and the development of recognizable symptoms.

Sex.—The incidence in the male is double that in the female.

Development of Leprosy.—The close resemblance of leprosy to tuberculosis in many particulars is no less remarkable than the likeness of its stages to the development of syphilis. The initial lesion is believed, by some authorities, to be represented by an ulcer or excoriation on the nasal septum. There is a *prodromal stage* resembling the second stage of syphilis in that it is associated with transient fever, headache, joint pains, and commonly with epistaxis, possibly due to the *septal ulceration*, and after several months or

Primary sore and constitutional symptoms.

even a year or two a febrile paroxysm is accompanied by *macular eruptions*. The circumscribed erythematous patches are generally distributed, chiefly affecting the back of the hand, the forearm, the face, the malar and supra-orbital regions being especially affected, the scalp usually spared.

Macular eruption.

The macules may be *hyperesthetic* early, but later become *anesthetic*, either centrally or wholly. They are at first transient, but tend to become *persistent and pigmented*. Without further advance they may show a loss of pigment and then constitute the *dead white*, *shiny* areas of "white leprosy."

Macular anesthesia.

"The silver men."

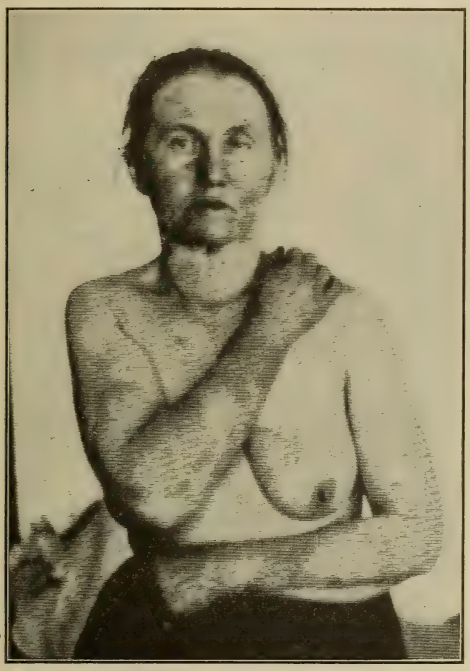


FIG. 540.—Macular anesthetic leprosy. (*Deycke.*)

A definite *third stage* is represented by three forms, the *nodular or tubercular*, the *anesthetic, and the mixed*.

Third stage.

Nodular leprosy may lack the macular stage and the *subcutaneous tubercles are flat, hard and elastic* to the touch, and *most numerous about the ears and face*. Their increase in size and the infiltration of the affected parts produces the "*leontine facies*." With occasional disappearance and reappearance in individual areas, they nevertheless multiply and ultimately involve the greater portion of the body and mucous membranes and break down into ulcers which discharge hosts of bacilli and bacillary emboli and cause horrible deformities from tissue loss and cicatricial contraction.

Leontine facies.

Necrosis and deformity.

Anesthetic Leprosy.—This is characterized by an extensive painful neuritis due to direct invasion of the nerves by the bacilli. *Extensive* anesthesia results, associated with bullæ, spreading ulceration, and subsequent contractures. Muscular atrophy is widespread, and *fingers and toes may be completely destroyed*.

Mixed leprosy represents merely a combination of the two forms.

Three diagnostic criteria.

Differential Diagnosis.—*Any difficulty is limited to the early cases of the macular or anesthetic type.* Three diagnostic criteria are available. (1) *The easy recognition of the bacillus in the blood of a leprous nodule or, in anesthetic cases, in the fragment of a nerve, usually also in the nasal secretion, which may be increased by the administration of an iodide.* (2) *The central anesthesia of early macular areas.* (3) *The administration of pilocarpin to show the absence of perspiration in the involved areas* (Baelz).

FIG. 541.—Characteristic leprous nose. Areas of pigment atrophy on arms. (*Deycke.*)

ANTHRAX

(*Malignant Pustule, Charbon, Wool Sorter's Disease*)

Etiology and Distribution.—This world-wide scourge of cattle and sheep may be conveyed to man through direct contact or by the handling of wool and hides, the channels of entrance being chiefly wounds or abrasions of the skin, the pulmonary tract, the infected stings of insects, and the gastro-intestinal tract. The germ is strikingly resistant, the spores especially so, infection being possible after years through laboratory cultures or in pastures containing the buried bodies of infected animals. It grows readily on ordinary media at temperatures between 12° and 45°C., producing in gelatine stab cultures the inverted pine-tree figure. They produce spores only in culture and stain with all ordinary dyes. The disease is rare in the United States, common in England, France, Germany, Austria, Italy, South Africa,

Sources of infection.

Vitality of spores.

South America, Turkey, and Asia Minor, very frequent in certain parts of Russia, particularly Siberia.

Symptoms.—The disease may be external or internal, in the former assuming the type of "*malignant pustule*" or "*malignant anthrax edema.*" The latter form is usually pulmonary, more rarely intestinal.

External forms.

Malignant Pustule.—From three days to a week after exposure the development of a small itching papule is followed by a rapidly increasing inflammation and infiltration and the formation of a hemorrhagic vesicle which becomes necrotic and bears upon its infiltrated marginal zone similar smaller vesicles. Coincident with the rapid spread of the inflammation adjacent lymphangitis and glandular swelling are evident. The induration and edema are extreme and the necrosis may be extensive, *yet pain is slight or absent.* Marked constitutional symptoms are evident in forty-eight hours when the germ reaches the blood, but the active septic phenomena *including the fever* subside in the later stages, the septic typhoid state preceding death.

Characteristic lesion.

Constitutional symptoms.

FIG. 542.—Anthrax bacillus (*Bacillus anthracis*). A non-motile, non-chromogenic, sporogenous aërobic bacillus, readily cultivated, stained by all methods and highly resistant.

Malignant Anthrax Edema.—Primary constitutional symptoms, excessive gangrenous edema, with a predilection for the eyelids, head, hand, and arm, characterize this variety. The patient's mind may be clear and free from apprehension in either of the preceding forms.

Curious predilection.

Internal Anthrax.—(*a*) *Wool Sorter's Disease.*—A sudden onset with chill, prostration, severe pain in the back and extremities, a temperature (102° to 103°), with chest pain, rapid breathing and in some instances marked bronchial symptoms, characterize this form. The pulse is weak, vomiting and diarrhea may occur, violent cerebral symptoms are noted, and death may result within twenty-four hours. (*b*) *The intestinal form* is due to ingestion of the germs and is characterized by chills, moderate fever, vomiting, diarrhea, marked prostration, dyspnea, phlegmon, or petechiæ and enlarged spleen. Hemorrhage may occur from free mucous surfaces, and, unlike the external form, anxiety is marked and there may be terminal convulsions. It is rare in man, but is the usual form of the disease in cattle, sheep, and goats.

Pulmonary, gastro-intestinal and cerebral symptoms.

Hemorrhage.

Diagnosis.—The occupation of the individual is usually suggestive and the history of an itching papule and its vesication and necrosis should lead to an immediate examination of the contained serum, either microscopically or by inoculation of white mice which are very susceptible. *A progressive course with falling fever* eliminates phlegmonous erysipelas and diffuse cellulitis and malignant edema shows tissue crepitation. The germ can often be recovered from the blood within forty-eight to seventy-two hours after the onset, but may be long delayed.

Occupation.

Falling temperature.

Prognosis.—The mortality is variable but always high in the internal forms and those of the external type in which the face is involved; less so if the extremities are affected.

HYDROPHOBIA

(*Rabies, Lyssa*)

Saliva the medium.

A fuller knowledge and radical precautions have rendered rabies a rare disease in America, England, and Germany. It depends upon a specific as yet unproven virus conveyed to man chiefly by the dog, *and always by direct inoculation.* Though the disease affects cats, skunks, wolves, cows, sheep, and even birds, it is extremely rare in horses and hogs. *The salivary secretion is the chief carrier, the nervous system the main focus* of this hitherto unknown virus.*

Important factors.

The incubation period varies with the age of the victim, is shortest when following wounds of the face and head, but depends also upon the size of the wound, the protection of clothing, and the animal conveying it.

Incubation period.

Ten days and six months represent the extremes of incubation, the average time being forty days. The infected dog stands below the wolf and cat in *virulence*, though far oftener infected.

Incidence and exposure.

It is stated that infection follows in about 15 per cent. of dog bites, as compared with about 40 per cent. for wolves, but it would seem that the severity of the lesion might be chiefly accountable. The mortality varies from 50 to 80 per cent., but has been greatly reduced by the Pasteur preventive treatment.

Symptoms.—*The Premonitory Stage.*—This is characterized by renewed redness of the wound, irritability, insomnia, apprehension, and marked mental depression. Photophobia and hyperacusia may be present. Hoarseness and dysphagia usher in the second stage.

Extreme irritability and spasms.

The patient may be suspicious, solitary, morose, excitable, and loquacious, show delusions or, later, maniacal delirium.

Period of Hyperesthesia and Spasm.—(Duration two or three days.) Excessive reflex irritability appears, the slightest stimulus, physical or mental, excites painful and violent reflex spasms, affecting chiefly the mouth and larynx and accompanied by subjective dyspnea. The mind is clear except during the convulsions and the paroxysms excited by attempts to swallow even water account for the use of the term "*hydrophobia.*" Fever may be absent, but is usually present in moderate degree. Attempts to injure others are rare and confined to the paroxysms.

The Rabies Virus and the Negri Bodies.—Negri's bodies are angular, round or oval, varying from 1 to 20μ in diameter. Negri's original claims have been substantiated by other observers and the bodies seem to be almost or quite constant and found in no other disease. They are found most readily in the *Cornu Ammonis.* By this means a diagnosis can be made within twenty-four hours, and fortunately the bodies are quite resistant to postmortem changes and are present *early* in the disease. That they are the cause of the ailment remains unproven but their presence in a suspected animal is proof of rabies. Pröscher has described extremely small (0.2μ), diplococci as a constant finding, and Noguchi has recovered and successfully

* The richer the nerve supply of the inoculated part, the more rapid and severe the course of the disease.

cultivated, from the brain and spinal cord of infected rabbits killed before the period of exitus is reached, what he believes to be the rabies virus.

He finds granular chromatic corpuscles varying in size from 0.3μ to limit of visibility, together with minute, pleomorphic chromatoid particles 0.2–0.4μ in width and 0.4–0.5μ in length.

These bodies have been carried through several generations and produce typical rabies in susceptible animals. He also encounters occasional larger corpuscles resembling Negri bodies and the sporulation forms originally reported by Negri, but hitherto unconfirmed.

Certain changes in the sensory and sympathetic ganglia have been reported by van Gehucten and Nelis and are believed by these observers to be specific and constant.

Terminal and Paralytic Stage.—Subsidence of the spasms, progressive paralysis, gradually developing coma, and progressive cardiac weakness terminate life.

LYSSOPHOBIA

(*Pseudo-hydrophobia*)

This is merely an hysterical manifestation on the part of one bitten by a suspected animal. Fever is rare and the signs of hysteria usually manifest, though naturally the irritability, apprehension and mental depression may exactly simulate the premonitory stage of the true disease. The excessive hyperesthesia, true spasm, and other indications of actual infection can hardly be exactly simulated.

Imaginary hydrophobia.

Diagnosis.—*The greatest enemy of the patient and diagnostician is the officious public servant or citizen who promptly destroys and* **disposes of** *a suspected animal, for the first step should be the transfer of the living animal or of the fresh brain and medulla to the hands of the bacteriologist for the purpose of the inoculation of animals and the infinitely more important detection and identification of the Negri bodies.*

Hydrophobia scares.

If the dog is apparently well, its isolation may be undertaken, but if the slightest evidence of rabies exists he should be scientifically dispatched in such a manner as will leave the brain uninjured.

Such action not only promotes prompt diagnosis and efficient treatment, but often prevents an unfounded hydrophobia scare and the persecution and slaughter of innocent animals.

Persons bitten in the face should be placed under Pasteur preventive treatment at once, without waiting even for the results of the laboratory tests.

TETANUS

(*Lockjaw*)

Extraordinary Virulence.—The bacillus is inoculable in animals and its peculiar toxin is fatal in the extraordinary dose of 0.23 mg.

Marvelous toxicity.

As tetanus neonatorum, the disease especially affects new-born children in countries or districts where uncleanly methods are followed in the care of the navel. The soil of certain regions is peculiarly infectious, and the germ

A soil dweller.

usually enters the body through a *punctured* or *contused* wound. The fatalities attending the use of toy pistols are well known.

Under the new National law all serums and vaccines must now be tested fully for freedom from tetanus bacilli or spores by animal inoculation before being placed on the market.

The Great War, of necessity brought about the systematic wholesale use of antitetanic sera, and the results tended to establish its great value with respect both to lessened incidence and reduced mortality.

Incubation.

Symptoms.—After a period of incubation of about a week or ten days, or more rarely two weeks, a subjective sensation of constriction of the neck and jaws with difficult mastication is followed by a tonic spasm of the muscles which lock the jaw, and an outdrawing of the mouth and raising of the eyebrows causing the *risus sardonicus*. By gradual involvement the muscles of the trunk and extremities are affected, producing rigid extension (*orthotonos*), or violent posterior flexion of the spine (*opisthotonos*), and more rarely lateral (*pleurosthotonos*) or anterior flexion (*emprosthotonos*). The duration of the spasms is variable, *relaxation usually incomplete;* they are initiated by the slightest irritation and are extremely painful. The fever is usually moderate or perhaps absent, more rarely there is hyperpyrexia. Spasm of the glottis may cause dysphagia and paralysis of the facial muscles may occur, but in children spasm may be limited to the production of the risus sardonicus.

Locked jaw.

Risus, sardonicus.

Spinal flexion.

Fever.

FIG. 543.—Tetanus bacillus (*Bacillus tetani*). A long slender bacillus with single polar spore, flagellate, motile, anaërobic, non-chromogenic, sporogenous, liquefying, readily stained by all methods but cultivated with difficulty.

Diagnosis.—*Strychnin poisoning lacks the early involvement of the muscles of mastication and the rigidity between spasms.*

Prognosis.—Of idiopathic cases about 50 per cent. die, traumatic cases 80 per cent. The head tetanus, due to wounds of that part, in its acute form, is almost invariably fatal; if chronic, the mortality may be only 25 per cent.

Head tetanus.

GLANDERS

(*Farcy*)

Primarily a disease of the horse, it is communicable to man through the agency of the *bacillus mallei*. It is essentially an infective granuloma, actual contact is the usual method of conveyance, and it is encountered in both acute and chronic forms.

Acute Glanders.—(*Infection through the nostrils.*) Three or four days after infection there is *fever and evidence of local sepsis*. The mucous membrane of the nose becomes involved within forty-eight or seventy-two hours, nodules forming which undergo necrosis, the ulcers yielding a mucopurulent discharge. All erysipelatous-like facial swelling is followed by a pustular eruption, swelling of the hands, arms, legs, and feet may occur and there is marked lymphangitis and glandular involvement in the drainage areas

Primary intranasal lesion.

affected. Pneumonia is a frequent complication, and the disease is fatal in from eight to ten days.

Acute Farcy.—(*Infection by the skin.*) The symptoms are those of an acute septicemia following local phlegmon representing the site of inoculation, together with an acute lymphangitis characterized by subcutaneous nodules in the course of the lymphatics, which may suppurate and are called "farcy buds." The urine may show the germs. Death usually ensues in from ten to fifteen days. *Both varieties of the disease show a chronic form lasting for months or even years and occasionally terminating in recovery.*

Dermal lesion.

"Farcy buds."

Chronic glanders.

The inoculation of animals and cultural methods are often necessary for diagnosis. In all forms the occupation serves as a valuable suggestion and it is usually possible to find the source of infection in a diseased horse.

THE MYCOSES.—Modern research has developed the fact that a great number of the coarse fungi are pathogenic for man. Most of these produce exclusively or predominantly lesions of the skin and find no place in this volume.

Some of the more important of those producing internal symptoms will receive brief consideration.

Aspergilloses.—*Aspergillus fumigatus* is the form usually found. The pulmonary form of aspergillomycosis has been discussed already in another section. While unusual this is of great interest to the diagnostician.

Mucormycosis.—In rare cases of enteritis the chief part may be played by *Mucor corymbifer.*

Trichophytia profunda, Trichophytia unguium, Trichophytia superficialis, Epidermophytia cruris and Trichophytia tonsurans capillatii, represent the activity of *Trichophyton tonsurans* in producing respectively, parasitic sycosis, ringworm of the nail, ordinary ringworm, eczematous ringworm and "barber's itch."

Pityriasis versicolor is caused by the *Microsporon furfur,* and "erythrasma" by *microsporon minutissimum.*

Blastomycosis.—The blastomycetes are yeasts (budding fungi) and may or may not produce mycelium.

The oidium albicans or "thrush" stomatitis is the best known and oftenest seen, but the same ailment may be caused by several other forms.

Of these, *Endomyces albicans* is only less common than *O. albicans.*

SYSTEMIC BLASTOMYCOSIS.—This is a very important ailment, despite its rarity, by reason of the severity of its symptoms and its tendency to deceptive localization.

Cause.—*The Cryptococcus gilchristi* is a budding fungus, which in pus or tissues appears as a single paired or budding cell varying in diameter between 10 and 16μ and enclosed in a highly refractile membrane.

Mode of Entrance.—It seems probable that inhalation implants the pathogenic organism.

Symptoms.—The first effect is pulmonary, a bronchopneumonia developing with a history of pain in the chest, perhaps chill, and evidences of severe or primary bronchitis with blood-stained sputa.

Misleading symptoms.

Fever is present and with the coming of subcutaneous nodules, abscesses, single and successive or in crops, chiefly on the face and neck, together, usually, with ulceration, secondary, or even primary and independent of abscess sites, a chronic infection becomes established, which may assume the clinical form of an excessively chronic septicemia or pyemia. Ulcerating areas by fusion may cause large sluggish fungating areas. The joints may become very much enlarged in certain cases. The blood shows a moderate or high leucocytosis, but is otherwise not greatly affected unless the invasion of the medullary substance of the bones produces myelocytic forms.

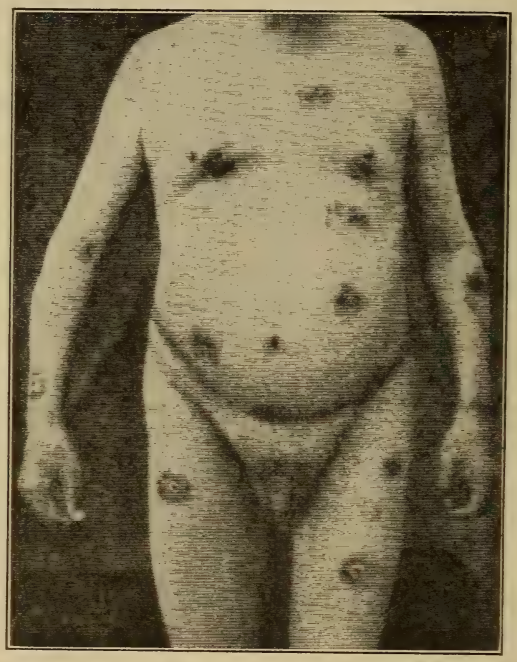


FIG. 544.—Disseminated, polymorphous, ulcerative sporotrichosis, simulating syphilis, tuberculosis, ecthyma, and furunculosis. (*After C. H. Plaut.*)

Diagnosis.—This depends primarily upon the physician's recollection that such a disease as systemic and dermal blastomycosis ("oïdiomycosis") exists; secondly, upon the recovery of the fungus from the pus or, in some instances, from the sputum or urine, or even the blood.

The spores are easily recognized if the pus is heated with 20 per cent. sodium hydrate solution or even with no preliminary mixing. A stain is neither necessary nor desirable.

These cases are mistaken for other ailments and especially for tuberculosis of the skin, lung and bones unless the disease is held in mind as a possibility.

The direct diagnosis is then so easy as to make unnecessary the specific tests for syphilis and tuberculosis.

Coccidioidal granuloma must be excluded by examination of the fungus present in the discharges or tissues.

Prognosis.

This type of disease is quite different from blastomycosis, the course almost invariably being progressively and rapidly fatal.

Both diseases may involve not only the skin, lungs, bones and joints, but also the kidneys, liver and spleen.

COCCIDIOIDAL GRANULOMA.—As in blastomycosis this, an almost invariably rapidly fatal disease, presents dermal symptoms with evidences of overwhelming systemic infection.

The disease is more rapid in progress, resists the iodides, in decided contrast to its simulator, affects the lymph glands more decidedly and shows a very different form of fungus in the discharges or tissues.

This is the *Oïdium coccidioide*, which in simple preparations appears as spherical cells within a highly refractile membrane, but in a 48- or even 24-hour culture shows mycelial growth. It reproduces itself by endosporulation.

SPOROTRICHOSIS.—This ailment was first reported by E. F. Schenck in 1896 who isolated its cause *Sporotrichum schenckii.*

Symptoms.—The chief interest lies in the strong resemblance of the sporotrichosis nodules to syphilitic gummata. These soften and break down into most obdurate and resistant ulcers resembling tuberculous lesions or ecthyma.

Sore throat, eye involvement and sporotrichosis of bones and joints have been noted.

Systemic symptoms are lacking, superficial closure of a discharging opening may take place before the abscess is healed within.

Gougerot distinguishes three forms: 1st. That of disseminated gumma-like nodules which soften, break down and ulcerate.

2d. A dominantly ulcerative type, the lesions resembling those of tuberculosis.

3d. A hard, chancroid, localized, eroded lesion.

Diagnosis.—This depends upon the recognition of the fungus.

This is a short rod, large (3-5μ by 2-3μ), oblong or ovoid, somewhat granular, enclosed by an unrefractile membrane which refuses the ordinary stains.

In cultures it shows mycelial growth and oval or round ectospores.

THE NOCARDOSES.—The nocardoses (streptotrichoses) include true actinomycosis, elsewhere fully described.

Practically the same processes may result from infection by the *atypical* actinomycoses, identical with the true form save that they lack the club-like swellings.

Other nocardoses of importance are the members of the mycetoma group which are associated clinically and genetically with Madura foot, and the pseudo-actinomycosis group comprising several of the nocardia.

Madura Foot (*Mycetoma*).—This curious ailment is most frequent in and near Madura, India, and is much less common in other portions of the Empire.

It occurs as a widespread disease in northern Africa, and is occasionally reported from Greece, Italy, West Indies and the United States.

Symptoms.—The foot alone (75 per cent.) or sometimes other parts of the body, become greatly swollen and the site of multiple discharging fistulæ

leading from the surface to deep-seated granulomatous areas, which prove to be casting off small granules similar to those present in true actinomycosis.

As a rule but one form is present and, according to Stitt, this usually is the *Nocardia maduræ*, showing pallid fish-roe like granules. *Nocardia asteroides* and others may be causative, and a discharge of black granules like gunpowder grains indicates the presence of *Madurella mycetomi*, *Aspergillus bouffardi*, or *Sporotrichum beurmanni*.

The disintegration of the tissues may be so great as to convert the foot into a mere "cheesy" mass, and an inflammatory edema is constant.

Atrophy of the leg on the side affected serves to emphasize the deformity. Pain is slight or absent and relief is sought by the natives because of the burden imposed by the enormously enlarged useless member.

Amputation is the only procedure of value, and cases are seldom seen in the earliest stage when there is a mere swelling of the sole.

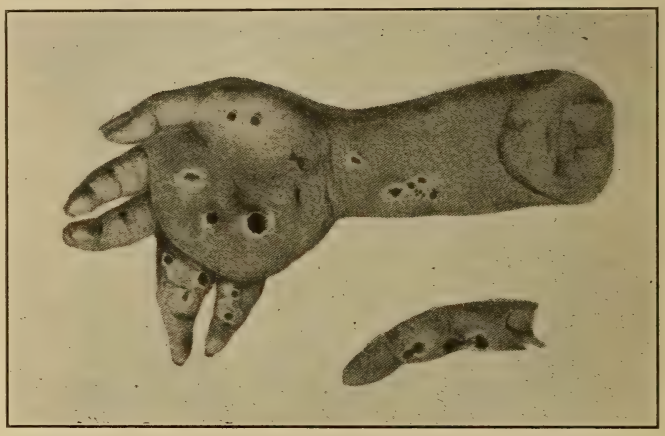


FIG. 545.—Madura hand. (*After Carter.*)

Pseudotuberculosis Hominis Streptotricha.—As stated elsewhere the nocardoses may present a clinical picture almost indistinguishable from that of tuberculosis of the lungs, and cause pyorrhea, stomatitis, quinsy, enteritis, and even appendicitis in rare instances.

The streptothrix is acid fast but less obdurate than the tubercle bacillus. It tends to branching forms and its units are longer and of equal length.

In the stomach it may be taken for the Boas-Oppler bacillus.

Another unpleasant quality is that of forming metastases in distant organs.

In relation to this as to so many other of these rare conditions the diagnosis depends chiefly upon remembrance of the fact that such causes of disease exist and that the very terms "syphilis" and "tuberculosis," especially, must carry with them the suggestion of the nocardoses and mycoses as rare possibilities.

ACTINOMYCOSIS.—This disease which also belongs to the infective granulomata is caused by the *Streptothrix actinomyces* or "ray-fungus," and

constitutes the "lumpy jaw" of cattle. Successful cultivation and animal inoculation have proven the fungus, and the pus from the lesion shows characteristic opaque yellow granules from ½ to 2 mm. in diameter. Infection occurs, doubtless, through the entrance of the fungus into the mouth or air passages of man, and the disease is divisible into four forms according to the seat of the primary process.

"Lumpy jaw."

Characteristic yellow granules.

If the upper portion of digestive tract be involved, there may be swelling of the face, usually unilateral, but sometimes bilateral, closely simulating sarcoma or tertiary syphilis. The tongue may be affected or the disease

FIG. 546.—Madura foot. (*After Carter.*)

may be found along the lower digestive tract where it may produce appendicitis, colitis, and even peritonitis, but the liver is rarely affected.

Pulmonary actinomycosis is a chronic, wasting febrile disease with marked pulmonary symptoms simulating chronic bronchitis, the usual pulmonary abscess, disseminated tuberculosis, broncho-pneumonia, or tuberculosis associated with interstitial changes and cavities.

Simulates pulmonary tuberculosis.

*In the digestive form the organism may be recovered from the stools, in the pulmonary form the presence of actinomycoses in the sputa and the absence of tubercle bacilli may sometimes make the diagnosis clear.**

Cutaneous actinomycosis closely simulates tuberculosis of the skin. *Cerebral actinomycosis* yields symptoms of brain tumor or abscess.

Diagnosis.—The disease so closely simulates a multitude of tumor

* The author has encountered more closed than open cases and knows of no more obscure condition.

formations and inflammatory lesions that usually nothing short of the demonstration of the specific organism suffices for diagnosis.

LUPINOSIS (*"Lathyrism," "Chick-pea Disease"*).—This ancient disease is due to the prolonged use of the common vetch or chick-pea as food and is common during times of famine in British India, France, Italy, and Algiers.

Symptoms.—With gastrointestinal disturbance as a prodrome, the onset of pain with weakness and tremor is followed by spastic paralysis of the legs with exaggerated reflexes and normal sensation. Death occurs from intercurrent disease.

MEAT POISONING.—Whether due to the ingestion of poisonous sausage, pork, beef, veal, milk, or cheese, the symptoms are malaise, anorexia, and nausea or certain vague pains, abdominal, cervical, or general. In some instances chilliness or rigor, vertigo or tremor precede the attack. The actual onset is characterized by acute gastrointestinal disturbance resembling cholera morbus, violent abdominal pain, marked prostration, sweating, severe headache and thirst. Fever varies from 100° to 105°F., and the pulse is usually rapid.

LACQUER POISONING.—This disease of lacquer workers strongly resembles the ivy (Rhus toxicodendron) poisoning of this country, differing only in the greater severity of its symptoms. The base of lacquer is the balsam of the Rhus vernicifera.

THE CHIEF ANIMAL PARASITES

RHIZOPODA.—*Entameba histolytica* is the chief member of this group and has already been discussed under "Amebic Dysentery." Other forms of ameba apparently non-pathogenic are occasionally encountered in the stools, urine, mouth (pyorrhea alveolaris) and lung cavities, the mode of introduction being little understood.

FIG. 547.—*Trichomonas vaginalis.* (*After Tyson.*)

FIG. 548.—*Megastoma entericum.* (*Grassi and Schewiakoff.*)

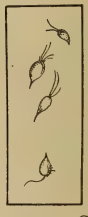


FIG. 549.—*Cercomonas hominis* (*Trichomonas intestinalis*). (*Pribram and Davaine.*)

FLAGELLATA.—The three varieties ordinarily encountered are probably non-pathogenic and merit no extended description. The pear-shaped *Trichomonas vaginalis* is from 15 to 20 micra long, and 7 to 12 micra wide. Its blunt end carries a nucleus and three or four flagellæ. It is a common constituent of acid vaginal secretion. Another pear-shaped organism, *Megastoma entericum*, is 10 to 21 micra long and 5 to 12 micra wide. From the edges of a marked depression upon its anterior surface project three pairs of cilia, another pair springing from its pointed end.

The Cercomonas hominis or Trichomonas intestinalis, also pear-shaped. measures from 10 to 16 micra, carries two or three flagellæ, and sometimes a visible nucleus.

The Balantidium coli may or may not be pathogenic. It is a small oval ciliated organism and carries two nuclei, one spherical, the other reniform. Its blunt end shows a funnel-shaped opening. It has been found on and within the intestinal mucosa and submucosa, as well as free in the stools, and can cause an ulcerative dysenteric colitis.

FIG. 550.—*Balantidium coli. (After Malmsten and Leuckhardt.)*

TREMATODA (*Flukes*).—*The most important of these organisms is the Bilharzia hematobia or Schistosomum hematobium* which causes endemic hematuria prevalent in Egypt, North and South Africa, Arabia, Persia, and the West Coast of India.

Only isolated imported cases are encountered in Europe and America. The accompanying illustration shows the peculiar relation of the sexes. The embryos are free swimming and the organism reaches the intestinal tract directly or indirectly through contaminated water. Once ingested, the organism is found chiefly in the portal veins, but ultimately in other portions of the body, especially the bladder and rectum. The eggs laid in tissues may also make their way to the bladder and intestines and escape with the urine and feces. In the tissues they may cause papillomata and fibroid processes or even form the nucleus of a vesical calculus.

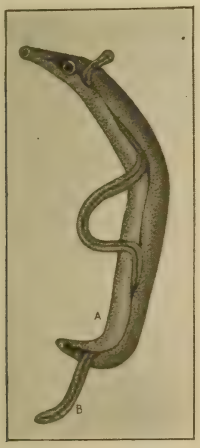


FIG. 551.—Male (*a*) and female (*b*) of *Bilharzia hematobia*. (*After Looss.*)

FIG. 552.—*Fasciola hepatica.*

Symptoms.—The foregoing description indicates the seat, diverse nature, and variety of symptoms, which are of course chiefly those of hematuria, abscesses, and irritation of the urinary tract, dysenteric stools, rarely Glissonian cirrhosis and a varying degree of anemia. The diagnosis depends upon the finding of the characteristic eggs.

The Fasciola hepatica or *Distomum hepaticum* of sheep, a small fluke,

measuring 20 to 30 mm. in length, carries fine spines, and is a rare source of infection in man. Its eggs measure 130 to 150 micra.

The Distomum lanceatum (*Dicrocoelium lanceatum*) or "lancet fluke" is 8 to 10 mm. in length and 1.5 to 2.5 mm. in width, and carries no spines. Infection in man is excessively rare, the intermediate host being the snail.

The Opisthorchis felineus measures 8 to 10 mm. in length, and 1.5 to 2 in breadth. It is a translucent yellowish-red parasite not uncommon in Siberia and occasionally found in Germany.

The Opisthorchis sinensis (*Clonorchis endemicus*) is like the preceding variety in size and color, its egg measuring, however, 20 to 35 by 15 to 21 micra. It is abundant in India, China and Japan, and is occasionally seen in America.

In all probability the pathogenic effects ascribed to it formerly must be charged against *Fasciolopsis buskii*, a very large and common fluke, whose habitat, probably, is the upper intestine.

The foregoing liver flukes may produce a chronic disease chiefly affecting children, often members of one family, and characterized by marked hepatic and gastrointestinal symptoms, chief among which are irregular diarrhea, becoming bloody, hepatic enlargement, slight fever, pain, intermittent jaundice, anemia, emaciation, and finally, general anasarca.

The Paragonimus westermanni is a pulmonary fluke of a reddish-brown color and oval form, measuring 8 to 10 by 4 to 6 mm., its eggs measuring 56 by 90 micra. It prevails widely in China, Formosa, Korea, and Japan and is occasionally found in the United States. The symptoms produced are repeated attacks of hemoptysis, trivial or severe, and chronic cough with rusty sputum. It should be remembered that all of the flukes found in man are flat, leaf-shaped bodies with ventrally placed suckers, and that all save the Bilharzia are hermaphrodite.*

CESTODES (*Tapeworms*).—These are long, flattened, and segmented worms, each carrying both male and female sexual organs and lacking a digestive tract. The head or scolex carries suckers and in some varieties encircling hooks. Unless the head be removed, the worm may continue its life and growth by budding. Each and every segment carries sexual organs placed along the border in the teniæ and in the median line in the bothriocephalus. The number of eggs is enormous and they may show a developed embryo.

FIG. 553.—*Tenia saginata.* (Mediocanellata.) (*After Pribram and Wood.*)

Tenia Saginata or Mediocanellata.—This is the common tapeworm of this country and Europe, sometimes called the "unarmed" or "beef-tapeworm" because lacking the hooklets and often conveyed by beef. Its head is larger than that of the tenia solium, measuring 2 mm. or more in breadth. Its form is shown by the accompanying illustrations. Its segments measure from 17 to 18 mm. by 8 to 10. The ova are not distinguishable from those of tenia solium. A length of 15 to 20 or even 30 ft. may be attained.

* The measurements throughout this section for the most part follow the descriptions given by F. C. Wood, James Tyson and Pribram.

Tenia Solium.—The tenia solium or pork tapeworm, is common in Europe or Asia, but infrequent in this country. It is shorter than the tenia saginata, seldom exceeding 12 feet in length. The head is smaller, rounder, and provided with double circlet or hooklets in addition to its suction discs, the neck is narrow and threadlike. The segments measure 1 cm. by 7 to 8 mm. The structure of the uterus and general form of the worm is shown in the accompanying illustration. In man and pigs the ova develop and the free armed embryos pass to different parts of the body and develop as cysticerci.

FIG. 554.—(A) Scolex, (B) egg and (C) of *Tenia solium*. (*Pribram and Wood, modified segments.*)

Dibothriocephalus latus.—This cestode, though largely confined to Switzerland and Japan and the coast of the Baltic, is occasionally encountered in immigrants and has recently been found in Minnesota in a number of cases reported by the late Frank F. Wesbrook. It is conveyed by the pike or other fish; the adult worm measures 25 or more feet in length, is unarmed and carries two lateral grooves upon its head. Among the other forms of cestodes may be mentioned the *Diplogonoporus grandis* or *Krabbea grandis* found in man only in Japan, the *Dipylidium caninum* common in the dog, very rare in man and also occurring in the canine and human flea and dog lice. The *Hymenolepis nana* or *Tenia nana*, a small worm but 5 to 45 mm. in length and 0.7 in breadth. This worm rarely found in man finds its host in the rat, its habitat chiefly in Italy. Its head carries four suckers and a row of from 24 to 28 hooklets. The egg is oval or round and measures from 30 to 37 micra, the embryo carries six hooks.

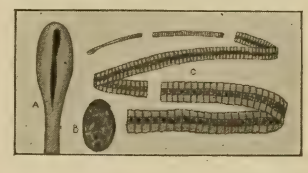


FIG. 555.—*Bothriocephalus latus*. (A) head (magnified), (B) egg, (C) head, neck and proglottides.

Hymenolepis diminuta or *Tenia flavopuncta*. This small worm measures 30 to 60 cm. by 3.5 mm. The head (0.2 by 0.5 mm.) is armed and oval. The eggs, 60 to 70 by 70 to 80 micra, are yellowish and show faint radial striation. Butterflies, beetles, mice, and rats are its commoner hosts.

FIG. 556.—*Hymenolepis nana*. (A) head (magnified), (B) egg, (C) hooklet, (D) head and proglottides. (*After Braun, modified.*)

Symptoms of Tapeworm Infection.—These may be those of anemia and slight gastrointestinal and nervous disturbances or an anemia undistinguishable from pernicious anemia save by finding the parasite. The commonest and most significant grouping embraces the following symptoms: (*a*) Excessive or ravenous appetite. (*b*) Indigestion. (*c*) Oppression, fulness or actual pain over the abdomen. The symptoms may be present only in part, however, or almost *wholly* absent.

Davainea Madagascariensis is a rare parasite found in British Guiana, India, and Africa. It is but 30 cm. in length and has an armed head with 90 hooks and four suckers. The ova measures from 6 to 8 micra, has two spikes and a double shell.

TENIA ECHINOCOCCUS.—This interesting tapeworm inhabits the dog which becomes its conveyer to man. Australia and Iceland are the chief sources of infection, nearly half the Australian dogs being affected and about one-third of those in Iceland. In other countries it is rare. The adult worm consists of about three or four members and measures about 2.5 to 6 mm. in length. The breadth of the head is 0.3 mm. It carries four suckers and is supported by a short neck. The head is armed with a double circle of from 28 to 50 hooks.

Mode of Infection.—Direct transfer from dog to man may result from close contact, especially in such countries as Iceland and Australia, where dogs are numerous and in close relation to their master and the family. Contamination of drinking water is also easily understood. If the small six hooked embryo is released by digestion of its shell, it passes through the intestinal mucosa to various portions of the body, chiefly, of course, the liver.

Hydatid cysts.

Wherever it lodges small double-walled cysts about 1 mm. develop, the hooklets disappear, and a fibrous envelope results from the slight inflammatory reaction. These primary cysts later develop secondary cysts through budding from the inner layer and these daughter cysts are structurally identical with the primary ones. Later they are released and a dozen or more may be found within the parent cyst. Each daughter cyst may itself reproduce by budding until the original cavity represents a long family line. Scolices develop carrying hooklets and sucking discs and representing the young worms. Thus a multitude may arise from a single cyst. The fluid is primarily non-albuminous, limpid, and of a specific gravity from 1005 to 1010. The cysts may endure for many years, ordinarily undergoing a resorption and ultimate calcification, the hooks persisting even in obsolete cysts. Rupture is always possible, symptoms and danger depending upon the location. Suppuration is a serious but unusual event, most common in the liver.

"Daughter cysts."

Distribution.—About 50 per cent. of echinococcus cysts occur in the liver, 20 per cent. in other abdominal organs, some, less than 10 per cent. each, in the pulmonary tract and nervous system.

Symptoms.—The size and location of the cysts determine the physical signs and subjective symptoms and the discovery of the hooklets in the feces, urine, sputum, etc., makes the diagnosis positive. Obscure symptoms referable to the organs most often affected are especially suggestive in Icelanders and Australians. The hydatid thrill is almost pathognomonic, but often absent even in relatively superficial cysts. It may be felt as a prolonged exquisitely fine vibration echoing the stroke of ordinary finger plexímeter percussion, but more clearly when the middle one of these plexímeter fingers is firmly percussed without the usual recoil of the percussion digit. In one case observed by the author the case was almost symptomless though evidently one of hepatic involvement, but the thrill at once solved the problem.

Misleading symptoms.

Hydatid thrill.

Aside from the foregoing points, a minute discussion of the host of symptoms is futile, as there are none other than those of similar growths in different anatomic areas. Suppuration produces the picture of abscess, septicemia, or pyemia. A specific complement-fixation test is now established.

Perforation.

Perforation may involve any adjacent structure and produce the symptoms peculiar to lesions of that organ. Urticaria is said to be a peculiar and frequent accompaniment of perforation. Multilocular elastic or fluctuating tumors always *suggest* hydatids, especially when in the hepatic and renal regions, but as pulmonary phthisis and gangrene, all forms of abscess, pericardial effusion, cardiac dilatation, pleuritic effusion, hydronephrosis, renal calculus, pyelitis, a distended gallbladder, and even hepatic syphilis and carcinoma have been confounded with it, it is evident that exploratory puncture is often required. Hooklets are seldom absent in the fluid withdrawn. In the central nervous system the symptoms are those of tumor. Finally it should be emphatically stated that *in many cases no symptoms of importance appear*, that in general the impairment of health and nutrition is relatively slight in the absence of complications and that recovery under operation is the rule.

Ascaris lumbricoides.—This, the most common intestinal parasite requires no intermediate host and infests the upper portion of the small intestine. The male measures 15 to 17 cm., the female 20 to 25 cm.; it is earthworm-like in form. Being migratory, they are most frequently recovered from the stools, but may be found in the appendix, the bile ducts, in vomitus, or may even enter the pharynx, larynx, trachea, Eustachian tube, nasal passages, or lachrymal duct. Usually but from two to ten are present, but they may exist in quantities sufficient to cause intestinal obstruction. Rare cases of intestinal perforation are reported, but are probably instances in which the presence of the worm is accidental rather than causative.

FIG. 557.—*Ascaris lumbricoides.* (A) female, (B) male, (C) egg, (D) head. (*Pribram.*)

Largely gastro-intestinal or nervous.

Symptoms.—A few worms may be present without symptoms. Pallor, restlessness, irritability, nose rubbing and picking, bad breath, anorexia, colicky pains, tympanites, malaise, impaired nutrition, disturbed sleep with teeth grinding, complex and misleading nervous symptoms, such as epileptiform seizures, general convulsions, tetany, vertigo, aphasia, and even actual paralysis, localized or hemiplegic, as well as recurrent febrile attacks, are among the symptoms *attributed* to this parasite. As a matter of fact, each and all are merely suggestive and the diagnosis can never be made unless the worms or their eggs are found. They are excessively rare in infancy and most common from the third to the tenth year.

Misleading symptoms.

Not distinctive.

Oxyuris Vermicularis.—The "pin" or "thread" worm measures 4 mm. for the male, 10 mm. for the female. These migratory parasites infest the

folds of the lowest bowel, seldom being found above the cecum, though rarely they migrate as far as the pharynx or perforate the intestine and form "verminous tubercles." In the stools they resemble bits of white thread and are readily dislodged by appropriate medication. The prominent symptoms are genital and anal itching with nocturnal exacerbations and such other symptoms as have been described as associated with the round worm, save the nervous symptoms are less prominent and mucous colitis and such reflex irritation symptoms, frequent urination or incontinence, and balanitis, vaginitis, and perhaps masturbation are common. Here also the worm or its ova are essential to diagnosis, and are easily found, often indeed about the *external* genitals or anus. The worm is conveyed from person to person or by infected water or food.

Itching.

Nervous symptoms.

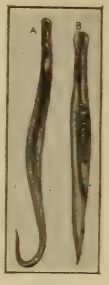


FIG. 558.—*Oxyuris vermicularis*. (A) female, (B) male.

TRICHINIASIS.—In man trichiniasis depends upon the ingestion of infected raw or imperfectly cooked pork. Those suffer most who eat raw and underdone meat, and most of the cases encountered in this country have been Germans. It cannot be a rare condition as Williams reports 5 per cent. as showing infection in a series of 500 autopsies, but it is often unrecognized. The larvæ require about seventy-two hours of residence in the intestines to become sexually mature and at the end of a week each female may have discharged into the lymph spaces or blood stream several hundred embryos. In due course these pass to the muscle fibers in which they create a reactive inflammation and become encapsulated. They may or may not so live for years. In man the appearance of an old cyst is that of "an opaque oat-shaped body" (Osler) owing to late calcification.

Underdone or raw pork.

Prolific females.

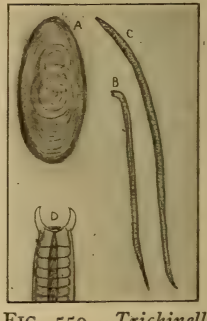


FIG. 559.—*Trichinella spiralis*. (A) encysted in muscle, (B) male adult, (C) female adult, (personal observation) (D) male genital apparatus.

Symptoms.—Assuming that a sufficient number of trichinæ have been ingested, symptoms appear in about one week and are usually characterized by the conjunction of acute gastrointestinal disturbance, and fever, usually remittent or intermittent, followed by *muscular pain and tenderness, limitation of movement, edema of the face*, extremities and surface over affected areas, and a marked leucocytosis in which the eosinophiles often reach 50 to 70 per cent. of the total leucocytes. The attention should be fixed upon *the muscle symptoms, swelling of the eyelids and eosinophilia*, as the case may assume the typhoidal form, be complicated by albuminuria, pleurisy, pneumonia and dysphagia, aphonia, etc., etc. In other instances symptoms are of the scantest. The mortality is extremely variable (2 to 30 per cent.). The diet and the grouping of cases may also be suggestive, but oftentimes the muscles must be directly examined if the stools, ingested food, and other more available material fail to show the organism. Harpooning the muscle is neither necessary nor proper if local anesthesia and a clean incision can be employed.

Muscle pain and tenderness.

Eosinophilia.

Puffy eyes.

Mortality.

Direct diagnosis.

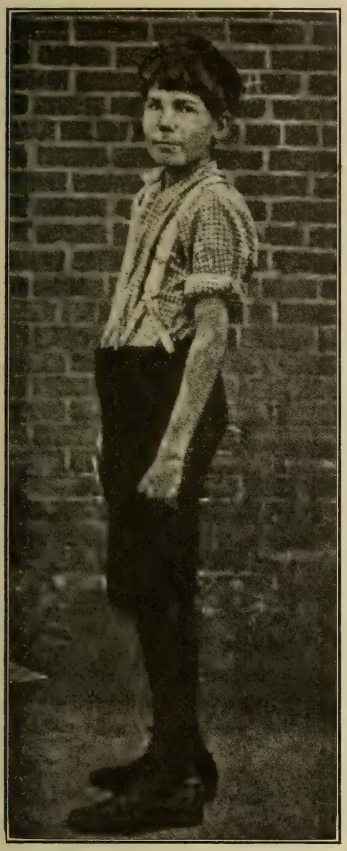


FIG. 560.—Ankylostomiasis. Retarded development and typical *facies* in a young man.* (*After Carter.*)

Many cases are mistaken for rheumatism in spite of the lack of joint involvement and the peculiar muscle symptoms.

UNCINARIASIS (*Ankylostomiasis, Egyptian Chlorosis, Hook-worm Disease*).—Once regarded almost exclusively as a tropical disease, uncinariasis has been proven wide-spread and by no means limited to the tropics, though more widely prevalent in southern latitudes. It may be found in Egypt, England, Europe, Switzerland, India, the Malayan Archipelago, the West Indies, and in our Southern States it is extensively distributed. Stiles has described an *Uncinaria americana* (*Necator Americanus*) differing slightly from the older form (*Ankylostoma duodenale*). The dimensions of both are for the male 7 to 11 mm., for the female 10 to 18 mm. (see plates). No intermediate host is necessary, and the organism thrives in dirt and puddles. There are many reasons for believing that the skin may be a channel of infection. Adult worms may be found in any portion of the gastrointestinal tract, but chiefly in the duodenum and rarely in the stomach or colon.

Symptoms.—There may be none in cases showing the worm in the feces, but ordinarily gastrointestinal symptoms co-exist with anemia, simple and mild, or so profound as to imperfectly simulate pernicious anemia from which it is distinguished by normoblast predominance and a low color index. Stiles emphasizes the peculiar lack-luster stare and the muddy or waxy white skin. The remaining symptoms are those of profound anemia with anemic bruits, breathlessness, late edema, and a peculiar ascites. The characteristic feature of the anemia is the excess of eosinophile cells, both relative and absolute, most marked in early cases. The proportion of these cells to the whole leucocyte count varies from 4 to 50 per cent., the average being between 20 and 25 per cent. A rise in the count under treatment is a good omen, a low original count or a falling one is a bad one. Leucocytosis is present in a majority of the cases, but

FIG. 561.—*Uncinaria* (*ankylostoma*) *duodenale.* (A) female, (B) male, (C) eggs, (D) male and female showing actual length but about one-third greater width than the actual specimens. (*Pribram, slightly modified.*)

Anemia.

Eosinophilia.

Color index low.

*Mental and physical retardation is commonly noted.

is seldom high, a count above 12,000 or 13,000 being exceptional. The more advanced the disease, the lower is the leucocyte count. The color

FIG. 562.—Fatal case of ankylostomiasis. Erythrocytes 810,000, hemoglobin 15 per cent. Note marked edema (*United States Naval Bulletin*). (*Stitt.*)

index is, as a rule, low, and in the tropical cases especially so, the same being true of the red cell count. Cell deformity is marked in the severer cases, but normoblasts predominate.

Three chief forms.

FILARIASIS.—This extraordinary parasite includes three chief species. *The Filaria bancrofti*, also called *nocturna*, is found in the peripheral blood only at night or during sleep, whereas the *Filaria loa*, or *diurna*, is found only during the waking hours or in daylight. *The Filaria perstans* is present day and night and is said to be present in from 50 to 90 per cent. of West African negroes, being apparently non-pathogenic. *The Filaria demarquayi* is found in the West Indies, persists day and night, and is actively motile. The *Filaria ozzardi* found in British Guiana, and a large species, the *Filaria gigas*, has been found at Sierra Leone. The first two forms, viz., *nocturna* and *diurna*, are the only ones of clinical importance. *Filaria nocturna* is found in all tropical and many subtropical countries. Most of those observed in the northern part of this country are

Peculiar habits of parasite.

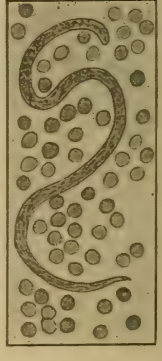


FIG. 563.—*Filaria nocturna* in blood.

imported cases. In certain tropical regions 70 per cent. of the population of certain villages may be affected.

Morphology (*Filaria nocturna*).—The parent worm inhabits the lymph channels, the embryo the circulating blood, the body of the mosquito serving as intermediate host. The adult form is from 3 to 4 inches in length and of the thickness of a fine thread. The embryo is $\frac{1}{80}$ inch in length and about

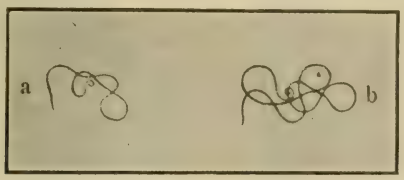


FIG. 564.—Male (a) and female (b) of *Filaria bancrofti*. Natural size. (*After Manson.*)

$\frac{1}{3000}$ inch broad, delicate and transparent, and enclosed in a transparent sheath. If the blood of a sleeping patient be examined day or night, or that of a waking patient by night, the microscopic findings are usually simple and definite. If a large drop, a broad smear, or a large wet preparation be taken, no staining is necessary and low-power lenses are sufficient. Both *culex* and *anopheles* may act as intermediate hosts.

FIG. 565.—*Filaria loa* above; *filaria perstans* below. (*After Fülleborn.*)

Symptoms.—No symptoms appear in the majority of cases, the embryos indeed being innocuous. The parent worm produces at times either lymphatic varix or edema from lymphatic obstruction, hence there are produced a group of symptomatic conditions of lymphatic origin. (*a*) *Hematochyluria.* Chylous urine always suggests filariasis. Its appearance is described elsewhere and its cause is a leakage of chyle, usually from the lymphatics around

Often absent.

Chyluria.

the kidney, pelvis or bladder. The urine coagulates on standing and the contracted white clot ultimately floats in the milky liquid. The condition is almost symptomless, though rarely clots may cause retention or loin weariness may exist. (*b*) *Lymph scrotum* is merely lymphatic varix of the scrotal channels, and painless soft tumors, usually in both groins, also occur

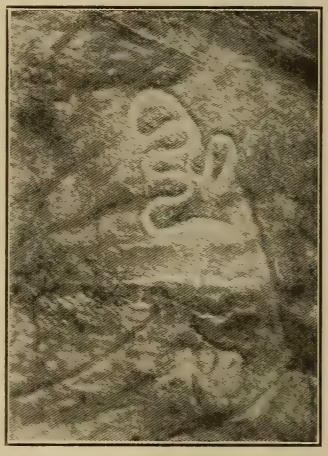


Fig. 566.—*Filaria loa* in the subcutaneous tissue, twice normal size. (*After Fülleborn.*)

Fig. 567.—Elephantiasis of the Feet. (*After Castellani and Daniels.*)

Fig. 568.—Elephantiasis of the labium. (*After Scheube.*)

Fig. 569.—Elephantiasis of penis and scrotum. (*After Hunt.*)

through varicose lymph glands. In either condition pain indicates lymphangitis. (*c*) *Elephantiasis and Elephantoid Fever.* These conditions occurring in the tropics are no doubt generally due to the Filaria, but this is not true

Fever.

of the elephantiasis of the temperate zone. The symptoms are chill, high fever, heat, redness, and congestion over the area of lymphatic involvement

with inflammatory induration which persists after the crisis. Abscess sometimes occurs. The specific pathogenic activity of the *Filaria diurna* is not yet established.

Dracontiasis (*Guinea-worm Disease*).—*The Dracunculus or Filaria medinensis.* The male worm is unknown, the female, cylindrical and smooth, measures 50 to 100 cm. by 2 to 3 mm., and carries a blunt hook on the head.

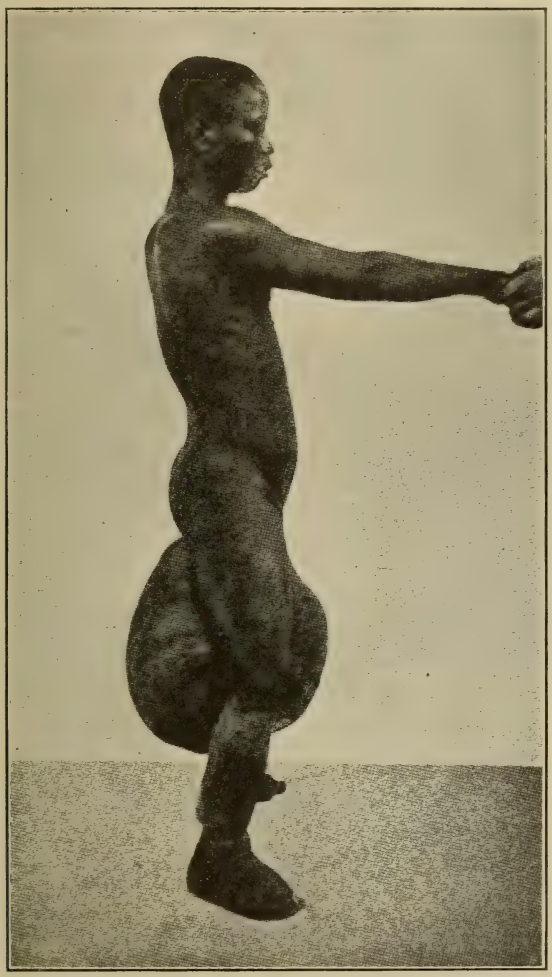


FIG. 570.—Elephantiasis of scrotum. (*After V. Schilling-Hannover.*)

The embryos are 500 to 750 micra by 25 to 30 and live in moist earth or muddy pools, finding an intermediate host in a small aquatic cyclops. It is found in India, Persia, Arabia, Africa, and Brazil. Rare cases have been reported in this country. Its preferred site is the subcutaneous tissue of the foot and leg, where it forms ulcers from which the head may protrude. Nevertheless the stomach is the portal of entrance, the worm penetrating the intestine and traveling downward to the lower extremity. If hot water be poured upon

the ulcer the embryos may in part be discharged and after spontaneous parturition the worm may voluntarily come forth.

Another method of gradual extraction is shown in our illustration.

FIG. 571.—Elephantiasis. Varicose glands and elephantiasis of genitals.

Trichuris trichiura (*Whip-worm*). This denizen of the cecum and colon measures from 40 to 50 mm. and has a thread-like anterior and thicker hinder portion, conical and pointed in the female, obtuse and often coiled in the male. The

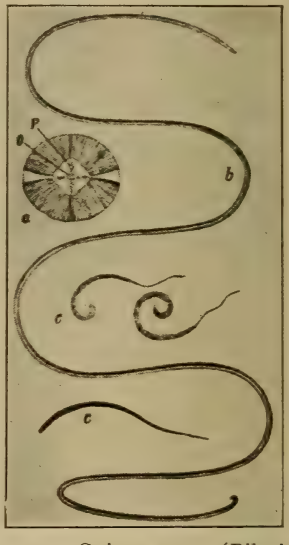


FIG. 572.—Guinea worm (*Filaria medinensis*). (*b*) female; (*c*) embryos; (*o*) mouth. (*After R. Blanchard.*)

eggs, 0.05 mm. in length, carry a bud-like projection. The only pathogenic symptoms associated with it are anemia and enteritis, and these are rare.

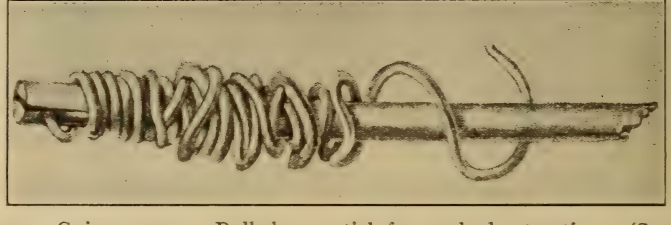


FIG. 573.—Guinea worm. Rolled on a stick for gradual extraction. (See text.)

Dicotophyme gigas or Eustrongylus gigas.—This foot-long worm, usually seen only in animals, is occasionally found in the renal region of man.

Strongyloides intestinalis.—This small worm measures 2 mm. by 0.06 mm. It is frequent in southern China and has been found in Manila,

Germany, Italy, and America. If present in great numbers they cause enteritis and anemia.

TRYPANOSOME FEVER AND THE SLEEPING SICKNESS.—The former is an irregular fever characterized by swelling of the spleen and lymph glands, weakness, and emaciation. The latter, its secondary stage, is a peculiarly latent and fatal disease native to Central and West Africa, and present in a peculiarly virulent form in Rhodesia (*Trypanosoma rhodesiensi*), is characterized by early apathy, mumbling, hesitant speech, tremor and difficult locomotion. The latent period may last five or more years, the increasing drowsiness becomes a deep and continuous sleep or coma, and the patient dies ordinarily of septic meningitis. The disease is fatal, occasionally attacks Europeans, and usually lasts from three months to a year, dating from the commencement of active symptoms.

Limited range.

Course.

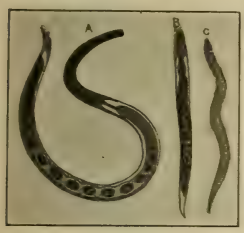


FIG. 574.—*Strongyloides inteslinalis.* (A) female, (B) rhabditiform larva, (C) filiform larva. (*After Braun.*)

FIG. 575.—*Trypanosoma gambiensis.* (*Dutton and Laveran.*)

The chain of proof is not complete, but it is certain that the *trypanosomes* are uniformly present in this disease and that they, respectively, can be carried and conveyed to man by the *Glossina palpalis* and *Glossina morsitans*,

Tsetse fly.

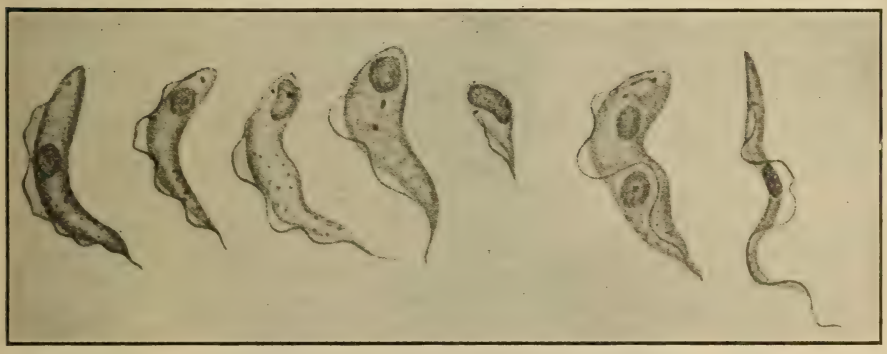


FIG. 576.—Different forms of *Trypanosoma rhodesiensi.* (*After Stevens and Fantham.*)

species of *tsetse fly*. The form of the organism is shown in the accompanying plate. It is small, actively motile, non-pigmented, and transparent, measures 10 to 20 micra and carries a curious flagellum on one side of its body. It is extra corpuscular and possesses an oval nucleus and a centrosome nearly opposite the attachment of the flagellum. It apparently multiplies by fission.

Parasite.

Brazilian Trypanosomiasis.—This curious and interesting form chiefly affects children and produces its chief effect upon the *thyroid gland* or the brain.

It is caused by *Schizotrypanum cruzi*, the carrier being a bug, ***Lamus megistus***, and in infants the mortality rate is very great, the ailment pursuing

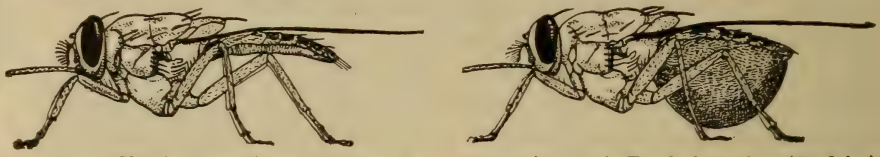


FIG. 577.—*Glossina morsitans.* Tsetse fly. Transmitter of *T. rhodesinsi.* (*Doflein.*)

FIG. 578.—*Glossina palpalis.* Tsetse fly. Transmitter of T. gambiense. (*Doflein.*)

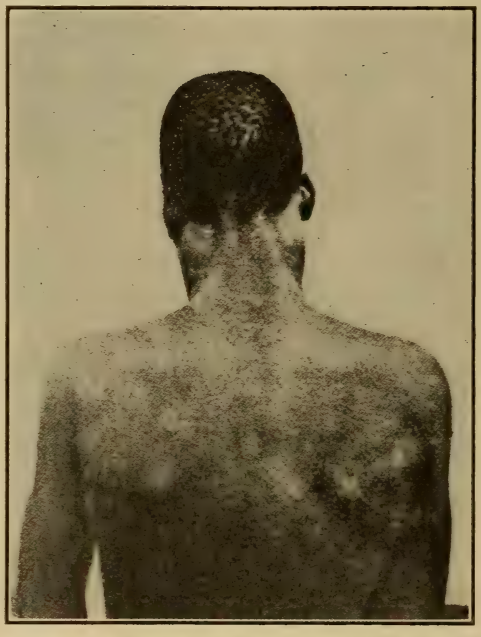


FIG. 579.—Sleeping sickness. Negro with the characteristic glandular swellings. (*After Koch.*)

an acute, rapid course with high fever, enlargement of the spleen, liver and lymph nodes and evidences of acute myxedema.

In the cerebral infantile form it is an equally fatal meningoencephalitis and those whom it does not kill it leaves hopelessly diseased.

In adults and in older children it pursues a chronic course, interrupted in many cases by febrile exacerbations, during which the causative organisms may be recoverable from the blood.

In this form the dominant symptoms may be cerebrospinal, those of myxedema, or even of Addison's disease (Stitt).

FIG. 580.—Terminal stage of sleeping sickness. (*After B. Nocht.*)

SARCOPTES SCABIEI (*Acarus scabiei*).—The female is the active agent, the male usually absent. The habitat is a burrow in the epidermis, often of the axilla or the front of the abdomen, and yet more commonly the finger webs and the lateral opposed surfaces of the digits. Characteristic scratch-marks and erosions direct attention to the burrows, which appear as short dark lines leading to a shining spot. The female, which may be seen with the naked eye, may be removed by passing a needle along the black line of egg deposit to the spot which indicates her presence.

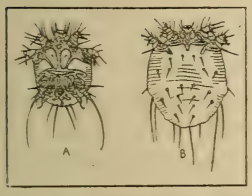


FIG. 581.—*Sarcoptes scabei.* A, Male. B, Female.

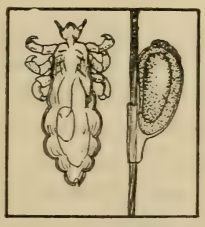


FIG. 582.—*Pediculus capitis.*

FIG. 583.—*Pediculus pubis.*

Pediculus capitis.—This produces irritation chiefly about the posterior margin of the scalp and the eggs or nits indicate by their position in the hair the duration of the process.

Pediculus corporis.—This should be sought in the seams of the clothing. The bites are dark, centered, hemorrhagic spots with a pale areola. Scratch-marks are, of course, common, chiefly over the upper back and shoulders. It is the chief conveyor of typhus fever.

Pediculus pubis.—This should be sought in and about the hair of the genital region, but is occasionally found in the axillæ and the eyebrows.

The *maculæ caeruleae* (*tache bleuâtre*) are subcutaneous bluish spots from 5 to 10 mm. in diameter, upon the abdomen and thigh, and are due to the irritation of body lice.

Vagabond's disease is a deep pigmentation resembling that of Addison's disease, but due to the constant irritation and scratching produced by the continuous presence of body lice.

Cimex lectularius (*bed-bug*) and **pulex irritans** (*flea*) need no extended description.

The sand flea or jigger (*Pulex penetrans*) is especially frequent in the West Indies and South America. It usually produces pustules or vesicles in the skin of the feet under which it grows and is considered more fully farther on.

THE MYIASES AND DERMATOPHILIASIS, CUTANEOUS MYIASES

The Screw-worm.—In the United States the most common affection due to the larvæ of flies is caused by the screw-worm encountered in some of our Southern states.

The fly, a blue-bottle *Chrysomyia macillaria*, lays her eggs in wounds showing an offensive discharge. The larvæ cause a horrible destruction of the soft parts by burrowing into them and the mortality is large, 15–25 per cent. in 23 cases reported by Younts (quoted by Stitt), 18 of which were nasal.

Ver Macaque Myiasis.—The name is that of the first larval stage of the gad-fly *Dermatobia cyaniventris*, a blue-bellied yellow-headed fly, common in Central and tropical South America.

They are supposed to be deposited in the wounds made by the bite of a large tropical mosquito to which they have been found *cemented*.

The gad-fly boils resulting are blind and extremely painful at first, breaking down later and discharging a purulent fluid.

The timber fly disease of Africa presents almost identical symptoms.

The Creeping Eruption.—This somewhat weird cutaneous manifestation results from the burrowing of fly larvæ, as yet unidentified, which march daily a distance of from one to several inches raising the skin above them to form a pinkish line which marks their advance.

They are found in Asia, Africa, South America and Southern Russia.

Dermatophiliasis.—The *chigoe* or "jigger" flea (*Dermatophilus penetrans*) infests the dry sandy soil throughout nearly the whole *tropical* world, attacking man and lower animals alike.

The site of election for the burrow of the impregnated female is the foot, and especially the lateral aspect of the toes near the nails. Other regions are frequently attacked, however, and hands, buttocks, scrotum, penis or labia may show the dark points within a tense, itching, white, pea-sized, or smaller, circle which marks the presence of the insect.

These points tend to ulcerate if the flea is not removed and the wound cavity sterilized with pure carbolic acid, followed by alcohol (Stitt).

CERTAIN DISEASES OF THE JOINTS AND MUSCLES, OF PROVEN OR PROBABLE INFECTIOUS ORIGIN

PRELIMINARY STATEMENT.—*The present status of the commoner types of joint disease, no less than those of the muscles, is such as to strongly suggest that they are of infectious origin, none, however, save those of a tuberculous or gonorrheal character, may be said to be absolutely and finally proven as to the specific causative organism. On the other hand, the clinical and experimental evidence tending to establish their infectious origin is sufficient already to justify their entire removal from the older grouping and to refer some of them with considerable confidence to specific bacterial agencies.*

ACUTE RHEUMATISM

Definition.—An acute febrile, non-suppurative inflammation of the joints, strongly suggesting an infection of the septic type, characterized by profuse acid sweats, the outpouring of an exudate within the synovial pouches and tendon sheaths of the joints affected, a peculiarly constant and significant relationship to antecedent and pharyngotonsillar infection, a decided tendency to recurrence and to the involvement of the endocardium, pericardium and myocardium during its course.

Fever, sweats and arthritis.

PROBABLE PORTALS AND AGENTS OF INFECTION.—The infectious nature of this acute non-suppurative arthritis, while technically unproven, is practically certain, and the older theories of perverted metabolism in which uric and lactic acid played so large a part must be regarded now as wholly obsolete.

The peculiarly direct and almost constant relationship of acute rheumatism to antecedent attacks of tonsillitis, the study of the bacterial flora of that ailment, the almost constant recovery of *Frederick Poynton's micrococcus rheumaticus* from the subserous connective tissue of the inflamed joints and the success attending the attempted experimental production of non-suppurative multiple arthritis, endocarditis, pericarditis and myocarditis in susceptible animals, by the use of pure cultures of this organism, are established facts of the utmost significance and importance.

Antecedent tonsillitis.

The pleomorphic micrococcus (streptococcus) rheumaticus is said to be merely a streptococcus strain lying between the streptococcus hemolyticus and streptococcus viridans, the two dominant organisms in acute and chronic tonsillar infections of the ordinary type. Poynton has shown that the organism readily disappears from the circulating blood, is inconstant in the joint exudate, but relatively constant in the subserous periarticular connective tissue.

Septic Foci.—The favorable results obtained in cases of recurrent acute or subacute rheumatism or even chronic (toxic) arthritis by the complete removal of the tonsils and, less often, by the elimination of other septic foci, peridental, antral, prostatic, cholecystic, appendiceal, and the like, together with the known streptococcic dominance in the bacterial flora of these conditions makes a strong case not alone for placing acute rheumatism under the head of infectious diseases, but for the specificity of the streptococcus strains lying between *S. hemolyticus and S. viridans.*

Potent in causation.

Predisposing Factors.—The existence of such chronically infected tonsils, carrying multiple strains of streptococci must now be considered as chief factors in predisposition to, and in the causation of, acute rheumatism.

The Tonsils.—*Acute tonsillitis is an extremely common ailment and clinical experience justifies the belief that practically all persons so affected or at least those who have suffered repeated attacks, carry the potential causative factors of rheumatism and many other ailments in their throats.*

Incubators and transformers.

The tonsils are natural incubators and furnish the exact conditions, not only for persistence of growth, but for the peculiar nutritional variations, and graduated oxygen supply, necessary to streptococcic growth.

Determine attacks.

Lowered Resistance.—*The potency of poor nutrition, bad environment, fatigue, and exposure to cold and wet as predisposing factors is clinically well established, as is also the effect of local injury to a joint.*

Season and Climate.—The disease is most prevalent in cold, wet, changeable climates and its incidence falls chiefly upon the late winter and the spring months (75 to 80 per cent.).

Age.—Maximum susceptibility is noted during the second and third decades of life, 75 per cent. of the adult cases occurring in males, though in childhood female preponderance is usual and occupation exercises a considerable influence with respect to the subordinate predisposing factors.

In infancy and old age it is rare; in children under ten, most trivial as to arthritis, and readily overlooked, *but extremely prone to involve the endocardium, pericardium, and myocardium or all together (pancarditis).*

Frequency.—In hospitals located in the slum districts of London, the admissions under this head may reach 10 per cent. of the ward population.

Heredity.—This apparently exercises a considerable influence in predisposition to streptococcic infection and therefore to acute rheumatism.

All possible grades.

Variability.—*Acute rheumatism, whether occurring endemically or epidemically, presents all possible variations in intensity. Furthermore a history of antecedent tonsillitis or pharyngitis in little children may be the only evidence of the disease, other than a slight tenderness of the joints often wholly overlooked even by the mother.*

The sore throat which precedes acute rheumatism may be present when the attack occurs, but usually precedes it by a period varying from several days to three weeks and usually is past when the polyarthritis occurs. The development of rheumatic fever in this respect presents the characteristics of "slow sepsis."

SYMPTOMS.—*The usual case presents an abrupt onset with moderate*

slight, or, in rare instances, excessively high fever, troublesome drenching sweats of a peculiar and quite characteristic sour, musty odor and a polyarthritis. Striking picture.

The larger joints are involved bilaterally in succession, usually beginning in one ankle or sometimes in some joint which may have been bruised or strained previously or represents that chiefly involved in occupational use, then passing to the corresponding joint of the opposite side.

In many cases the joints of the knee, ankle, wrist, elbow, and shoulder ultimately all become inflamed, red, swollen, tense, exquisitely tender, and more or less defensively fixed.

As suggested previously, any grade of involvement may be present from that of a *single joint* to an almost universal implication which does not even omit the spinal column, jaws, ribs, or the sacro-iliac or pubic articulations. Cases of universal involvement.

It is a genuine arthritis in all severe cases, sparing neither tendons, bursæ nor periarticular connective tissue, and according to the author's experience, some of the most persistent and viciously cardiac cases in adults begin and end as a rheumatic tenosynovitis. Tenosynovitis.

The condition of the victim of universal polyarthritis or the commoner multiple form constitutes one of the most pitiful of clinical pictures, but fortunately modern methods of treatment may prevent in most cases the extreme pain manifested under the older methods.

In spite of the extreme degree of inflammation the joints do not suppurate nor become ankylosed in true rheumatic fever. A fact of value.

FEVER.—*The fever rises with every new invasion or complication whether arthritic or cardiac and falls decidedly with every sweat.*

Chilliness is common and due largely to the almost constant dampness of clothing and bedding alike, the frequent changes of which add greatly to the patient's discomfort, however skilful and gentle the nurse.

The arthritis seldom persists in one joint for over one week and those first attacked may be wholly normal before the last affected show full development.

Duration.—"The cure for severe rheumatic fever is six weeks in bed" is an old saying, not only true, but often an understatement if both patient and physician are wise, though the actual duration of the active arthritis may be relatively short under modern management.

HEART COMPLICATIONS.—*It is wholly improbable that any case of acute severe rheumatism wholly spares the heart, but undoubtedly the myocardium, pericardium, and, in certain instances, even the endocardium, may be involved in what may prove to be but a temporary and relatively slight lesion.* Temporary cardiac lesions.

The myocardium itself rarely, perhaps never, wholly escapes and doubtless we have little conception of the damage wrought as relating to future degeneration of the muscle, becoming manifest only after the lapse of many years.

It is sometimes said that the danger is equally great in light and in severe forms, but it is probable that this is based largely upon the remarkable frequency of cardiac involvement in the acute rheumatism of young children showing slight articular lesions. According to the author's experience with the older children and with adults the danger of cardiac involvement is

certainly graver in the severe attacks, though heart involvement may, and often does, occur in the case of slight monoarticular lesions.

Frequency of Complications.—On the average at least one case in three will show a permanent residual endocarditis in a first attack and if one includes the cases in which the endocardium apparently is affected, but seems to clear up with convalescence, the percentage is raised to James Mackenzie's figure, 58 per cent. In young children 80 per cent. is given by some authorities.

Were cases watched, as they should be, always, for several months after apparent convalescence, we should raise our present figures by the inclusion of mitral stenosis and residual chronic myocarditis now largely overlooked on account of the slow development of definite physical signs.

Errors of Omission.—*The usual method of handling the rheumatic patient both during and after convalescence and the well-nigh universal failure to recognize the minor dilatations and the lesser signs of decompensation prevent, in many instances, a full recovery from the* **myocardial toxemias** (90 + *per cent. of the severe cases show demonstrable slight dilatation*) *or actual acute myocarditis* (30 *per cent.*), *which conditions the author believes constitute the chief and most direct causes of the persistent physical weakness present in, and often persisting long after, the severe attacks* (*see "Minor Dilatations,"* etc.).

Pericarditis *is a very common complication* (7 to 10 *per cent. in adults and* 10 *to* 20 *in children*) *and the physician must remember the frequency with which it runs a relatively painless course both during this intrinsically painful primary ailment and when itself dominating the field. He also must be careful to avoid the common error of mistaking a weak and widely dilated heart of myocarditic origin for pericarditis with effusion or vice versa.*

Recurrences.

Universal Involvement of the Heart Tissues.—*In children a severe pancarditis is relatively common, and with each recurrence of acute rheumatism or tonsillitis the danger of cardiac involvement increases, and old lesions are likely to be relighted.*

When a persisting endocarditis does occur, between 90 and 95 per cent. of the cases will show mitral involvement which in many instances proves ultimately to be a double (stenotic and regurgitant) lesion. Isolated aortic lesions constitute 5 per cent.; mitral lesions alone, 72 per cent.; combined aortic and mitral lesions, 18 per cent.*

Localization.

The Right Heart.—The right heart largely, but not invariably, escapes permanent recognizable *endocardial* damage though almost invariably more or less dilated in severe attacks. **The valves of the hardest worked side of the heart suffer most and oftenest.**

Rapid deterioration.

Blood.—As might be expected from the nature of the microörganisms apparently responsible for its occurrence, a rapidly induced anemia of a decided, but seldom extreme, grade, is a pronounced characteristic of acute rheumatism. It is of the secondary type, usually improves rapidly under

* The latter figures are those of A. R. Edwards. It should be remembered that all available statistics represent ward cases in public hospitals. Private practice amongst well-to-do people should yield much lower figures.

treatment when the stage of convalescence is passed, *but may be extreme or relatively intractable in some instances,** *an occurrence which usually means the continued activity of a focal source of infection. A moderate leucocytosis is the rule.*

Blood Cultures.—*Blood cultures are usually negative for the "Micrococcus (seu Streptococcus) rheumaticus" which does not reproduce itself in the blood or endure for long even in the joint exudate. Cultures from the exudate within the subserous periarticular connective tissue are said to be positive usually.*

Urine.—Albuminuria with an occasional cast and the usual findings characteristic of a urine of fever and toxemia are common, especially in the fully developed cases so largely represented in public hospital admissions. Febrile albuminuria.

Skin.—*Sudamina, white or red or usually both, are extremely common.*

Rheumatic nodes seem to represent a subcutaneous expression of rheumatism running somewhat parallel in occurrence to endocarditis and pericarditis with which complications they are usually associated. Their embolic nature and structure are practically identical with that of the endocarditic vegetations.† Curious parallelism.

Varying in size from that of a pin's *head* to that of a very large pea, or even an almond, they appear chiefly, though by no means exclusively, in children and during the third or fourth week of illness. They are localized usually over the region of the tendons, joint capsules or bony prominences almost everywhere. The wrists, fingers, elbows and spine are oftenest affected. Slightly stretching the skin over them makes them plainly visible by oblique light. Chronic cases often show them and in such cases some fibrosis is present. They may come and go within three or four days' time. Size and appearance of nodes. Distribution.

They are hard and painless and must be carefully distinguished from the tender and painful cutaneous nodules of malignant recurrent endocarditis. Important distinction.

Erythema multiforme and purpuric rashes are not uncommon.

Gastrointestinal Tract.—Constipation is the rule and appetite is usually wholly lacking until convalescence is well under way.

Respiratory Tract.—Aside from the rare *crico-arytenoid arthritis*, the still rarer acute laryngeal edema, terminal pulmonary edema, and infarction arising from complicating endocarditis or myocarditis, the only pulmonary complications are the pleurisies and pneumonias so frequently associated with rheumatic pericarditis and pancarditis. Complications of a complication.

Nervous System.—*Cerebral rheumatism is a rare and relatively fatal complication indicated by hyperpyrexia, delirium, convulsions and coma. The hyperpyrexia may be lacking in some cases.*

It may be *preceded* by vomiting, headache, delirium, excessive insomnia, cutaneous irritation or frequent micturition. It should be borne in mind

* Jochmann quotes Grawitz as stating that in rheumatism the hemoglobin content, blood count, and specific gravity *are usually unaffected.* Such a statement is wholly out of accord with the findings of such careful investigators as Poynton and his co-workers and certainly opposed to the personal experience of the author. Furthermore, it is wholly out of harmony with the clinical type of the disease.

† Poynton and Payne have shown sections of the nodules showing them to be the result of bacterial thrombi and resulting exudation necrosis.

that, although it usually occurs in the third or fourth week, *it may arise during apparent convalescence.* About 50 per cent. of such cases die.

Chorea is extremely common in children who have had rheumatism, especially so in those carrying a chronic endocarditic lesion.

Poynton states that of 500 juvenile cases of heart disease reporting for treatment, 220 came "from chorea." Of these 220 cases, 122 had obvious organic heart disease (51 per cent.); "thirty-eight had dilatation with rheumatic arthritis and pain" (17 per cent.); ten followed a sore throat (4 per cent.); four developed later demonstrable organic heart disease—81 per cent. in all more or less directly showing a rheumatic association.

Joint abscess.

DIFFERENTIAL DIAGNOSIS.—Septic arthritis is usually associated with *rigors*, a higher leucocyte count, the frequent existence of a known gross pyemic focus, a purulent joint exudate and *lack of early recession.* Theoretically, the differentiation may be difficult; practically, this is seldom the case.

Pneumococcus Arthritis.—This is seldom polyarticular though two joints may be affected. The joint exudate may show the pneumococcus.

Acute Tuberculous Arthritis.—In the acute stage tuberculous arthritis *may* be clinically undifferentiable, *but it is usually actually or dominantly monoarticular and persistent,* the onset is less sudden,* the fever less high; an active pulmonary focus may often be demonstrated, and the previous health and family history may be most suggestive.

Certainly no mistake is likely to occur if the clinical history of acute rheumatic joint swelling is held in mind.

Seldom obscure.

Scarlatinal Rheumatism.—*This is distinctly a serous or, less often, septic arthritic complication of a known case of scarlet fever (4 per cent.) and can seldom be called "out of its name." It occurs usually during the second week of this disease and, according to Ker, almost invariably appears if the scarlatina victim previously has passed through an acute rheumatic arthritis.*

Miscellaneous Secondary Arthritides.—In many other diseases arthritis is a known complication, a more or less distinct possibility, or a recognized component, and the primary disease names the associated joint disturbance. Such are pneumonia, cerebro-spinal meningitis (serous or septic), typhoid fever ("typhoid spine"), Malta fever, dengue, *peliosis rheumatica*, secondary syphilis, influenza, etc.

Anterior Poliomyelitis.—In the early stage multiple joint pain may be present but every other sign of acute rheumatism is lacking.

Osteomyelitis.—Every case of apparent joint inflammation in the child must raise the question of possible osteomyelitis, for mistakes are frequently made, too often without justification, for in most instances the swelling of osteomyelitis is unlike that of rheumatism in appearance, feel, and location.

It is maximal at the epiphyses, local and constitutional symptoms are more intense, leucocytosis is usually high, blood cultures are often positive and rigors are common.

* Unfortunately certain cases of *juvenile* rheumatism also develop slowly over several or many days.

Localization *of the inflammatory focus and prompt operation are the chief requisites in osteomyelitis, and here an error in diagnosis usually costs a life.*

Acute Arthritis Deformans.—*This cannot be differentiated with certainty from acute rheumatism by any means known to the author until its intractability to antirheumatic remedies of known potency and its persistent course become manifest.*

In the few cases seen by the author a significant persistence or unusually long duration of the individual joint swelling has been present. *In these cases one might see all affected joints in full career for a considerable period, a condition quite contrary to the rule in acute rheumatism.* Predilection for the wrist-, finger-, ankle-, and toe-joints often exists, but can only serve to arouse suspicion as such involvement may be decided in true rheumatic arthritis.

Gonorrheal Arthritis.—A monoarticular arthritis affecting the wrist or knee alone is in itself sufficient to arouse suspicion, no less than its peculiar fixity or sluggishness, relative intractability and slight or absent response to anti-rheumatic remedies.

When polyarticular it might confuse diagnosis, yet the persistence of the joint swelling is usually suggestive.

Furthermore, any acute gonorrheal polyarthritis is almost certain to be associated with a readily demonstrable urethral or vaginal discharge.

Direct detection of the causative coccus is then easy and the complement fixation test is definite.

Recurrence in Acute Rheumatic Arthritis.—*Unless the foci existing at the portals of infection are eliminated, recurrence is common when the malign conjunction of excessive fatigue, exposure to cold and wet, and "sore throat," arises, and relapses may occur repeatedly and extend over several months.* Foci must be eradicated.

In the latter case one may be obliged to consider tonsillectomy or other necessary procedure even during the attack itself.

The younger the individual once attacked, the greater is his vulnerability to renewed attacks and his danger from cardiac complications.

PROGNOSIS.—The direct mortality is trivial; the indirect, deferred and remote death-rate is frightful. No disease outranks rheumatism in importance and it kills almost wholly by the complications it induces. Ultimate mortality great.

ARTHRITIS DEFORMANS.—Preliminary Comment.—Although the specific cause of this formerly hopeless and progressively crippling ailment cannot be positively stated at the present time, it seems to be clearly shown to be the result of infection and to arise in most instances from chronic cryptogenetic foci containing streptococcic strains strikingly resembling or identical with the *Streptococcus viridans*.

The gratifying results obtained in many instances and by many individuals, including the author, by the institution of radical procedures in the removal of chronic foci of streptococcic infections have been both striking and illuminating, and it is altogether probable that within a reasonable time we may speak definitely as to the specific etiologic factors in relation to this disease.

Affects Women Chiefly.—This chronic and obscure disease of the joints chiefly affects women and in them is frequently associated with the menopause. It is most common between the ages of thirty and fifty, but not rare in younger persons, and differs from gout in the absence of arthritic deposits of sodium urate, and from so-called chronic rheumatism in its marked atrophic or hypertrophic changes in the cartilages and bones.

Three Types.—*The three recognized types depend upon both pathologic and symptomatic differences and we recognize a predominance of changes in* (*a*) *the periarticular and synovial structures,* (*b*) *predominating atrophy of bone and cartilage,* (*c*) *hypertrophy.*

Haygarth's nodosities.

Osseous proliferations occurring at the joint margins are known as osteophytes, or, on the knuckles, *Haygarth's nodosities.* Any joint and even the whole spine may become ankylosed. Marked deformity and muscular atrophy and contractures are often associated with neuritis and atrophic changes. The lateral nodules frequently seen on the distal phalanges are known as *Heberden's nodes.* They are seldom or never associated with cases involving the larger joints. Single joints may be involved, the spinal column, shoulders, hip, and knee being the common sites. This form is most common in men and at advanced ages.

Heberden's nodes.

"Poker spine."

The spine alone may be involved (spondylitis deformans, "poker back") associated with ascending degeneration of the cord, and pain, muscular atrophy and anesthesia, due to involvement of the nerve roots. If the hip and shoulder joints are also involved (*spondylose rhizomélique*) the nervous symptoms are moderate or absent and the whole or only a part of the spine may be affected in certain instances.

Acute form like rheumatism.

General Progressive Arthritis Deformans.—The onset of the acute form exactly simulates subacute rheumatism and differentiation is at first impossible. The absence of a complicating endocarditis and the persistence of the inflammation in the joints first affected, together with the lack of reaction or response to anti-rheumatic medication, are points of value, strengthened by the later development of fixation and crepitation. Mental depression and emaciation are marked and the attacks frequently bear a relation to lactation and child-bearing or other nutritionally depressing influences.

Exhaustion and malnutrition.

The chronic form usually succeeds one or more acute attacks, and there is pain on movement, inflammatory swelling, and a variable amount of effusion. The tendency is to gradual progression and symmetrical involvement, the hands, knees, and feet being usually the earliest points of attack.

The ordinary form of chronic, or subacute recurrent, rheumatism, associated with a history of previous rheumatic attacks and lacking, as a rule, any *extreme* deformity or ankylosis, is usually of relatively slight importance. *In gouty arthritis* there are deposits of sodium urate in the soft parts which lie just beneath the skin and often perforate it, differing from the nodules of hypertrophic arthritis in their mobility. The apparent deformity is often very marked, the actual destruction relatively slight.

Pain variable

Pain may be moderate or extreme and is of the neuritic type. Crepitation appears, there is marked deformity, and finally a firm ankylosis due to

periarticular infiltration and adhesion. The muscular atrophy, usually that of disuse, is nevertheless marked and may be so rapid as to suggest a central cause, which presumption is strengthened by the glossy or pigmented skin, onychia, and paresthesia. The anemia may be marked and the disease may render the patient absolutely helpless.

Crepitation and ankylosis.

Trophic symptoms.

CHRONIC OR RECURRENT INFECTIOUS ARTHRITIS (*Toxemic Arthritis, Chronic Rheumatism*).—In the light of modern knowledge with respect to the etiologic factors active in cases of acute and subacute rheumatism, and noting the pronounced tendency to exacerbations evident in this formerly obscure ailment, together with their relationships to antecedent tonsillar infection or the presence of cryptogenetic septic foci, one can hardly doubt the correctness of the old view that it represents chronic rheumatism, and is primarily *of streptococcic origin.**

Foci of Infection.—The most astonishing results have followed the work of both dental and medical colleagues of the author in the hospitals of the University of Minnesota and in private office practice following the radical removal of diseased tonsils, the cure of chronic sinusitis, the correction of peridental infection, *and the establishment and maintenance of improved nutrition.*

It should be stated that in many of these cases, as in those of "*arthritis deformans*," many of which show a most decided and favorable response to the same measures of treatment, the disease may persist for several weeks or even months, usually in an attenuated form, even though actual progress may have ceased. It has seemed probable that in some of these cases a residual streptococciosis, of the rheumatic type in the former group, viridans in the latter, tended to persist and for a time undergo reproduction in the subserous synovial tissue.

STILL'S DISEASE.—This is a chronic progressive arthritis *especially affecting children during the first dentition*, is insidious with respect to onset and course alike, appears first in the knees or wrists, rather than in the small joints as is the case with its congener in the adult, and is associated with leucocytosis, enlargement of the spleen, swelling of the glands tributary to the joints, subnutrition, and arrest of development in many instances.

SYPHILITIC ARTHRITIS (*Syphilitic Rheumatism*).—Luetic arthritis is never generalized and though the acute form involves several joints, more than three are seldom affected. The subacute form is usually monarticular. Beyond a mild remittent fever a variable amount of localized pain and tenderness and its prompt response to properly applied specific treatment, the ailment offers nothing of special importance. The fact that it occurs usually *at the time of onset of the secondary manifestations of syphilis* makes opportunities for error negligible.

GONORRHEAL ARTHRITIS (*Gonorrheal Rheumatism*).—*This troublesome and persistent complication may result from infection of any mucous membrane with the gonococcus of Neisser and, as in the case of acute rheumatism any antecedent joint injury, strain or inflammation seems to invite arthritic*

* The author feels that as a title, *recurring subacute arthritis* would best suit the greater number of these cases.

localization of the organism. In most instances an active urethral or vaginal discharge exists at the time of the onset of the arthritis, but if, as is commonly the case, relapses or recurrences arise, all such evidence of its true causation may be lacking.

A myth.

It was supposed formerly that male predominance was very marked, but this is not wholly true and, in large public clinics especially, *Neisserian arthritis* is nearly as common in the female as in the male. Under the same conditions, it is astonishingly frequent in children and the specific infection is readily disseminated in public schools or other institutions of a similar type. In children the disease is frequently or generally polyarticular and may affect both the large and small joints. In the adult the knee-joint is primarily affected in about three-fourths of the cases. The active inflammation and pus formation are usually lacking and the joints may be either relatively painless or excessively tender.

Children.

An aid to diagnosis.

According to the author's experience the *acute polyarticular* cases are fortunately associated with an active visible or demonstrable genital infection in nearly every instance.

Incubation Period.—With respect to its onset in relation to the date of the primary infection, we find that it is usually postponed for three or four weeks, but that it may occur during the first ten days.

Mode of Onset.—Its onset is usually relatively *gradual*, associated with a moderate elevation of temperature and in most instances unattended by chill. Its duration is extremely variable but chronicity and relapse constitute the rule and a considerable degree of atrophy of disuse or actual deformity may result.

Localization.—As previously stated, the disease is usually monarticular involves by preference the wrist or knee joint, but may also affect the shoulder, wrist, hands, feet, hip, sacro-iliac joint, temporo-maxillary or even the sterno-clavicular articulations.

Diagnosis.—The polyarticular cases are quite commonly mistaken for an acute rheumatism, especially if the examination is superficial or any active discharge from the urethra lacking. The ailment, however, varies strikingly from acute rheumatic arthritis in the behavior of the joints affected both as to acuteness of onset and relative promptness of recession on the part of individual joints. The superficial signs of inflammation are less marked. Tenderness usually but not always much less extreme.

Unlike acute rheumatism.

The diagnosis is usually made relatively easy by the recovery and staining of the gonococcus from an active or latent discharge and the complement-fixation test is positive.

Complications.—Heart complications are fortunately rare but when they do occur may prove serious and intractable.

STATIC AILMENTS.—Flat-foot, relaxed sacro-iliac joints, chronic bursal inflammations and the like call for no consideration in this volume but belong to the surgeon and the orthopedist. It may be stated incidentally that in many cases of sacro-iliac relaxation apparently responsible in some instances for persistent pain in the sacro or lower lumbar region are distinctly associated with the visceroptotic manifestations of universal congenital asthenia.

ACUTE NON-SUPPURATIVE POLYMYOSITIS

(*Dermatomyositis*)

Extraordinary combination.

Definition.—*A rare febrile ailment representing an acute, progressive interstitial myositis combined with dermatitis and edema of the tissues overlying either the flexor or extensor surfaces of the muscles affected.*

Etiology.—The exact cause is unknown, but the entire course of the disease, together with recent investigations relating to its bacterial flora, indicate its infectious nature. No important predisposing factors are constant and the disease usually attacks persons in full health.

Symptoms.—A sudden febrile onset, lacking chill, with general malaise, and considerable pain and tenderness over certain muscles, is the first indication. This is followed by swelling and induration of the affected parts and associated with increased fever and usually a dermatitis and edema which destroys the normal contour of the region involved.

Progresses by groups.

In most instances the muscles are involved in succession and by groups, and to such an extent as ultimately to make the process practically universal. In certain cases, however, only a few groups are affected. The edema, on the other hand, usually involves at first only the extensor or flexor surfaces, giving the affected regions a peculiar and somewhat characteristic appearance. It was supposed formerly that the muscles of the eye, larynx and tongue were spared, but many cases are now reported in which these were affected as well as those of the pharynx, the latter complication often leading to a fatal termination through inability to take food.

Peculiar distribution.

Serious complications.

Speech may be affected by the involvement of the muscles of the tongue and in many cases severe and fatal asphyxia occurs from involvement of the muscles of respiration or the patient dies of broncho-pneumonia.

Misleading resemblance.

The edema may not pit on pressure and as seen in the face and eyelids gives the skin a tense, alabaster-like immobility suggesting acute Bright's disease or trichinosis.

Tenderness is usually extreme and the *dermatitis* may be associated with or substituted by bullæ, eczema, erythema or urticaria, or strongly resemble true erysipelas.

"Neuromyositis."

The nerves ordinarily are not involved though a certain form usually described under another name presents both neuritis and myositis, this conjunction being probably merely fortuitous.

Line of march.

The proximal muscle groups are usually first involved and, fortunately, the wrist- and ankle-joints are ordinarily spared any excessive involvement. The disability and helplessness of the patient together with the pain which steadily increases makes a pitiable clinical picture.

Duration.—The duration of the ailment is usually from two to eight weeks. In the subacute form it may be from four to nine months, or become chronic and endure from one to two years.

The mortality is extremely high, even under modern treatment averaging about 60 *per cent.*

Differential Diagnosis.—*Trichinosis* presents a superficial resemblance in its muscle pain and disability as well as the swelling of the face, but is

readily differentiated by the lack of trichinæ in the excised muscle and in the stools no less than in the striking and somewhat characteristic outward symptoms present in this form of myositis and the high grade of *eosinophilia* invariably present in trichinosis.

Primary suppurative myositis can hardly be confounded with the non-suppurative form because of its distinctly septic type of fever, profuse sweats, localized indurated swellings and its occurrence in one of three forms, each of which asserts its character at the outset, the one being multiple from the start, the other a single focus from which multiple areas of localized and circumscribed suppuration develop later, the third, infiltrating. The entire course is radically different from that of the non-suppurative myositis.

Syphilitic myositis is usually discussed but in none of its forms does it bear any decided resemblance to the disease now under consideration.

HEMORRHAGIC POLYMYOSITIS.—Definition.—A disease of unknown origin affecting the muscles, resembling non-suppurative myositis, running an acute, subacute or chronic course and characterized by interstitial hemorrhage between the muscles, a tendency to a purpuric or morbilliform exanthem, an almost invariable tendency to serious involvement of the myocardium, and frequent nephritic complications.

Peculiar exanthem.

Etiology.—No specific organism has been proven though staphylococci have been present in some cases. Nevertheless its infectious character cannot well be doubted, and it usually follows tonsillitis.

Pathology.—The changes are primarily those of intermuscular hemorrhages and degeneration of the muscle fibers succeeded, in the chronic stages, by an extensive production of connective tissue.

Symptoms.—In general these are almost identical with those of non-suppurative acute primary myositis and the same tendency to involvement of muscle groups in progressive succession is shown. The pain is sharply circumscribed and definitely localized. The onset is more abrupt than in the non-hemorrhagic form and the fever somewhat lower. *Symptoms of decided cardiac involvement appear early and collapse is a frequent occurrence.* The peculiar skin eruption is striking and somewhat distinctive in its combination with an unmistakable primary myositis. Hemorrhage may occur from mucous membranes *and nephritis is extraordinarily frequent as a complication.*

Differential Diagnosis.—No confusion is possible if the striking and characteristic symptom-complex is held in mind.

Prognosis.—Its mortality probably is about that of the acute non-suppurative myositis, viz., 60 per cent.

ACUTE PRIMARY SUPPURATIVE MYOSITIS.—Definition.—An acute myositis presenting the clinical picture of an acute septic infection and unquestionably due to the action of pyogenic organisms. Presumably it is a secondary septic infection with a peculiarly specific localization in most instances.

Three forms.

Varieties.—It occurs in three chief forms: one, disseminated foci; two, the large isolated abscess; three, diffuse purulent infiltration. A large proportion

of the cases show but a single suppurative focus, and the pathologic changes described by various observers are only those that would be anticipated in an ailment of this peculiar character and localization.

Symptoms.—The onset is abrupt and florid, an initial chill being followed by high fever, profuse sweating and the general malaise and aching characteristic of the toxemia of severe, acute infections. Pain, at first general, progressively increases and becomes definitely localized in the muscle or muscles affected and these become the seat of tender, painful swelling involving the entire muscle and of an extremely indurated resistant character. Slight, overlying edema may be evident, the muscle is contracted and the part affected completely disabled. In nearly every instance the primary swelling undergoes the usual changes incident to abscess formation and demands prompt and radical surgical intervention which is usually followed by recession of symptoms and complete recovery after an interval of several weeks. If the abscess is neglected the usual dangers of general pyemia are encountered. Death may occur from this cause or from pneumonia but nearly all cases recover under proper treatment without permanent disability of the part or parts affected.

Septic manifestations.

SYPHILITIC MYOSITIS.—Luetic myositis may occur in any one of three different forms, two of which pertain to the secondary stage; the third to its early tertiary period. The secondary forms are: (1) A peculiar swelling slowly becoming indurated; (2) a circumscribed or diffuse primary infiltration, painless and associated with no tenderness except at the point of insertion of the tendon of the muscle affected.

Three forms.

In this latter form, or perhaps in both, fever may be present but is of a moderate grade and the disease shows a peculiar and characteristic predilection for two muscles of the body, viz., the *biceps and the rectus femoris*.

Comment.—It is important that these secondary manifestations be borne in mind for in them as in the tertiary form of luetic myositis which is merely that of gummatous deposit, that which is a troublesome somewhat persistent and to some extent disabling lesion will vanish promptly under antisyphilitic medication. The tertiary syphilomata if promptly treated will recede usually without breaking down and forming scars. If not so treated they may soften and discharge outward. Inasmuch as the tongue together with the sternomastoid muscle are the favorite sites, considerable discomfort and humiliation may be avoided by prompt therapeutic measures. It is unnecessary to remind the reader that whatever the *preferential* location of such syphilitic lesions may be, *any muscle* may be affected.

Responsive to specific medication.

TUBERCULOUS MYOSITIS.—This exceedingly rare form of myositis may complicate a miliary tuberculosis and doubtless represents one variant of primary hematogenous tuberculous infection. It may involve one or many muscles, occurs in rare instances as a complication of chronic pulmonary tuberculosis and should be remembered as a remote but not unimportant clinical possibility.

Excessively rare.

MYOSITIS FIBROSA.—This condition is one of pathological interest chiefly, wholly lacking constitutional symptoms and, usually, absolutely or

A medical curiosity.

relatively painless, save in those cases in which an extensive transformation of muscles into fibrous tissue occurs as a result of months or years of development.

Etiology.—It is said to be associated with chronic simple or septic inflammatory changes in the muscles primarily or in neighboring joint structures and occasionally follows extensive, long-sustained skin ulcer or eczema.

"The ossified man."

MYOSITIS OSSIFICANS.—Definition.—An extraordinary and excessively chronic disease constituting one of the curiosities of medicine and characterized by an excessively gradual transformation of the muscle fibers and interstitial connective tissue of the aponeuroses, tendons, and their sheaths into bony tissue following a stage of painful swelling of the affected structures.

Primary and Secondary cases.

Varieties.—There are two chief forms: one, a spontaneous progressive *polymyositis ossificans* developing in youth or early adult life and primarily involving in most instances the muscles of the back and neck; second, a secondary form apparently owing its inception to chronic injuries or preëxisting disease of the joints, excessive muscular strain or like conditions. The pitiable victim of this ailment often falls into the hands of the proprietors of side shows where he is shown as the "ossified man."

MUSCULAR CRAMP.—This constitutes one of the most painful and distressing of the relatively common affections and would seem to fall into two groups with respect to etiology. First, the cases immediately or at least definitely attributable to excessive muscular activity, strain or exhaustion as affecting specific neuro-muscular units, conditions frequently seen in connection with forced marches, acute overexertion on the part of such as are unfit, or, the chronic fatigues of certain monotonous occupations demanding the persistent daily use of certain muscle groups.

Two etiologic types.

Fatigue.

Second, the toxemic cramps associated with chronic nephritis, diabetes and many other ailments acute or chronic.

Toxemia and vascular disorders.

Symptoms.—These represent merely an involuntary, extreme and, usually, excessively painful, spasmodic tonic contraction of the muscle group affected, this in the commoner form being the muscles of the calf of the leg. Such attacks may be brief or prolonged, recurrent, isolated, or, in certain occupational neuroses, may attain a definite chronicity of occurrence.

MYALGIA (*Muscular Rheumatism*).—**Preliminary Comment.**—It is wholly probable that the term "muscular rheumatism" more accurately expresses the basic etiology of this formerly obscure and extremely common ailment than does the term "*myalgia.*" Recent investigations suggest that the causative factor in this excessively painful and troublesome ailment must be sought in the cryptogenetic foci so potent in relation to the causation of acute rheumatism and many other less concrete ailments. Like rheumatism, this so-called myalgia occurs frequently as a sequel of tonsillitis and is peculiarly likely to develop after exposure to cold and wet under conditions of fatigue or overheating of the body.

Preferential Sites.—Myalgia usually takes the form of lumbago, torticollis or so-called pleurodynia, though almost any muscle group may be involved if the predisposing or determining factors mentioned are present.

Symptoms.—Save in the case of pleurodynia the amount of pain present in this ailment is dependent chiefly upon the voluntary use of the muscle group affected. In the former, movement is obligatory, but, in general, spontaneous pain is relatively slight or wholly absent, though localized tenderness may be extreme.

Diagnosis.—The presence of tenderness over the *muscles* involved and the prompt pain response to movement make the diagnosis. Care must be taken lest a persisting torticollis due to organic disease be mistaken for myalgia, which is ordinarily of but a few days duration, and *pleurodynia must never be accepted as genuine without a careful examination of the heart, lung, and pleura.* Furthermore, a strikingly similar pain of true intercostal neuralgia characterized by tenderness along the course of the nerves affected, paroxysmal pain independent of movement and definite points of maximal tenderness must be borne in mind. Another condition of which the preceding ailment may be the precursor is "*herpes zoster*," one of the most excruciatingly painful of all minor afflictions and characterized by its long-deferred declaration of identity *in the form of multiple herpetic vesicles* at the known points of superficial, cutaneous, intercostal distribution.

Torticollis.

Pleurodynia.

"Shingles."

CERTAIN IMPORTANT CONDITIONS OF UNKNOWN CAUSATION OR LACKING A PROPER DESIGNATION

DIABETES MELLITUS

Definition.—*A disease of general metabolism of unknown causation characterized by a permanent inadequacy on the part of those organs or tissues normally charged directly or indirectly with the storage, fixation, consumption or orderly release and proper distribution of grape-sugar; such inadequacy being manifested invariably by an excess of sugar in the circulating blood, by resultant glycosuria, and, in the established and advanced disease, by a ketone acidosis.*

Endocrin disturbance.

A Modern Hypothesis.—The pancreas is functionally a gland of both internal and external secretion and in its so-called "islands of Langerhans" it possesses tissue elements embryologically and functionally distinct from that of its glandular acini which are concerned with the production of its external secretion and strikingly resistant to many adverse conditions which cripple or destroy the body of the pancreas.

Islands of Langerhans.

These islands, the so-called "insular apparatus," are believed at present to produce normally a *hormone* which is essential to the adequate control of glycogenesis and the formation and elimination of acetone (ketone) bodies in the human organism, and to be in great measure independent of the pancreatic tissues concerned in external secretion.

Hormone production.

A proper balance between the action of this pancreatic hormone and adrenalin is thought to be indispensable, the former building up glycogen in the liver and muscular system; the latter breaking it down and accelerating its transformation into sugar.

Balanced activites.

The old experiment of Claude Bernard, who produced glycosuria by

puncture of the floor of the fourth ventricle, in all probability depended upon the production thereby of a splanchnic stimulus to the chromaffin tissues which resulted in the unloading of an excess of adrenalin and a dominance of the adrenalin influence over that of the insular pancreatic hormone devoted to conservation and supply.

Falta summarizes the subject admirably in the following words:

Action of pancreatic hormone.

"*The pancreatic hormone is an exquisitely assimilatory hormone, and governs glycogenesis in the liver and muscles. In the light grades of insufficiency the disturbance in carbohydrate metabolism occurs only when there are instituted great demands on glycogenesis in the liver (alimentary overloading with carbohydrates). In the severer disturbances there occurs, in addition to the disturbance in anabolism, a marked increase in catabolic processes and thereby a faulty decomposition of higher and lower fatty acids (ketonuria).*"

Diabetes does not depend wholly upon a disturbance of function or anatomic integrity of the pancreas.

It would appear that two distinct elements enter into clinical diabetes mellitus; one, the disturbance in storage, proper distribution, and assimilation preliminary to properly regulated combustion; the other, to an excessive and illy-regulated transformation of the alimentary intake into sugar.

Associated with the former there is an imperfect transformation of albumin and fats which results in the excessive formation of acetone bodies (acidosis).

The introduction of Allen's methods of treatment makes the prognosis better than ever before. Carbohydrate tolerance can now be accurately and scientifically determined and fewer patients die from faulty dieting.

Neurogenous diabetes.

It is evident that the adrenals and the chromaffin system may play a chief part in some instances and so-called "neurogenous diabetes" is thought to be the result of persistent stimulation of the chromaffin system, these originating in the higher centers and acting through the splanchnic and many glands other than those mentioned may play a part.

The scope of this volume permits no further advance into this fascinating realm and we must turn to the more prosaic clinical aspects of diabetes mellitus.

Glucose.

Clinical Definition.—*In a purely clinical sense* glycosuria comprises all conditions under which the urine contains grape-sugar (glucose) in sufficient quantity to respond to the ordinary tests. *In short, all diabetics have glycosuria, but all who have glycosuria are not diabetics.** Sugars of various kinds may be found in the urine, but glucose is the one of especial interest to the physician. *Normal urine contains it, as it does albumin, but not to an extent that can produce any confusion in clinical work; hence any urine that shows sugar, when tested according to methods here recommended, is abnormal.*

Clinical glycosuria.

Intermittent cases.

Furthermore, glucose or dextrose may appear either persistently or intermittently in the urine of apparently healthy persons and no sharp line separates the benign from the pathologic condition, save that suggested by Stengel, who says that all cases fall under one of two classes, viz.: *First,*

* It must be remembered that in *diabetes* sugar may be elaborated from substances other than carbohydrates.

those easily controlled (*simple glycosuria*); *second, those that are relatively intractable* (*diabetes mellitus*). The urine of one who takes into a fasting stomach phloridzin or large quantities of glucose, a woman recently delivered, or the man with a brain injury may temporarily contain sugar. The stout high-living individual may carry such a condition for many years (lipogenic diabetes) without serious impairment of health; *but lapse of time alone suffices to prove the benign nature of any given case.* Glycosuria may entirely disappear for long periods, only to recur, and, not infrequently, to assume a malignant form. Such periods of latency cannot be justly estimated, and the very causes that are assigned for benign glycosuria are quite commonly associated with the development of diabetes mellitus. The conditions under which pathologic glycosuria develops are in brief the following:

Temporary recession.

Age.—It occurs at all ages, but chiefly in obese individuals in their fifth and sixth decades. Its prognosis is inversely as the age of the individual. Children rarely recover save when the disease onset is mild, recognition prompt and treatment early rational and efficient, and in them the course may be astonishingly acute.

Sex.—Eighty per cent. of the cases occur in males.

Race.—Certain races suffer greatly from disease. In Tunis and in Malta its ravages are comparable to those of tuberculosis in European countries. This, however, applies chiefly to city dwellers. Hebrews are especially liable to the disease. An enormous increase has been noted in certain cities during the last three decades. In Danish cities and in Paris, for example, the mortality is said to have quadrupled during that period. Due allowance must be made for improved diagnostic methods.

Influence marked.

Heredity is very marked. Schmitz reports that diabetes has occurred in the blood relations of 998 of the 2115 individuals whose cases he had investigated, fourteen cases having been reported in one family. It is said to be present in 20 per cent. of all cases.

Diet.—Alimentary glycosuria is under certain conditions quite unimportant. A definite limit may be placed to the amount of sugar that should be disposed of under normal conditions, without producing glycosuria, and various writers have suggested a test amount varying from 100 to 250 grams, this to be taken at one sitting, and on a fasting stomach. If sugar then appears in the urine, the individual is a suspect.

Test of tolerance.

A simple rule provides that one who can take 50 grams of *grape-sugar* on a fasting stomach and show no reaction in the urine is absolutely normal. If not more than 1 per cent. is demonstrable, he may be held as merely *functionally inadequate* unless it be found that carbohydrates alone cause glycosuria.

Exercise.—There can be little doubt that a rich diet, associated with sedentary habits and great mental activity, are potent predisposing factors in causation.

Gout.—Gout is distinctly associated with diabetes, though it is claimed that the arthritic form is comparatively benign. The reason for such an association is evident, the same habits of life and hereditary elements being

Arthritic glycosuria.

present in both diseases. It should be noted, however, that the consumption of alcohol is not often a prominent factor in the causation of glycosuria.

Antecedent or Complicating Diseases.—Aside from those mentioned in the opening paragraph, gout, tuberculosis and nephritis are occasionally associated with diabetes, the two latter being usually secondary conditions.

Lipogenic.

Obesity.—A large number of glycosurics are obese, and this lipogenic form is ordinarily easily controllable and often curable; nevertheless, many cases apparently benign at the outset become true diabetes.

Pancreatic Disease and Sclerosis of the Spinal Cord.—It is probable that a large proportion of the cases of diabetes are pancreatic in origin. Sclerosis of the cord may be secondary or primary, and the relationship of the two conditions has not been sufficiently worked out.

The three P's.

SYMPTOMS.—The so-called "three P's," polyuria, polyphagia, polydipsia, represent the fundamental symptoms as seen in a *typical* case and the thirst, increased frequency of urination, and large amount of urine, are usually the first symptoms noticed. An excessive appetite is seen in nearly all advanced cases. Rapid loss of weight and strength is marked in true diabetes and often prominent in the benign forms. *The Skin.* The skin is dry, eczema, boils, carbuncles are common and gangrene is often a terminal complication. *The Eye.* Neuro-retinitis, hemorrhage and cataract are often seen. *Muscles.* Besides pronounced weakness, muscle cramps are common in advanced cases. *Sexual Organs.* Impotence, amenorrhea, pruritus vulvæ and balanitis occur and pregnant women are likely to abort. *Nervous System.* Neuritis, headache, mental irritability or depression, drowsiness or insomnia may occur, true melancholia may develop, and in 50 per cent. of the severe cases the knee-jerks are absent unilaterally or bilaterally. *Lungs.* Pulmonary tuberculosis is a common terminal event.

Coma.—Diabetic coma may be the first *recognized* symptom of the disease. The author recalls a casual meeting on the street which led to the recognition of unsuspected advanced diabetes in the infant child of a friend, the babe dying a few hours later in diabetic coma. In this case the breath had the peculiar aromatic sweetness present in all cases of threatening coma and presumed to be due to the presence of acetone. Other premonitory symptoms are headache, drowsiness, nausea, vomiting, dyspnea and rapid pulse, the dyspnea is a definite air hunger, the term accurately describing the symptom. Constipation is often marked and symptoms of mild or severe indigestion may be present. In actual coma the breathing may be slow and deep, sighing, superficial, or Cheyne-Stokes. Certain cases are characterized by profound collapse, and in others ataxic symptoms are prominent.

"Fruity" breath.

Precautionary Measures.—The discovery of a definite acetonuria demands immediate action on the part of the physician.

Carbohydrate increase.

One would depend upon the administration of *added amounts of carbohydrates* and if deemed necessary the internal administration of sodium bicarbonate 30–40 gm. daily until neutralization of alkalinity of the urine was attained.

Alkalis.

In the severer cases or those suggesting an impending coma, this treatment would be pushed rapidly (2 gm. every 20–30 minutes).

In beginning coma or the established condition, sodium bicarbonate would be given *intravenously, never subcutaneously* (one liter 4 per cent. solution).

Hitherto, acting upon the established fact that starvation of the normal individual produces an acidosis, we have feared it for diabetic patients, but Allen claims that occasional recoveries from prolonged diabetic coma are due to the clearing of the blood as a result of starvation, that the treatment for acidosis is *fasting*, the use of alkalis being proper but of doubtful efficacy. Starvation.

Intermittent fasting, when scientifically, rationally and intelligently applied is without doubt the best means of controlling glycosuria and preventing acidosis.

Every medical man should be familiar with the technic of this procedure and its important limitations and safeguards.

The sudden radical withdrawal of sugar and starches is an invitation to acidosis, a complication responsible for two-thirds of the deaths from this ailment.

The non-withdrawal, or strict limitation of fats constitutes another important source of danger from the same cause.

The Blood.—Bremer's test may be valuable in coma if for any reason a specimen of urine is unattainable. Two smears, one of diabetic blood, the other of normal blood are made in the ordinary manner. Tehse are fixed by heat and stained for two minutes in a 1 per cent. aqueous solution of congo-red. The diabetic smear remains unstained or shows a pale or greenish-yellow. The control smear of normal blood is stained red. If methylene blue is used the diabetic smear takes a yellowish-green; the normal, blue. On the other hand, a solution of Biebrich-scarlet stains the diabetic smear scarlet and does not affect the normal blood. The reaction is not caused directly by glucose itself, but by some unknown substance present in the blood of diabetics. Bremer's test. Glycemia.

Glycemia.—Normal blood contains sugar in an amount varying between 0.060 and 0.120 per cent., the average content being 0.085 per cent.

If the blood sugar content is increased to about 0.170 grams per 100 cu.cm. the "threshold of retention" is passed and glycosuria becomes manifest.

On the other hand even though the diabetic patient's urine may have been rendered sugar-free, the blood may continue to show an excess above the normal though insufficient in quantity to cross the threshold and appear in the urine. The amount shown in the blood of diabetics varies from 0.130 to 0.400 per cent. and in coma may rise to 1 per cent.

Einstein's modification of Benedict's method for the estimation of the sugar content of the blood is given on page 120 and is the simplest and most rapid process known to the author.

Hyperglycemia in Conditions other than Diabetes.—An excess of sugar in the blood may follow the ingestion of an excess of starches and sugars, precede

and accompany the menstrual period, accompany pregnancy, or attend cerebral hemorrhage, brain tumors, skull fractures, or severe concussion.

Emotional shock has been known for long to be a competent cause, and diseases of the liver, pancreas, or endocrine glands, and carcinoma of the viscera may be associated with a markedly high degree of glycemia.

Ether anesthesia and major surgical operations often bring about a marked temporary increase, and in Graves' disease it maintains a relatively high level.

Classical case.

The Urine.—Total quantity increased, the amount sometimes reaching or exceeding 20,000 c.c. The specific gravity is high, varying from 1030 to 1060. Sugar varies from a mere trace to 8 or 12 per cent. in extreme cases.

Important Variations.—It must be remembered that in glycosuria as in albuminuria the abnormal constituent of the urine may be totally absent for considerable periods, may be lacking at one time of the day and present at another, and may be wholly removed by careful dieting or fasting, thus making it possible for a glycosuric to pass a life insurance examination.

Removable sugar.

Low specific gravities, etc.

Furthermore, the specific gravity is not always high, and a specific gravity of 1010 *does not justify the omission of the test for sugar, as is so generally believed.* No one of the cardinal symptoms is absolute. Polyuria is not an invariable symptom and, as might be inferred, both polyphagia and polydipsia may be absent and even in true diabetes long periods of latency may occur.

Diacetic Acid and Acetone.—(See under Urinalysis.)

Important factors.

Prognosis.—The prognosis in glycosuria depends primarily upon the decision as to whether one is dealing with true diabetes or simple glycosuria. In the former, the prognosis is always grave; in the latter, it is favorable. In youth, the disease is almost invariably rapidly fatal, cures being extremely rare. The thin offer a more serious prognosis than the fat. The presence of diacetic acid is a danger signal. Apoplexy, carbuncles, tuberculosis, gangrene and coma are distinctly terminal events.

Heredity is of great importance. *Age* is a cardinal factor, for children under ten all die and even young adults (under twenty-five) seldom recover. At and above forty the prospect of control and life prolongation is good and many cases representing the preceding decade yield favorable results.

Cases of acute diabetes are described but the author has not encountered an impeccable one as yet in the adult.

DIABETES INSIPIDUS.—This curious ailment, occurring usually during the first three decades of life, and more frequently in the male, may be induced by head injuries, violent emotions, acute infections, tuberculous meningitis, sunstroke, cerebral syphilis or tumor (*especially, of the hypophysis*) and diseases of the abdominal viscera, including aneurysm. Heredity is sometimes evident and it may be congenital.

Involvement of the hypophysis cerebri (pituitary body) is relatively common and, possibly, constant, in these cases of idiopathic (true) diabetes insipidus.

The diuretic action of the internal secretion of the *pars intermedia* of this gland is well established.

The characteristic feature of diabetes insipidus of the idiopathic type is the peculiar response of the kidneys to increased sodium chloride or protein ingestion.

In the normal individual and in cases of primary polydipsia ("symptomatic diabetes insipidus") this response manifests itself chiefly or wholly, in an increased concentration of the urine, i.e., heightened specific gravity, increased total solids.

In diabetes insipidus also the increase of total solids is obtained, but only by the passage of an increased total amount of urine, the specific gravity remaining unchanged.

Unvarying specific gravity.

Morbid Anatomy.—The lesions consist merely of congestion and dilatation of the kidney and ureters, and hypertrophy of the bladder. Various lesions of the nervous system have been reported but none specific save as above.

Symptoms.—Excessive or unusual thirst, striking emaciation and a dry skin coexist with the passage of enormous quantities of pale, faintly acid or neutral urine (5000 to 20,000 c.c.) during the twenty-four hours. The specific gravity is extremely low (1001 to 1005), total solids for the twenty-four hours normal. Albumin and casts if present at all are scant and of no significance and the sediment is negative ordinarily.

Great quantity of light urine.

Differential Diagnosis.—The disease is most commonly confused with interstitial nephritis but the mistake can seldom occur if the total solids are estimated as should invariably be done, an increase being the rare exception in the latter disease. Furthermore, the cardiovascular symptoms of nephritis, in any case likely to be confounded with diabetes insipidus, usually would be marked.

Prognosis.—If associated with cerebral tumor or organic disease in any part of the body it is bad. Many of the hereditary cases recover or live *with* their ailment to a ripe old age.

GOUT

(*Arthrosia podagra*)

Definition.—*An ailment of unknown specific etiology chiefly affecting "well-to-do" individuals, appearing in acute, subacute, chronic, or, peculiar larval forms and characterized by an hereditary or acquired inability on the part of the affected individual to elaborate and maintain sufficiently active specific ferments and nucleases (oxydases) to secure to the individual organism such chemical changes in the purin bases as are essential to normal metabolism.*

Etiology.—The true cause of gout remains still unknown though the work of recent years has greatly enlarged and enriched our knowledge of its metabolic disturbances and of the ferments which are deficient or lacking in victims of this disease. It would appear that in the discussion of its etiology, we are still dealing largely with secondary factors.

Basic cause is unknown.

Inheritance.—This constitutes one of the most interesting and striking features in connection with gout and though a prominent English clinician

The wine cellar.

of wide experience has said that *the inheritance of gout is largely the inheritance of a good wine cellar*, we must, nevertheless, recognize the fact that a special predisposition to perverted and deficient metabolism exists in the descendants of gouty individuals and occasionally becomes apparent at a relatively early age.

Extraordinary figures.

In Great Britain, which includes the area of greatest prevalence of this ailment, from 75 to 80 per cent. of the cases are said to suggest an hereditary taint and even in the public services of the London hospitals the hereditary element is said to exceed 50 per cent.; nevertheless, one may easily overestimate the importance of such figures if he fails to take into consideration its wide prevalence and the habits of life, with respect to a generous dietary and the use of heavy ales and wines, which have prevailed throughout that country.

Mode of life.

Remarkable prevalence.

There can be no doubt that a very considerable proportion of the population of England is tainted with a more or less attentuated gout and the inheritance may take the form either of the main disease or some of its many so-called alternatives such as arteriosclerosis, chronic nephritis, eczema, certain forms of diabetes and chronic gastric intestinal diseases.

Possible source of error.

In a malarial country a marked tendency exists always to charge to that disease an enormous number of ailments which in a non-malarial country would be dignified by a separate clinical title and specific and individualized treatment. No doubt the same statement is true to some extent with respect to gout in its alternative forms and this fact further vitiates our statistical information.

Alternatives.

Any or all of the diseases mentioned as gout alternatives are merely expressions of toxemia occurring also in countries relatively gout-free.

Peculiarities of transmission.

It is said by English authorities that transmission from a grandparent through the mother, herself unaffected, is more evilly effective than transmission from father to son. The fact that women are relatively immune to the disease, even though descendants of gouty fathers and grandfathers, would lead one to attribute much of this immunity to their lesser indulgence in the pleasures of the table and wine cellar, and still further diminish one's belief in the present astonishing figures relating to the frequency of gouty inheritance.

Unusual in youth.

Age (*Juvenile Gout*).—In proven cases or typical forms, this is undoubtedly unusual and the disease is essentially one of middle age, incidence rarely occurring under thirty. It shows a suggestive tendency also to subside after the age of sixty when in most individuals the habits of life are greatly modified.

Lead Poisoning.—The frequency of lead poisoning in association with gout is so great as to raise serious doubts as to the true meaning of the figures offered. Garrod, for example, reports not less than 33 per cent. of gouty patients appearing in his public services as showing evidence of an antecedent saturnism; on the other hand, Frerich of Berlin failed to find one case of gout among the 163 cases of lead poisoning which he specially investigated.

Irreconcilable figures.

Habits.—Habitual heavy eating and the consumption of large amounts

of animal food and highly spiced rich dishes, especially when combined with a persistent indulgence in alcoholics, even though the daily allowance may not be actually excessive, and especially an habitual indulgence in the *heavier wines and ales*, would seem to be commonest factors in the production of cases of proven gout. Basic in predisposition.

Certainly the most important element in the treatment of chronic gout lies in the imposition of restrictions with relation to such habitual indulgence.

One might consider also the effect of excessive worry, mental strain and exhausting labor under adverse conditions, the first being not uncommon contributory factors in the gout of the well-to-do, the last a frequent element in "poor man's gout" occasionally encountered by the author in British clinics. Poor man's gout.

Occupation.—In general, gout is a disease of the well-to-do and of the classes of relative or absolute leisure. Workers in lead, butchers, those employed in breweries, public houses and the like are doubtless predisposed through occupation, yet manifestations of the disease even in them are usually associated with a history of heavy eating and steady, even if moderate, drinking. Predisposing occupations.

Climate and Race.—Some curious differences in racial predisposition exist and to what extent climate may play a part seems to be undetermined. With relation to the incidence of gout among the people, England leads the world, France and Holland come next, and Swedes, Norwegians, Russians, Italians, Spaniards and Germans show comparative immunity. "Normandy is said by Charcot to be especially affected as compared with the rest of France, and Scotland and Ireland suffer far less than England." Strange differences.

The foregoing statement certainly offers food for speculation and an opportunity for some practical statistician.

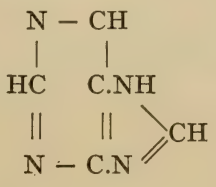
Essential Factors.—The investigations of the last decade have thrown a flood of light upon *the disturbed metabolic processes associated with gout*, and added much to our knowledge of its dietetic management. *Nevertheless, much remains to be explained and its basic cause is wholly undetermined.*

The changes wrought in the purin group through the action of oxygen on the one hand and NH_2 on the other evidently dominate the deficient metabolism characteristic of this disease.

These purin bodies derived from ingested nuclein-rich food, as well as those of the body tissue themselves (nucleins and nucleo-proteids), become of first importance in the modern view of gout. Exogenous and endogenous purins.

Uric acid belongs to the "purin group" which contains also adenin, guanin, hypoxanthin, xanthin, and caffein.

Purin is represented by the formula



By the introduction of oxygen or NH_2, or both, the oxypurins, hypoxanthin, xanthin, and uric acid, or the aminopurins, adenin and guanin, are formed.

The source of the purin bodies is nuclein, and their elaboration depends upon the presence of a sufficient amount of active ferments (nucleases, desamidases, and oxydases) in the human body.

"Under normal conditions the greater the nuclein content of the ingested food the more marked is the excretion of uric acid in the urine, and in the normal individual the uric acid excretion curve is singularly constant for like conditions of diet, activity, and body weight, though varying considerably in different individuals" (Lütje).

In gouty individuals the excretion of both endogenous and exogenous uric acid *is markedly diminished save during an acute seizure.*

Characteristic variation.

Clinical Characteristics.—*The most characteristic clinical features of gout are:*

1. *The persistence of uric acid in the blood even under a purin-free diet (i.e., eggs, cream, milk, cheese, olive oil, and other fats).*

2. *That under such a diet the excretion of endogenous uric acid (that not derived from food rich in nucleins but from the patient's own tissues) is abnormally slight.*

3. *That during an attack of gout the urinary uric acid content rises sharply and promptly falls below normal at its close.*

4. *That victims of gout placed upon a diet rich in purin bodies (thymus, pancreas, liver, etc.) fail to show the marked increase of exogenous uric acid excretion by the urine found in the normal individual.*

5. *The tendency to uratic deposits in the joint tissues in chronic cases and the formation of tophaceous deposits there and elsewhere.*

6. *The tendency of the disease to attack metatarso-phalangeal joints and, in acute seizures, that of the great toe, with peculiar frequency.*

7. *The frequency of larval and irregular gout of peculiarly protean and baffling symptomatology.*

8. *The tendency of the disease to change its form and pass from the stage of acute seizures followed by wholly symptom-free intervals, to that chronic form associated with permanent changes in the joints and other organs (kidneys, stomach, intestines, heart, and blood vessels), with greater or less impairment of health and strength.*

9. *The tendency of the disease to develop its severer manifestations after the third decade of life.*

SYMPTOMS.—The classical symptoms of acute gout are:

(*a*) *Agonizing vise-like pain in a metatarso-phalangeal articulation, usually that of the great toe.*

(*b*) *Dusky redness of the affected area.*

(*c*) *Swelling, with tense, hot, glistening, overlying skin, and ultimately a slight superficial edema.*

(*d*) *Exquisite tenderness.*

(*e*) *Fever, rarely exceeding* 102° *to* 103°*F.*

(*f*) *Moderate leucocytosis*—14,000, 20,000.

(*g*) *An excess of uric acid in both urine and blood (Garrod's thread test) at the height of the attack.*

Onset.—The attacks come on suddenly, usually in the early morning hours, but are commonly preceded by premonitory symptoms sufficient to warn any patient who has previously experienced such seizures.

Such premonitory symptoms are:

(*a*) Loss of appetite or dyspepsia, with acid eructations.

(*b*) Bronchitis.

(*c*) Unusual restlessness, nervous instability, or attacks of mental depression.

(*d*) Fleeting pains in the smaller joints.

(*e*) Headache, insomnia, or unrefreshing sleep.

(*f*) *Moderate leucocytosis*—14,000, 20,000.

The pain usually abates in the morning, only to reappear on succeeding nights with added intensity. One joint only may be affected, or the disease may appear in other articulations. An attack usually lasts for a week or more, gradually subsiding with desquamation and *local itching*, leaving behind it a tender swollen joint, which in most instances persists for at least a week and often much longer. Walking may be difficult for several weeks.

Transient albuminuria may occur even in the absence of established renal disease. As has been stated, the urine is scanty and the output of uric acid, markedly increased during the acute seizure, promptly shrinks as the attack subsides, and is markedly diminished during the free periods.

Cardiovascular Lesions and Gouty Kidney.—As is well known, the gouty habit and inheritance alike predispose to cardiovascular changes.

Arteriosclerosis is the usual accompaniment. It is usually widespread and frequently associated either with a true contracted kidney or with that of the arteriosclerotic type.

Glycosuria.—Gouty diabetes is well known as a complication. It is usually mild, essentially chronic in its course, and readily controlled by proper dietetic restrictions.

That such a condition should occur as a complication of gout is extremely interesting in its relation to the imperfect or perverted ferment production and activity which lie at the root of the symptomatic expressions.

Exciting Causes.—The factors precipitating a gouty seizure are often undiscoverable, but local joint injury, strains, and exposure to cold and wet are often the apparent causes; and fits of anger, psychic shock, or acute worry are undoubtedly sufficient to determine the explosion.

Retrocedent Gout.—This faulty term covers cases in which the symptoms relating to the brain, gastrointestinal tract, or heart are unusually severe.

Delirium, stupor, coma, and apoplectiform attacks, excessive purging, and persistent or intractable vomiting are alarming symptoms which fortunately seldom occur. Precordial pain, rapid, irregular pulse, and severe dyspnea are likewise of an alarming nature and serious import, but these

symptoms of severe toxemia furnish us with no adequate ground for assuming a separate type of the disease.

Many are uremic.

Futcher rightly states that most of these individuals are uremic, and to this statement one may add that the cardiovascular changes of long-established cases are sufficient in themselves to account for nearly all of the symptoms.

Acute gout presents no serious diagnostic problems in differentiation.

Chronic Gout.—*The seats of predilection in chronic gout are, in order of frequency, as follows: the great toe, tarsus, ankle, knee, hand, and carpus.*

As the disease progresses in its chronic form the joints become thickened and deformed. Bursal inflammations are common, especially in the region of the olecranon and patella, and the chronic painful course is frequently interrupted by acute or subacute seizures with or without fever.

The tophi may form masses as large as a plum, but the "Heberden's nodosities" so frequently encountered in clinics, are rare in gout and more often seen in connection with arthritis deformans of the hypertrophic type. These occur commonly about the terminal finger joints, seldom exceed a pea in size, are painless, and show no tendency to ulceration. They occur most commonly in women and during or after the menopause. Undoubtedly most cases of chronic painful multiple arthritis occurring in the male, and in gout-ridden countries, are due to gout, and this is especially true of such cases as are associated with marked arteriosclerosis or renal disease.

It is probable that the disease is on the increase in America, but good wine cellars, heavy ales, excessive meat-eating and lives of relative leisure, are still relatively uncommon in the United States, and, in consequence, gout is relatively rare and in many regions almost unknown.

The chronic arthritis of women is more likely to represent arthritis deformans, or the rarer genuine chronic rheumatism, both of which are probably due to chronic infections.

Irregular Gout.—This comprises that vast group of symptoms arising in the case of tainted individuals who have escaped the acute manifestations of inherited or acquired gout.

Not a concrete ailment.

The term "lithemia" has served in the past to cloak every diagnostic sin in the catalogue of human ills, but at present is falling into disuse, as is also the term "gouty diathesis," which falls so pleasantly upon the ear of those hypochondriacal victims of gastric neuroses and the like, who feel that at last a safe anchorage is found in the snug but ample diagnostic harbor represented by an aristocratic ailment.

A basic error.

"Irregular gout" is a perfectly proper term, and the ailment merits most careful consideration. Its presence should not be predicated upon the mere presence of an apparent excess of uric acid in the urine, as is so frequently done, for, in true gout, the uric acid curve runs below the normal and only by the most elaborate analyses based upon a carefully controlled dietary can one determine the endogenous and exogenous uric acid curve of the individual.

Hence, the absolute diagnosis of irregular larval gout is surrounded by too many technical difficulties to render it available for most practitioners. The tentative or probable diagnosis is only justified when the family history or habits clearly indicate the tendency in the individual.

Correct diagnosis difficult.

The symptoms ascribed to it cover almost every variety of gastro-intestinal disturbance, eczemas, herpes zoster, headaches, including migraine, and too often a mere psychasthenia* or some of the many disorders reflecting the "congenital universal asthenia" of Stiller.

Bizarre and incoherent symptomatology.

The treatment so often prescribed is therefore poorly adapted to many of the ailments so often sheltered by this too ample diagnostic cloak, and quite generally the diagnosis of "lithemia" is applied to cases which are of a nature directly opposite to gout in etiology and in therapeutic requirements, or, to serious organic disease, needing both correct diagnosis and selective treatment.

RICKETS.†—An infantile disease characterized by general impairment of nutrition and peculiar changes in the bones.

Etiology.—The etiology remains unknown and it cannot as yet be classed with deficiency diseases. Improper food, *calcium deficit* in the osseous structures, and insanitary environment, quite frequently associated with a syphilitic or tuberculous hereditary taint. The disease is relatively rare in breast-fed children.

Basic cause unknown.

Symptoms.—The child appears delicate superficially and structurally, with small bones, less rigid than normal and *shows at the epiphyses*, especially of the wrists and ankles, *characteristic swellings* associated with imperfect ossification processes. *In the parieto-occipital region the bone may yield to finger pressure and in certain areas there may be parchment-like crackling (craniotabes). The broad forehead carries prominent frontal bosses due to hyperostosis, the ribs show the peculiar and characteristic beading at the chrondro-costal junction* (the rickety rosary), the clavicles appear short and abnormally curved, and there is frequent *pigeon breast* (*pectus carinatus*). The *abdomen is prominent*, the *liver and spleen are usually enlarged* and autopsy may show changes in the mesenteric glands. Such a child shows either an inability to walk or creep about or if walking may suddenly develop apparent weakness or disinclination. There may be slight fever, the *child is tender when touched or moved* and *extremely restless at night*, the pillow being soaked with perspiration and the hair often rubbed away at the occiput. As might be expected gross rickety deformities are common. *Spinal curvature, usually* but not always antero-posterior, appears and bow-legs of the most extreme type may be encountered.

Epiphyseal swellings.

"Cranio-tabes" and "bosses."

"Rickety rosary" and "pigeon breast."

Persistence of the fontanelles is a common symptom. The diagnosis is not likely to be missed in well-marked cases, but the slighter varieties occurring under unexpected conditions may cause trouble.

Prognosis.—The disease may indirectly cause a large mortality by weakening the child's resistance to other diseases, but there is no direct

* The term is used throughout in its literal sense.

† Included here for the sake of convenience rather than correctness of classification.

mortality. The development may be long delayed and permanent lack of resisting power and various deformities be left behind.

Many cases are encountered which lack the classic symptom group in greater or less degree.

NEURASTHENIA

(*"Nervous Prostration," "Nervous Exhaustion"*)

Definition.—*A term applied loosely to a large and heterogeneous group of symptoms indicative of instability and excessive lability, both mental and physical,*

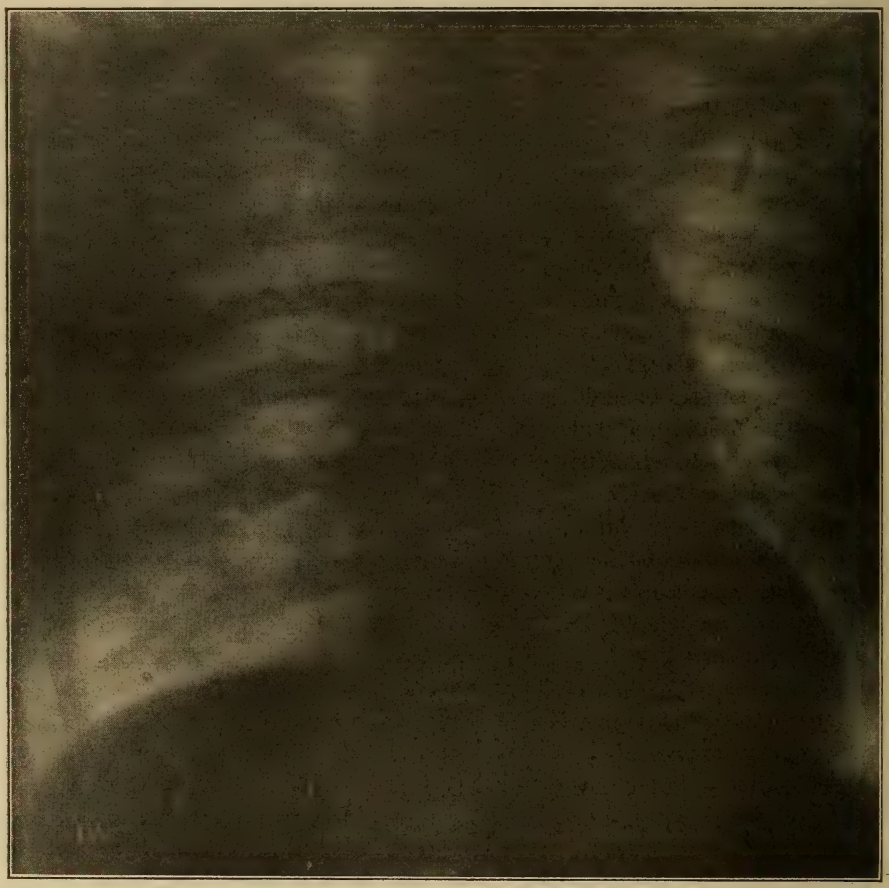


FIG. 584.—A remarkable case of cardiovascular syphilis with aneurysm and an enormous heart presenting typical "neurasthenic" symptoms. *No murmurs were present.*

such as occur also individually or collectively in a great number of organic diseases and known constitutional defects.

Its very existence doubtful.

Whether the syndrome ever exists as a primary and independent condition or as a concrete and definite symptom-complex is, at present, doubtful.

A melange of symptoms.

Preliminary Comment.—To deal satisfactorily with a condition totally lacking demonstrable specific pathology and presenting only a *melange* of

symptoms, individually and collectively such as may represent the commonest and most logical results of a great number of chronic and acute diseases, would seem to be, and undoubtedly is, a hopeless undertaking.

Like those archaic terms "heart trouble," "stomach trouble," and "liver trouble," the term neurasthenia has not only served as a cloak for our own diagnostic insufficiency, *but as a standing invitation to loose methods* A euphemism of "don't know"

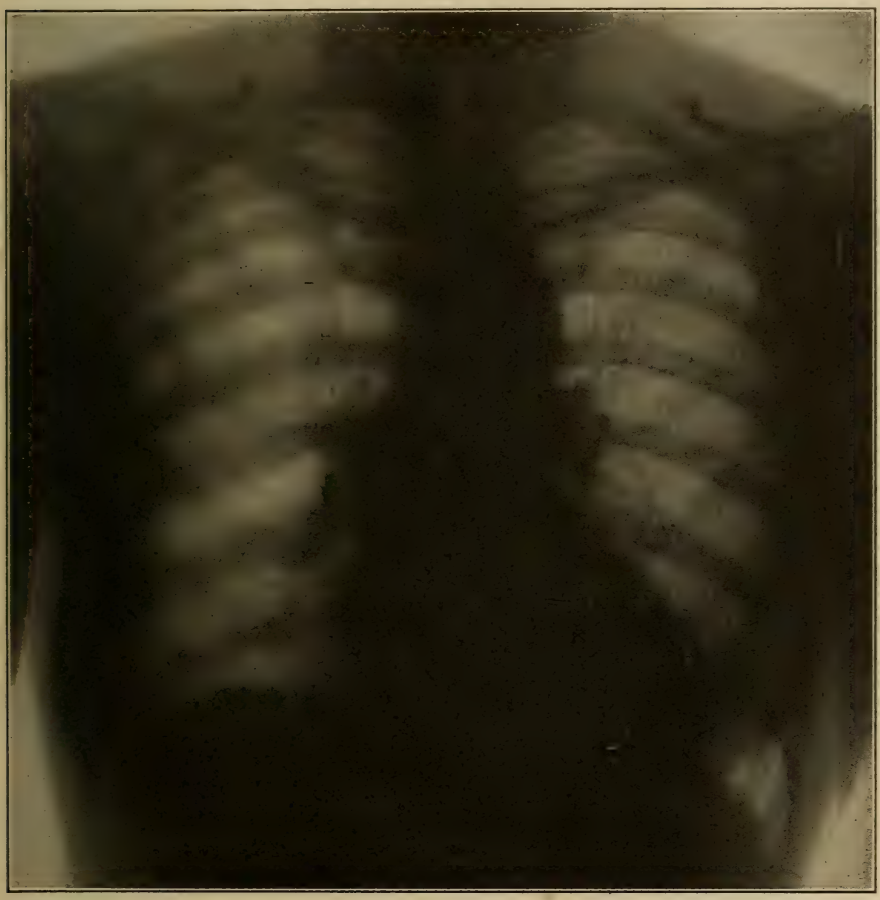


FIG. 585.—A case of cardiorenal disease with marked cardiac enlargement and decided hypertension. *No murmurs were present.* The symptoms of "neurasthenia" had proven most misleading.

in diagnosis, offering a too ready relief from some of the most vexing problems encountered in medicine. If it were only on this ground it would seem that the term should either be dropped wholly, restricted to the few cases which may possibly be entitled to such a designation, or strictly limited as to its use to the description of true psychasthenia, using that term in its literal sense.

Greatly Attenuated by Modern Diagnostic Methods.—*To the author it would appear that if we were to deduct from the cases of so-called "neurasthenia"*

*those proving under thorough and scientific methods of investigation to be examples of organic disease of the heart, kidneys and nervous system, chronic cryptogenic septic infection, chronic anemia, drug-habit, incipient insanity, hyper-, hypo- and dys-pituitarism or dys-thyroidism, larval syphilis and, especially, active obscure tuberculosis and chronic subnutrition, the residue would closely approximate zero.**

FIG. 586.—A case of frank mitral stenosis and regurgitation in relation to which the obtrusive symptoms of "neurasthenia" had been accepted at face value.

* To such of us as have doubted for years the very existence of a primary "*neurasthenia*" and chafed under the universal tendency to retain and employ this unfortunate designation in lieu of the specific terminology applicable to the ailments which underlie it, it is refreshing to read the recent statement of an eminent neurologist to this effect, viz., "The diagnosis of neurasthenia is becoming more rare as the methods of study improve, as our knowledge of the chemistry of disease grows, and as we examine patients with greater and greater thoroughness."

Chas W. Burr: Osler's "Modern Medicine," vol. v. p. 624, Second Edition, 1915. Dr. Burr believes nevertheless in its "*infrequent*" existence as a primary disease.

There are no symptoms or symptom groups peculiar to "neurasthenia" in the light of modern knowledge and its continued existence seems to depend upon the fact that in some instances a genuine scientific diagnosis is unattainable.

Larval organic diseases.

This obviously constitutes an uncertain tenure, especially as the syndrome reflects apparently a distinct toxemia or subnutrition of the centers, conditions which must often arise from cryptogenetic foci of infection, minor circulatory defects, faults of internal secretion, or renal permeability such as may be obscure or quite beyond detection for a time, but frankly manifested later.

A basic factor.

To a remarkable degree its manifestations depend upon *depressed general nutrition* associated with structural deficiencies of the type of "*universal congenital asthenia.*"

This condition, originally described by Berthold Stiller, of Budapest, and elsewhere considered is now becoming recognized at home and abroad as the condition basic in manifold disturbances of function, inadequacies of structure and predisposition and vulnerability to infection.

Keystone of neurasthenic arch.

It explains fully that frequent inadequacy to *sustain* the normal amount of stress, incidental to life and life's work, which, heretofore, has been regarded as the keystone of the "neurasthenic" arch.

A confusing factor.

Much confusion has arisen from a tendency to place certain obstinate or inveterate psychic deviations under "neurasthenia" or "neurasthenic psychoneuroses."

Some of the many thus classified in a recent work on nervous diseases are the following:

(*a*) *Agoraphobia.* The fear of crossing open spaces.

(*b*) *Claustrophobia.* The fear of any enclosed space.

(*c*) *Morbid doubt and indecision of an excessive type.*

(*d*) *Obsessions.* Even kleptomania and morbid impulses to kill.

(*e*) *Morbid lack of will power.* Associated with inability to perform certain simple acts save in complicated ways or the utter lack of power to step over a slight obstruction, etc.

With all of these are included those states of *morbid anxiety* combined with perfect lucidity of thought.*

Such fixed or inveterate *obsessions, phobias and "anxiety neuroses"* would seem better dealt with as states apart even from mere psychasthenia, which well might be used in its literal sense. There is unquestionably such a thing as "brain-fag," though it is astonishingly difficult to produce by mere brain-work or even by shock, grief or other emotional crises in perfectly sound, otherwise healthy and well-nourished individuals.

Hypochondria must be carefully distinguished from the perfectly natural mental depression, discouragement and anxiety so often present at the outset of treatment in many chronic conditions associated with illness of the most varied character.

* Headache, slight defects of memory, irritability, temperamental instability, readily induced fatigue, mental and physical, vertigo, etc., etc.

The great danger to the patient involved in the present unfortunate freedom of employment of the term "neurasthenia," as representing a primary ailment, is best illustrated by enumerating some of the many organic diseases coming under the author's notice, in which the primary diagnosis was "neurasthenia." Only those diseases are mentioned in relation to which diagnostic error has been represented in many instances.

These are: (*a*) Dilated, insufficient hearts both of the "drop" type and those of the primary myocardial or endocarditic-myocardial type (a host of them).

(*b*) Renal impermeability and even nephritis of the interstitial or arteriosclerotic type (a multitude).

(*c*) Carcinoma of the stomach or bowels.

(*d*) Gastric and duodenal ulcer.

(*e*) Early and even advanced cerebral arteriosclerosis. (Nearly all of the precocious or middle-aged cases.)

(*f*) Chronic cholecystitis.

(*g*) Insanity.

(*h*) Hysteria.

(*i*) Thoracic aneurysm.

(*j*) Brain tumor.

(*k*) Cerebral syphilis.

(*l*) Addison's disease.

(*m*) Active but obscure tuberculosis of the lungs (a great number).

(*n*) Tuberculous meningitis.

(*o*) Tuberculosis of the kidney.

(*p*) Paretic dementia.

(*q*) Drug habit (nearly all of the private cases).

(*r*) Atypical hyperthyroidism (great numbers of them).

(*s*) Atypical myxedema (the greater part of the thyroid "insufficiencies)."

(*t*) Cryptogenetic sepsis (probably one of the commonest of overlooked conditions).

(*u*) Chronic malaria.

(*v*) Secondary and even pernicious anemia.

(*w*) Leukemia.

(*x*) Chronic appendicitis.

(*y*) Locomotor ataxia.

(*z*) Lead poisoning and various other toxemias of a similar sort including concealed chronic alcoholism.*

The source of error.

Many more could be named and it is obvious that in most instances the primary diagnostic failure was simply the result of grasping the most obvious diagnostic solution, under the honest belief that asthenic and psychasthenic symptoms represent a common clinical entity and a common ailment, viz. neurasthenia.

The primary assumption should be that symptoms of asthenia and psychasthenia constitute the commonest of the indications of obscure infections and toxe-

* To this list "*endocarditis lenta*" should be added.

mias, of incipient, partially or fully established organic disease and of nutritional deficit usually associated with visceroptoses.

Symptoms.—These are so numerous and varied as to preclude a full enumeration and description but apart from certain instances wherein direct cerebral or spinal damage occurs, as in larval cerebral syphilis or the early stages of general paresis and multiple sclerosis, are alike in their reflection of lability and instability of the circulation, of body nutrition, and of the *psyche* of the patient, no less than in their tendency to persist until an adequate circulation is reëstablished **and the nutritional level raised.**

A general failure to grasp these fundamental truths is responsible for the extreme chronicity of the phases of psychic and functional depression which makes the so-called "neurasthenic" the bane of the general practitioner and the chief pillar and support of the innumerable Spas of Europe and the United States alike.

With respect to the mental state and deceptive localization of symptoms, the so-called "nervous dyspeptics" furnish the typical example. This is the commonest form of ambulatory invalidism or semi-invalidism, a lure for the unwise among surgeons, as is too often indicated by the multiple scars of futile operative procedure, and a constant source of therapeutic failure and consequent discredit to the family physician. The patient may be seen in any stage, but in its more extreme forms it makes a deplorable clinical picture clearly setting forth the minor but troublesome types of psychic instability which result from poisoning of the centers whether this is but a part of general tissue starvation, of deficient cardiac power and reserve, or the toxins of acute or chronic infections and dyscrasias.

Deficient endurance, submerged response, or excessive perverted or ill-controlled reaction must result.

Diverse symptoms.

This finds its expression in readily induced fatigue, psychic, physical, or both combined, in vagaries of gastrointestinal secretion and motility, in the decided dominance of symptoms in one local field or another, often excessive, because of the summation of persistent or recurrent defects of reaction to initial stimuli.

Introspection.

The patient tends to become self-centered and morbidly introspective; his moods are variable, but quite naturally he may become irritable and despondent. Every symptom becomes magnified; the pathways for pain and discomfort are better and better defined as they are the more persistently traveled, while morbid anticipation and expectation beckon them on.

Exaggeration.

The trifling ache or pain, the trivial flatulence, the disinclination or mere hesitation of the bowels to adhere to a strict schedule, all minor events such as occur to a majority of healthy men from time to time, unheeded, come to be of vast importance to the nervous dyspeptic or to any psychasthenic individual.

A man of infinite detail.

At his worst he is the man with the notebook, and in this is jotted down from day to day a multitude of interrogatories and finically noted trivial events for the consideration of the physician.

In his pocket is a wallet full of carefully preserved prescriptions, urinary examination records, differential blood counts, stomach analyses and the like.

Finical about food.

Every particle of food taken is admitted under the honor system, so to speak, for each article has long since been tried and measurably proven.

A haunter of "Spas."

If he is well-to-do he may have visited every "Spa," tried every fad from the dietetic zero, through the grape cure, the exclusive chopped or scraped meat diet, to the "molken Kur" and is more than ready and willing to discuss with the physician or any fellow sufferer each and any therapeutic system, individual diet, mineral water, or drug.

Such is the extreme but very common picture which leads one of the foremost of Swiss clinicians to pronounce the psychasthenic dyspeptics an "incurable group" (*diese unheilbare Krankengruppe*"), an expression which doubtless drew a heartfelt "amen" from the great body of practicing physicians; yet *nearly all of these patients are curable,* sometimes very easily, oftener only by the expenditure of several months of time and an amount of money sufficient to pay for residence in a hospital, a *selected* trained nurse, and a doctor.

Curability.

Diverse Localization of Symptoms.—Practically every organ in the body may be the *apparent focus and source* of the disease and the fountain-head of *selected* symptoms, but too often misinterpreted as surgical conditions.

This is especially true of the female pelvic organs and the abdominal viscera proper, which are chief sources of symptomatic expressions.

Futile surgery.

It is appalling to think of the amount of surgery which has been undertaken in cases lacking any organic lesion of a gravity sufficient to demand it, and of the almost invariable results, i.e., temporary improvement, ultimate increase of disability, and, not rarely, totally unexpected death either "upon the table" or within a few days following the operative procedure.

In these cases, above all others, radical surgical intervention must be based upon genuine need and definite findings and, when *emergency is* lacking, undertaken only when properly directed medical treatment has failed to relieve the condition, or the patient is so circumstanced that he must hazard the more direct method.

An unfortunate tendency.

Unfortunately these structurally deficient, almost invariably *visceroptotic* and functionally unstable, individuals seek operative relief joyously, usually to their own harm and the surgeon's discomfiture.

A detailed presentation of symptoms is quite unnecessary, but it should be remembered that cases of all grades of intensity are encountered; that in many instances the condition is temporary and recurs only when nutrition is brought below a certain level; and that, however decided the psychic symptoms may be, they do not often exhibit the characteristics of a true psychosis.

They usually are natural and logical results of the conditions present and of the peculiar handicaps of the individual.

In nearly all of the active cases the hallmark of *congenital universal asthenia* will be found, to which a more or less decided impairment of nutrition or loss of weight is usually added.

When these are absent a source of reflex irritation, often found to lie in the sexual apparatus, cryptogenetic foci of infection, incipient disease of the brain or spinal cord, drug habit or other source of symptoms must be sought.

Misinterpreted "fatigue."

The most striking psychic symptoms are those expressing readily induced or persisting "brain fag" or excessive irritability, and in many instances, the sense of physical fatigue is of psychic origin though this is oftener an expression of anatomic and temporarily inadequate myocardium (and the poor nutrition of the patient).

"Brain fag."

Failure of concentration or of the capacity for sustained mental effort, and defective memory, are extremely common. Restlessness, moodiness and irritability are natural results of the conditions present.

Sources of discomfort.

Vasomotor relaxation, troublesome throbbing of the abdominal aorta, a visible capillary pulse, and *vagus* arrhythmias, no less than those due to associated or causative minor cardiac decompensation or overstrain are occasionally present and may be so intensified subjectively by the patient's abnormal reactivity, as to become extremely troublesome.

This tendency to over-reaction is also accountable for the increased sensibility to pain and the hyperesthesia, so often evident.

Sexual causes.

In some instances sexual excesses and perversions lie at the root of the ailment or need correction before any measures of treatment can prove successful. Misleading testicular and ovarian tenderness occur and an exaggerated symptom-response on the part of a ptotic uterus, though wholly removed in many instances by treatment directed to the general improvement of nutrition (the ptosis persisting unchanged), often lead to operation and to repeated examinations and persistent local applications which tend to focus the patient's introspection upon the reproductive organs.

Misleading tenderness.

Sexual hyperexcitability, on the one hand, and nervous or fatigue impotence upon the other, are not rare.

Vertigo is a relatively uncommon symptom and a tendency to syncope may sometimes be observed.

The deep reflexes are normal or, oftener, exaggerated, the pupils may or may not be dilated but react normally to light and accommodation, and readily induced ocular fatigue is quite common.

Peculiar areas of anesthesia, hysterogenetic zones, and a lack of bilateral symmetry on the part of the superficial reflexes, indicate, not neurasthenia but hysteria, a thing apart, wrapped in its own veil of mystery.

Be thorough.

Diagnosis.—Inasmuch as a genuine primary "neurasthenia" must at most be barely "within the limits of visibility," it would be superfluous to do more than assert the absolute necessity for a thorough investigation in every case, directed to the detection of the actual underlying cause of any psychic and motor disturbances present.

Procedure recommended.

Primarily the question of recent or existing infection should be determined and that of recent weight loss or persistent poor nutrition decided. Organic disease must be excluded or, if manifest, properly evaluated, and the stigmata of congenital universal asthenia sought. In this connection especially,

but in every case, the condition of the heart itself should be determined, for each and every symptom of "neurasthenia" *may* be wholly or in large measure due to minor cardiac incompensation.

Sources of reflex disturbance must be considered, together with the habits and mode of life of the individual.

Drug habit, luetic infection, and larval or active defects of internal secretion are potent in causation and hysteria and incipient insanity must be given thought in otherwise clouded histories.

A common source of error lies in the presence of unsuspected larval hyperthyroidism or its opposite larval myxedema. The former may be strongly suspected if the blood shows a relative lymphocytosis and indeed in all cases of persistent and excessive pulse acceleration coexisting with even slight thyroid enlargement and a pseudo-Corrigan pulse. The latter condition may demand test doses of the thyroid substance.

Paretic dementia is very generally called "neurasthenia" in its earlier stages and the same statement applies to other forms of cerebral syphilis and disseminated sclerosis.

The cryptogenetic septic foci represented by chronically infected tonsils or accessory sinuses, peridental abscesses and the like may be of cardinal importance as causative or perpetuating factors.

Basic assumption

Finally the author would repeat and emphasize the statement that only by the primary assumption that so-called "neurasthenia" even in its clearest classical expressions represents almost wholly a mere symptom complex of an underlying, constitutional and demonstrable structural or nutritional defect, with or without any one of many organic diseases or toxemic states, can we hope to avoid serious error.

SEA-SICKNESS AND CAR-SICKNESS.—These interesting and common conditions still remain unexplained. We know that the vestibular apparatus is to some degree responsible and that to some extent the abdominal sympathetic system participates.

With many people, however, the condition may be absent at one time and present at some other, presenting perhaps far less of the pitching and "writhing" motion of which such sufferers chiefly complain. Many are wholly free in recumbency or even in the open air, with others a failure to take a brisk cathartic before a voyage proves disastrous.

Fortunately, of itself, it seldom or never kills, but in no other condition known to the author is there more complete temporary change of disposition, greater hopelessness, more profound depression, or even obscuration of niceties of life.

Even old sailors are not immune. One of the sickest of sea-sick individuals ever seen by the author was a Gloucester fisherman who for punishment was kept aloft in a heavy head sea off Cape Horn and forced to continuously slush down the masts.

DISEASES OF THE NERVOUS SYSTEM

Scope of section.

Scope of Section.—*The extreme complexity of this department of medicine renders its adequate discussion impossible save in works wholly devoted to the specialty it represents.*

Nothing beyond a helpful résumé or synopsis is attempted in this volume. The realm of the alienist is not invaded, nor is any serious attempt made to set forth in detail the complicated anatomy of the nervous system, a thorough knowledge of which, based upon special study and dissections, must be assumed as already possessed by the reader.

There is absolutely no "royal road" or "short cut" to the acquisition of knowledge with respect to the anatomy and physiology of the nervous system.

The Neuron Theory.—Though incomplete, the prevailing theory concerning the histologic structure and functional activity of the nervous system has the merits of simplicity and ready adaptation to known symptomatology.

As now conceived, the nervous system is an aggregation of like units, each consisting of an excitable ganglion cell and a cellulifugally conducting axis cylinder process of variable length (axon, neurit), which running naked, or becoming ensheathed as a medullated nerve fiber, gives off lateral branches (collaterals) which, like its terminal, split ultimately into fine fibrillæ known as *end brushes or arborizations*. The cell also has cellulipetally conducting protoplasmic processes known as *dendrites* or *dendrons*.

Ganglion cells.

Axis cylinder.

End brushes.

Dendrites.

With relation to the cell these represent respectively the transmission and reception of outgoing and incoming impulses.

Although the terminal axon arborizations of one neuron are intimately related to the dendrites of another, it is probable that there is no actual connection, but rather mere contact or contiguity. The function of the basic plasma is unknown, but in the cell body are compact coils of fibrillæ which are regarded as the true conducting elements and certain basophilic granule groups known as Nissl's tigroid substance, which are so modified or diminished under conditions of cell fatigue or disease as to suggest a trophic function.

Fibrillæ.

Chromophilic granules.

Mode of Action.—If one assume an afferent (sensory) impulse originating in any of the peripheral special sense organs (sensory dendrites) and considers the nature of the conducting mechanism, he can readily appreciate the facility of transmission from one set of neurons to another until a reflex or voluntary center is reached and the equal readiness with which its efferent (motor) fiber carries the message. This marvelous telegraphy is the basis of all acts, reflex, voluntary, or automatic, and involves a maze of complicated pathways and substations which relate to the orderly association and interaction of sensation, movement, and intellection necessary to human life and normal activity.

Conduction and transmission.

Regional distribution and function are both conserved by the *grouping* of cells and fibers to form sensory and motor tracts, reflex pathways, trophic centres, and primary governing centers.

Regional grouping.

Motor impulses.

Sensory impulses.

Parenthetic neurons.

Centrifugal vs. Centripetal impulses.

CONDUCTION IN MOTOR AND SENSORY AREAS.—Motor impulses originating in the pyramidal cells of the cortex pass through their axons to a terminal arborization in the anterior horn of the spinal cord or to a cranial nerve nucleus where they are received by the dendrites of the related neurons and through their axons conveyed to the periphery. The sensory impulse requires in addition to these central and peripheral neurons certain "parenthetic" neurons. For example, a pain impulse from the skin passes through the dendrites of the peripheral neuron *to* the spinal ganglion and through its axon to the cells of the cord or medulla oblongata, and from these cells, acting as parenthetic neurons, to the cortical center, hence in general, "centrifugal" corresponds to motor and "centripetal" to sensory impulses.

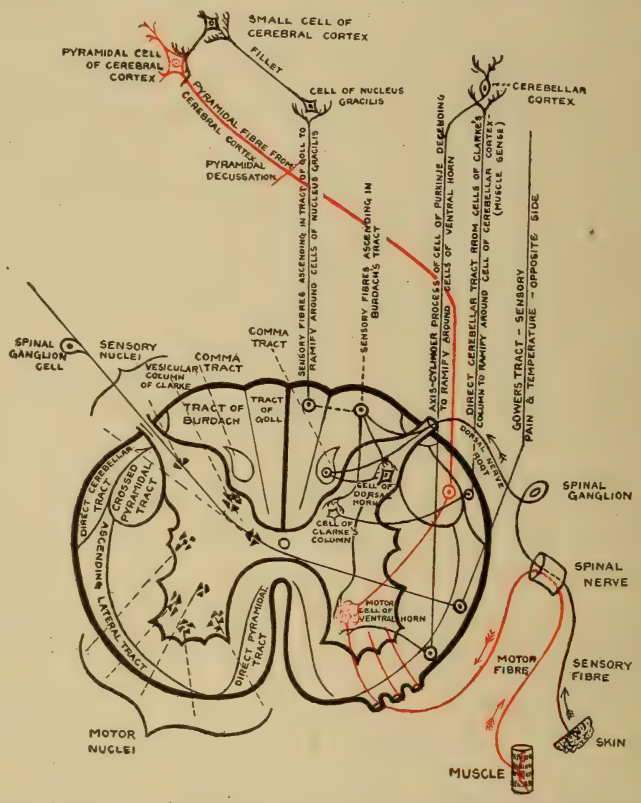

SMALL CELL OF CEREBRAL CORTEX
PYRAMIDAL CELL OF CEREBRAL CORTEX
FILLET
CELL OF NUCLEUS GRACILIS
CEREBELLAR CORTEX
PYRAMIDAL FIBRE FROM CEREBRAL CORTEX
PYRAMIDAL DECUSSATION
SENSORY FIBRES ASCENDING IN TRACT OF LISSAUER TO RAMIFY AROUND CELLS OF NUCLEUS GRACILIS
SENSORY FIBRES ASCENDING IN BURDACH'S TRACT
AXIS-CYLINDER PROCESS OF CELL OF PURKINJE DESCENDING TO RAMIFY AROUND CELLS OF VENTRAL HORN
DIRECT CEREBELLAR TRACT FROM CELLS OF CLARKE'S COLUMN TO RAMIFY AROUND CELL OF CEREBELLAR CORTEX (MUSCLE SENSE)
GOWER'S TRACT – SENSORY PAIN & TEMPERATURE – OPPOSITE SIDE
SPINAL GANGLION CELL
SENSORY NUCLEI
COMMA TRACT
VESICULAR COLUMN OF CLARKE
TRACT OF BURDACH
TRACT OF GOLL
CELL OF DORSAL HORN
CELL OF CLARKE'S COLUMN
DIRECT CEREBELLAR TRACT
CROSSED PYRAMIDAL TRACT
ASCENDING LATERAL TRACT
DIRECT PYRAMIDAL TRACT
MOTOR CELL OF VENTRAL HORN
SPINAL GANGLION
SPINAL NERVE
MOTOR FIBRE
SENSORY FIBRE
SKIN
MUSCLE
MOTOR NUCLEI

FIG. 587.—Showing course of sensory and motor fibers in the cord and the formation of a spinal nerve. (*After Keiller, Gerrish and Dana.* Slightly modified.)

Cortex.

Corona radiata.

Internal capsule, pons and medulla

THE MOTOR TRACTS.—The Direct Motor Tract *arises in the cortical cells constituting the motor areas* (see Fig. 513), *and its neuraxons pass through the corona radiata and are gathered into a narrow band occupying most of the posterior segment of the internal capsule; thence they pass through the crura cerebri to the pons variolii and medulla, giving off in the latter collaterals which, with the exception of the sixth nerve, cross the median line to the nuclei of the motor cranial nerves of the opposite side.*

The main bundle in passing through the medulla sends nine-tenths of its fibers across the median line at its lower portion to form the *motor decussation and the anterior pyramids*. Thus the original bundle is divided into *uncrossed* and *crossed* columns. Decussation.

The *crossed fibers enter the lateral column* of the spinal cord to form the *crossed pyramidal tract* which descending, diminishes in size as it yields fibers at different levels (spinal segments) to form the spinal motor nerves, in each case *ending in a terminal arborization about an anterior horn cell* which transmits the impulse through its own neuraxons (spinal nerves). Crossed tract.

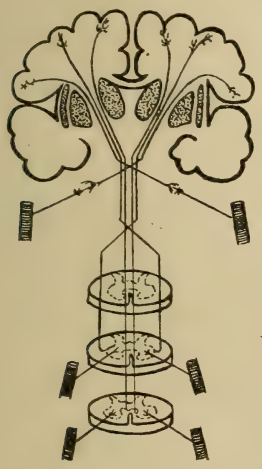


FIG. 588.—The direct (voluntary) motor tract. (*After Van Gehuchten.*)

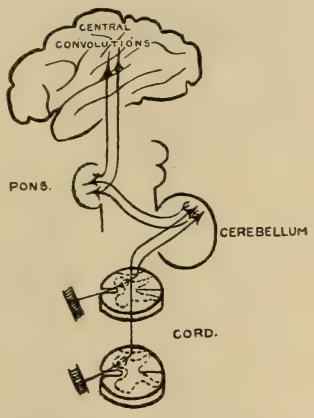


FIG. 589.—The indirect (involuntary) motor tract. (*After Van Gehuchten.*)

The uncrossed tract forms the "columns of Türck" (*"direct pyramida* tract") *and its fibers cross* in the *anterior commissure at the different levels*, to participate in the formation of the spinal motor nerves through an arborization around the anterior horn cells, exactly as in the case of the crossed pyramidal tract fibers. Direct tract. Motor nerves.

The "indirect motor tracts" follow primarily much the same course but after giving off terminal arborizations to the pons-nuclei cross the median line in the middle cerebellar peduncle, thence they pass to the cerebellar cortex, and thence, after arborization, to the lateral fundamental column and anterior horns. They are related to coördination and higher reflex and automatic movements.

DIRECT SENSORY TRACT.—From the *skin* the impulse passes along the *peripheral nerve* and to its cell in the posterior spinal ganglion, thence to cells in the posterior horns which receive the stimulus and for the most part transmit it by their neuraxons across the cord through the anterior commissure to the opposite antero-lateral ascending tract, and through the medulla and pons to the optic thalamus where another cell receives and by its neuraxon transmits it to the cortex.

THE INDIRECT SENSORY TRACT.—This conveys stimuli from muscles, joints and the viscera through the posterior root ganglia and thence along the sensory roots to the cord where a part enters directly, and ascends in the posterior column of the cord of the same side as far as the nuclei of the columns of Burdach and Goll (*nucleus cuneatus and nucleus gracilis*) in the medulla, from which centers the neuraxons (internal arcuate fibers) cross in the sensory decussation. Some go thence to the cerebellar cortex, whence they are transmitted by cells of that area through the superior cerebellar peduncles to the optic thalamus and red nuclei, and thence by yet

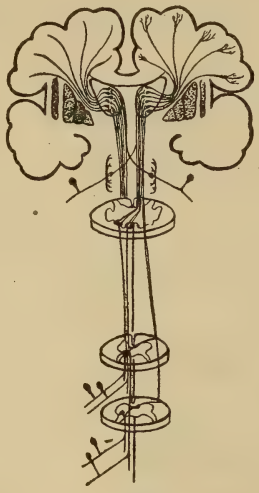


FIG. 590.—Direct sensory tract. Course of fibers and general arrangement of neurons. (*After Van Gehuchten.*)

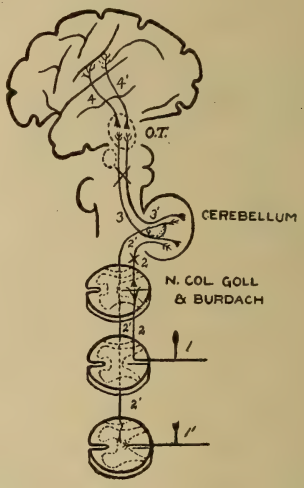


FIG. 591.—Indirect sensory tract. (*After Van Gehuchten.*)

another neuraxon to the central convolutions. Other impulses pass from the sensory root to the cells of Clark's column, and pass up the direct cerebellar tract to the cerebellum, thence to the optic thalamus, red nuclei, and cerebral cortex.

FUNCTIONS OF THE TRACTS OF THE SPINAL CORD.—*The functions of the various tracts of the cord are, so far as known, the following:*—**The direct pyramidal tract** is purely motor and its fibers cross at the level of the nerve emergence.

The crossed pyramidal tract is the chief motor area of the cord representing the fibers which cross in the lower portion of the medulla. The peripheral nerves of both tracts are dependent upon the trophic influence of the anterior horn cells.

Equilibrium.

The direct cerebellar tract represents chiefly visceral sensation and is concerned with equilibrium.

Gower's tract.

Goll's tract.

The antero-lateral ascending tract (*Gower's tract*) is concerned with sensations of pain, temperature and touch, conveyed from the opposite side through the anterior commissure. *Goll's tract* conducts sensation from the

joints, tendons and muscles *of the same side* while in the cord, *its fibers crossing in the medulla.* It is therefore associated with ataxia, deficient orientation and muscle sense in general, under conditions of disease.

Burdach's tract conveys tactile sensations from the opposite side, contains many associating fibers and is crossed by afferent sensory fibers conveying reflex, painful, articular or muscular stimuli, hence if diseased there may be anesthesia, ataxia, more or less pain and interrupted (lost) reflexes.

The fundamental columns are related to association of different cord levels with each other and with the brain stem and cortex and contain both sensory and motor fibers. The cells of the anterior horns are trophic in function, constitute the nutrient cells of the lower motor neurons, and if diseased produce atrophy, R.D.,* and paralysis, (flaccid paralysis) and loss

Anterior horn cells.

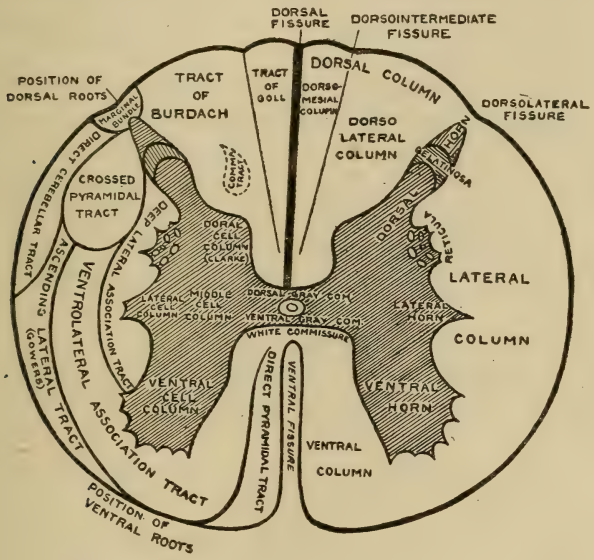


FIG. 592.—Diagrammatic transverse section of spinal cord. (*After Gerrish.*)

of the deep reflexes. The anterior horns are essentially motor, the posterior sensory. The cells at the posterior angle of the posterior commissure are probably automatic centers; others in the immediate neighborhood may be trophic, sensory, and vasomotor.

Anterior vs. posterior horns.

Automatic centers.

Functions of the Sensory Tract.—The direct sensory tracts represent chiefly *pain, touch* and *temperature sense, the indirect* sensory and motor tracts being intimately connected with coördinate movement, voluntary or involuntary and, of course with visceral sensation. These wonderful tracts govern the automatic and psycho-reflex acts of the human organism.

Pain, touch, temperature sense.

Visceral sensation and coördination.

CAUSES OF DISEASE OF THE CEREBRO-SPINAL SYSTEM.— These *may be both direct* or *indirect, primary* or *secondary, and though the greater number of nervous diseases proper are distinctly related to structural alterations of the nervous system, the clinician must always hold in mind the*

Remote ailments as causes.

*The electrical reaction of degeneration.

fact that remote disease may find its ultimate and perhaps chief and terminal expression in the brain, cord, or peripheral nerve.

For example, *arteriosclerosis* and nephritis are responsible for most cases of cerebral hemorrhage and thrombosis, as is *left-sided valvulitis* for cerebral embolus. *A peripheral neuritis* may be traced to *lead* or *alcohol* poisoning, and in such cases, reasoning from effect to specific cause, one reaches a definite and potent therapeusis. We may consider, therefore, without separating primary from secondary lesions, the following etiologic factors:

(*a*) **Toxemias and Direct Bacterial Activity.**—*Arsenic, lead, alcohol, and carbon bisulphid serve as the type of one form; diphtheria, erysipelas,* **syphilis,*** *and typhoid of another; nephritis, diabetes, and chronic auto-intoxication, severe anemias, and the cachexias, yet another.* In most instances such poisons produce changes in the peripheral nerves or the spinal cord, *i.e.*, polyneuritis, acute poliomyelitis, Landry's paralysis, locomotor ataxia, etc., the peripheral nerves being oftenest affected.

(*b*) **Secondary (metastatic) tuberculous, malignant, or septic foci.**

(*c*) **Affections of the heart and blood vessels,** *i.e.*, valvular lesions, arteriosclerosis, degenerative diseases of the heart muscles, etc.

(*d*) **Tumors.**—Under this we include gummata, sarcoma, carcinomata, tuberculous growths, cysticercus cysts, etc.

(*e*) **Traumatism, and its sequelæ,** including scar formation.

(*f*) **Meningeal inflammation,** acute or chronic, suppurative or non-suppurative.

(*g*) **Caries, gumma, osteomyelitis.**

(*h*) **Developmental defects and diseases of a distinctly hereditary type.**

(*i*) **Certain unexplained nervous disorders denominated functional,** such as epilepsy.

(*j*) **Certain changes of a purely senile type,** often premature in occurrence, and in general, degenerative and atrophic in type.

(*k*) **Hereditary and acquired predisposition,** *habits, and environment,* hold a prominent place.

DEGENERATION.—*Injury to the parent cell causes degeneration of its processes, and division of these leads to their death peripherally, but may not entirely destroy the function of the cell.*

It is interesting to note that *secondary* degeneration is limited to the neuron involved and that a lesser amount of atrophy may occur in a fiber not directly or exclusively connected with the affected cell.

There are three chief forms of degeneration: (1) Primary. (2) Secondary or Wallerian. (3) Toxic.

The first occurs in the cachexias, locomotor ataxia, and in senile processes.

The second is characterized by a degeneration chiefly affecting the peripheral segment and is a relatively rapid process, being complete within from two to four weeks.

* Vascular syphilis has assumed enormous importance in diseases of the nervous system as an etiologic factor, since the discovery of *Treponema pallidum* and the introduction of the Wassermann tests. (See Cardiovascular Syphilis" for discussion.)

As a rule, the degeneration follows the direction of normal impulse conduction, though in *association or commissural* fibers it is only partial and, as the *trophic center of the sensory nerve is the spinal ganglion*, if the injury or section be *between the ganglion and the cord* the peripheral nerves do not suffer. The *cells of the anterior horns are trophic for motor nerves* and injury to them as to the peripheral nerve itself means outwardly progressing degeneration.

The third type, toxic degeneration, is segmental but follows the laws of Wallerian degeneration.

Regeneration.—Nerve cells cannot be reproduced if destroyed, but nerve fibers can regenerate though only as to the peripheral fibers, and this process, moreover, demands that the trophic center be sound.

CLASSIFICATION.—Whatever be the cause of organic disease of the nervous system, we distinguish those of the general or irregular distribution from those which chiefly or perhaps exclusively affect a definite tract or neuronic chain ("system disease").

Focal lesions.

In "focal" disease a certain portion of the brain or spinal cord may be involved together with all contained structures. If two or more definite conduction paths are involved, one speaks of a **"combined system" disease.**

The distinction cannot be exact, but is convenient and conforms to known disease types, as in pernicious anemia. In grosser lesions, such as those of hemorrhage, thrombosis, and abscess, one finds a tendency to more or less rapid necrosis of tissue resulting in cyst formation, which may show gradual occlusion by contraction and scar formation.

SEQUENCE OF DEGENERATIVE CHANGES.—These may be thus summarized:*

Spinal Cord Lesions.—Ascending degeneration of sensory pathways, descending degeneration of motor tracts.

Region of the Mesencephalon.—Degeneration of the optic tracts and red nucleus, atrophy of the brachia, corpus dentatum of the cerebellum, pontine ganglia, etc., and both ascending and descending degeneration of the fillet.

Cerebellar Lesions.—Atrophy of the opposite red nucleus and the brachia; degeneration of the median cerebellar peduncle, restiform body, lower olive, etc.

Cortical Lesions.—Atrophy of such related structures as the red nucleus, fillet, opposite brachium, and the subthalamic region, with degeneration of the optic thalamus, all directly related projection fibers and partial degeneration of those of association. In young children Wallerian degeneration results in arrested growth of the parts involved as is well illustrated by those adults who have had infantile spinal palsy.

GENERAL RELATION OF PATHOLOGIC CHANGES TO SYMPTOMATOLOGY.—The following summary will suffice to show the direct and indirect results of (*a*) **irritation,** or (*b*) **destruction** of the motor and

* Following Jakob's classification.

A sharp contrast.

sensory cells and pathways. In the former instance we find morbid activity, in the latter loss of function.

SENSORY AREAS.—Irritation.—(*a*) *Increased response to peripheral stimuli, i.e.*, hyperesthesia or actual pain (hyperalgesia, violent neuralgic pains).

(*b*) *Subjective sensations*, such as tinnitus aurium, hallucinations, both visual and aural, and various paresthesias, such as numbness, tingling, or formication, according to the regions involved.

Absent response.

Destruction.—Entire or partial loss of sensory response to peripheral stimuli or of certain special sensations only (*i.e.*, anesthesia, thermo-anesthesia, analgesia, etc.), and partial or complete ataxia.

MOTOR LESIONS.—(*A*) **Peripheral or lower motor neuron** (*i.e., anterior horn cells, or cranial nuclei, to periphery*).—*Paralysis is flaccid, usually bilateral** (*if unilateral on same side as lesion*), *and must be due to a lesion involving* (*a*) *the cells of the anterior cornua,* (*b*) *the cranial nuclei, or* (*c*) *the peripheral nerves, cranial or spinal as the case may be.*

Flaccid paralysis.

Site of lesion.

Fibrillary twitching may be present but there is neither *contracture* nor *rigidity*, and by interrupting both the reflex arc and the trophic stimulus it *involves a loss of the reflexes* together with the supervention of *marked muscular atrophy* and the *electric reaction of degeneration.* Not only does the *presence* of reaction of degeneration conclusively *prove such a paralysis to be one of the peripheral neuron,* but its *entire absence* after several days means early recovery. *In general, the prognosis is good in inverse proportion to the degree of R. D.*

Lost reflexes atrophy and R.D.

Prognosis.

Spastic type.

(*B*) **Central Motor Neuron** (*i.e., cortical centers to cranial nuclei or anterior horn cell but not including them*).—*On the side opposite an intracranial lesion there is spastic paralysis* (*rigidity*) *with secondary contractures, whereas marked true atrophy* (*aside from that of disease*) *is lacking, R. D. absent* and *tendon reflexes increased by removal of cerebral inhibition.*

Neither R.D. nor atrophy.

Reflexes increased.

Irritative lesions affecting motor areas of the cortex may cause *tonic, clonic* or *epileptiform spasms, athetoid, choreic or wholly irregular movements and muscular twitching.*

Unilateral Spinal Cord Lesions ("*Brown-Séquard paralysis*").—**Loss of power** is complete on the side of the lesion with anesthesia on the opposite side *save for a zone corresponding to the level of the lesion and on the same side.* **Muscle sense** is *impaired on the paralyzed side,* retained *on the opposite* (*anesthetic*) *side,* and the type of paralysis is *spastic* save at the level of the lesion where the destruction of the anterior horn cells may produce a *flaccid paralysis in the limited area represented by their trophic influence.*

Complete Transverse Spinal Cord Lesions.—Symptoms.—If the transverse lesion be complete there is complete *flaccid paralysis* and anesthesia below the lesion with *lost deep reflexes, atrophy,* and *R. D.* Any spastic paralysis indicates an incomplete lesion.

Flaccidity and anesthesia.

* Cord lesions are usually *bilateral;* cortical lesions *crossed monoplegic;* lesions of the region below the cortex and above the motor decussation in medulla, *crossed hemiplegic.*

REFLEXES

*A reflex consists in the transmission of an impulse along a sensory neuron to the reflex center, and thence along the motor axon to the muscle.**† It requires therefore, an unbroken chain from the periphery to the center and back to the periphery, and excessive activity is prevented by a cortical governing (inhibition center). A reflex may be *localized and coördinate*, in that the primary stimulus produces movement in a single muscle or in a limited group

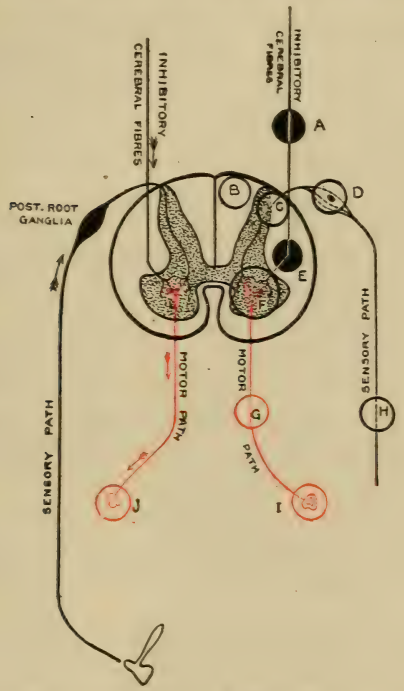


FIG. 593.—Diagrammatic representation of the reflex arc. Normal on left, interrupted on right. It will be noted that the arc proper extends from the percussion hammer (sensory stimulus) through the peripheral sensory nerve, through the ganglion of the posterior root, through the posterior to the anterior horn where the sensory impulse is translated into a motor response which in turn is modified by the inhibitory cortical fibers. It is evident that a lesion interrupting the arc at either C, D, G, H, or F, will abolish the reflex, and produce flaccid paralysis if the nerves themselves or the cells of the anterior horn are seriously involved. Spastic paralysis results, if the lesion be at E or A with consequent exaggeration of reflexes through loss of cerebral control (lateral sclerosis, cortical lesions). Lesions at C and D cause loss of knee jerk and, associated with a lesion at B, represent the usual seat of locomotor *ataxia*. (After Gowers, Butler, Herter, et al.)

Coördinate vs incoördinate.

of muscles, or *incoördinate or spasmodic*, and either general or confined to certain muscle groups. Such are seen in general convulsions from various causes or a reflex spasm associated with certain diseases, and yet another form of reflex may be both coördinate and purposeful. Reflex centers exist

* It is primarily a centripetal sensory impulse conveyed to the reflex center and there converted into a centrifugal or motor stimulus.

† In general the presence of normal *superficial* reflexes is much more important than their absence.

Automatic activity.

in the spinal cord, pons varolii and medulla, the former being largely related to muscular action, the latter to the vital but unconscious and automatic vital functions, *i.e.*, cardiac, respiratory, etc.

THE MORE IMPORTANT SPECIAL REFLEXES.—Ciliospinal Reflex.—Stimulation of the cervical sympathetic by pinching the skin of the neck dilates the pupil and its absence indicates a lesion of that nerve.

Conjunctival Reflex.—Complete anesthesia, deep stupor, or coma usually abolish the well-known spasm of the orbicularis palpebrarum following conjunctival irritation.

Palate and Pharynx Reflex.—The latter is absent in most hysterias.

Jaw-jerk.—(*A contraction of the muscles of mastication*).—Percuss the chin firmly by the mediate method while the mouth is moderately open. Inconstant in health, marked if reflex excitability is increased.

Supinator-jerk.—Tap supinator tendon just above styloid insertion. Usually absent in health (*fifth cervical segment*).

Triceps-jerk.—Support the arm at the elbow, carrying it outward at a right angle to the body, let the forearm hang vertically downward and tap strongly the triceps tendon just above the olecranon (*fifth, sixth, and seventh cervical segments*). It is absent or slight in health.

Biceps-jerk.—Flex elbow at right angles, strike forearm near lower end of radius. Biceps contraction occurs only in disease (*fifth cervical segment*).

Achilles-jerk.—Put the tendon Achilles slightly on the stretch by extending the leg and dorsiflexing the foot, or, have the patient kneel on a chair with the foot unsupported; tap the tendon sharply and in health a contraction of the calf muscles follows. Its early disappearance in locomotor ataxia is an important diagnostic sign (*fifth lumbar and first sacral segment*).

May precede loss of knee-jerk.

It may show a clonus just as in the case of the patellar reflex to be described.

Scapular Reflex.—Contraction of scapular muscles on stroking interscapular region (*fifth, sixth, seventh and eighth cervical and first dorsal segments*).

The Patellar Reflex* (Knee-jerk).—This is best obtained by having the patient cross one knee over the other or better sometimes, if he passes his own arm under the knee of the member to be tested over the opposite knee, in such manner as permits the leg to hang free and relaxed across the wrist.

Reënforcement.

Then the patient is told to close the eyes, join his own hands and pull when the word is given (reënforcement), coincidently the tendon, just below the patellar margin, is sharply tapped with the side of the hand, the edge of a book or a percussion hammer.†

Increased knee-jerks.

The forward jerk of the leg and foot or visible contraction of the muscle varies greatly in health and may be markedly increased in certain conditions of impaired nutrition associated with nervous instability, hysteria, tetanus,

* *Tendon Reflexes.* (Deep reflexes.) It will be noted that all of the tendon reflexes require that the tendon involved be *slightly* on the stretch.

† Usually the jerk can be obtained without reënforcement, but it saves time to carry out the entire procedure. In bed-ridden patients the same procedure can be carried out by utilizing one's own arm and the flexed opposite knee as points of support.

strychnin poisoning, rheumatoid arthritis or after sexual excesses and drinking bouts, and is pathologically increased in any disease which cuts off the cerebral inhibition fibers. Such are cortical hemiplegias and sclerosis (lateral or amyotrophic) of the lateral columns.

For clinical purposes one may assume that it is invariably present in health. Its absence "Westphal's Sign," indicates a lesion interrupting the reflex arc, viz., a lesion involving the sensory or motor fibers (neuritis), the posterior roots or columns, the anterior cells or even the motorial end plates.

Lost knee-jerk.

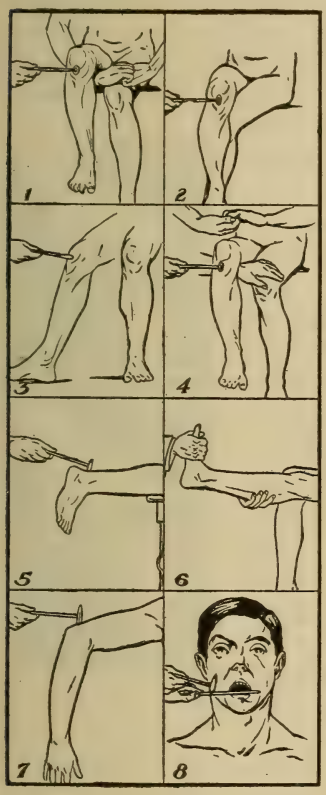


FIG. 594.

1. Knee-jerk elicitation. Method sometimes employed by author.
2. Simple method.
3. A common method.
4. Ordinary reënforcement method.
5. Method of obtaining Achilles-jerk.
6. Method of eliciting ankle clonus.
7. Method of obtaining triceps-jerk.
8. Method of obtaining jaw-jerk.

Hence it is lost in locomotor ataxia, anterior poliomyelitis, transverse myelitis of the second and third lumbar segments (seat of reflex), Landry's paralysis, Friedreich's ataxia and sometimes in chorea, diabetes and severe toxemias such as diphtheria. Cases of pathologic exaggeration may yield a series of "jerk," on tapping the quadriceps tendon. In such cases especially "ankle clonus" should be sought.*

Patellar clonus is elicited by grasping the knee-cap firmly with the thumb and forefinger and pushing suddenly downward and hold momentarily.

Crossed Adductor Reflex.—This is an adduction of the opposite thigh following the tap upon the patellar tendon.

Ankle Clonus.—*The knee is very slightly flexed, the heel rests in the palm of the examiner's left hand, his right grasps, extends and suddenly dorsiflexes the foot upon the leg. An initial series of clonic involuntary contractions of the muscles of the calf repeated under sustained pressure of the flexing hand constitutes clonus.*

True clonus has the same significance as exaggerated knee-jerks in its relation to *organic* disease, being most common in lateral and disseminated sclerosis. If contractions appear before the degree of foot flexion exceeds a right angle and are evidently voluntary, irregular and fleeting, one is dealing with spurious clonus, usually hysterical (*second and third lumbar segments*). Tonic contraction of the anterior tibial group may occur.

True clonus prolonged.

Pseudoclonus, transient, irregular and voluntary.

* The *real test* symptom is contraction of the quadriceps muscle rather than actual movement of the foot. Hence the examiner's free hand should be placed upon the muscle.

Wrist-jerk.—Let the hand drop at the wrist (which is supported), strike the extensor tendons of the dorsum just proximal to the wrist-joint. (Usually absent or slight in health.)

Epigastric.—Retraction of epigastrium following downward stroking of chest in nipple line. A continuation of this line of stroking from the costal margin downward produces contraction of the abdominal muscles. It is of little importance or clinical value (*fourth, fifth, sixth and seventh dorsal segments*).

Abdominal Reflex.—Elicited by stroking outer side of abdomen at various levels, (*eighth, ninth, tenth, eleventh and twelfth dorsal and first lumbar segments*).

Gluteal Reflex.—This consists in contraction of the glutei on vertical stroking of the buttocks (*fourth and fifth lumbar segments*).

Cremasteric Reflex.—Quick retraction of testicle upon stroking inner and upper part of thigh. (Not dartos contraction.)

Caution.—*All reflexes are best elicited when the patient's attention and vision are diverted. The superficial reflexes merely prove the integrity of the region governing them.*

Babinski's Toe Reflex.—This consists in *deliberate* dorso-extension and separation of the toes and especially of the great toe, followed by dorsiflexion of the ankle-joint, when the sole is gently or firmly stroked with a quill point, finger-nail, etc. It is usually associated with disease involving the *pyramidal tracts* and in any event is pathologic. It is said that in healthy young children not yet able to walk a similar but *more rapid* response is obtainable.

FIG. 595.—Plantar reflex. Upper plate shows normal reflex plantar flexion. Lower:—Dorsiflexion of great toe. (Babinski's sign.)

Oppenheim's Reflex.—This is a Babinski reaction elicited by forcibly stroking the inner or median border of the tibia. Gordon applies deep pressure to the calf muscles (Gordon's paradoxical flexor reflex).

Plantar Reflex.—*The chief value of this sign lies in its constancy in health, its frequent absence in hysteria, and its reversed form* constituting the Babinski sign. If the sole of the foot, thoroughly dried, be stroked, the patient's attention being diverted, his position supine, the knee and thigh semiflexed with the leg in outward rotation, plantar flexion of the toes beginning with the four outer, and dorsiflexion of the ankle result (*second and third sacral segments*).

Adductor-jerk.—Abduct thigh and tap adductor magnus tendon. Adductor contraction may occur on both sides in conditions of *high reflex irritability*.

Organic Reflexes.—These involve chiefly respiration, deglutition, micturition, and defecation, and a *full* consideration of their complicated mechanism is out of the question in a volume on medical diagnosis.

They are best considered in relation to the symptomatology of the individual diseases.

Defecation.—To test the action of the rectal sphincter a digital examination is necessary, the strength of the resistant or grasping contraction being noted. In health contraction follows a prick of the anal region.

Rectal incontinence may be met with in all conditions associated with coma or profound toxemia, such as typhoid, as well as in certain organic nervous diseases. There is sometimes a true reflex spasmodic incontinence.

Micturition.—Dribbling of urine usually means overfilling of the bladder and calls for the use of the catheter. A true reflex incontinence may exist such as seen in young children. Both rectal and vesical centers are in the lower lumbar (fourth and fifth) and upper sacral (first, second, third) portion of the cord.

SYNKINESIAS OF DIAGNOSTIC VALUE (*Uninhibitable Associated Movements*).—These represent a limited return from the dissociation of training to fetal pathways of association, through the effects of disease and may be illustrated by several phenomena of clinical interest. The term "reflex" is not fully explanatory of them.

Brudzinski's Signs. *Collateral Reflex.*—In meningitis and cerebral disease, passive flexion of one leg may cause involuntary flexion of its opposite ("identical collateral reflex"). If extension occurs instead, it constitutes the reciprocal collateral reflex.

Neck Sign.—The patient being in the dorsal recumbent position the head is flexed on the chest; flexion of the thighs and of the knees resulting. This is a valuable sign in meningitis.

Strümpell's Tibial Phenomenon.—In lesions of the upper motor neuron the simultaneous making of pressure upon the thigh, light pressure upon the dorsum of the foot of that side, and an effort on the part of the dorsally recumbent patient to raise the thigh forcibly may result in strong contraction of the tibialis anticus muscle.

The Oppenheim-Babinski Hip-flexion sign is of interest but of slight clinical importance. It consists merely in an uninhibitable flexion of the thigh of a spastic paralyzed lower extremity upon attempting the exchange, the *flat* dorsal-recumbent for the sitting posture.

Grasset's sign or phenomenon is of some value in hysterical simulation of *partial* organic hemiplegia or simulation by the malingerer.

In full recumbency with arms crossed in front and legs separated (as should be done in the Oppenheim-Babinski maneuver), the patient finds it impossible to lift both lower extremities *simultaneously* though each may be raised *separately*. The trap for the simulator is obvious.

Hoover's Sign.—This has essentially the same differential value as the preceding maneuver.

In the partial dorsally recumbent "hemiplegic" and the normal person alike, the attempt to raise one leg alone involves downward pressure by the opposite extremity in a degree *proportional to its strength.*

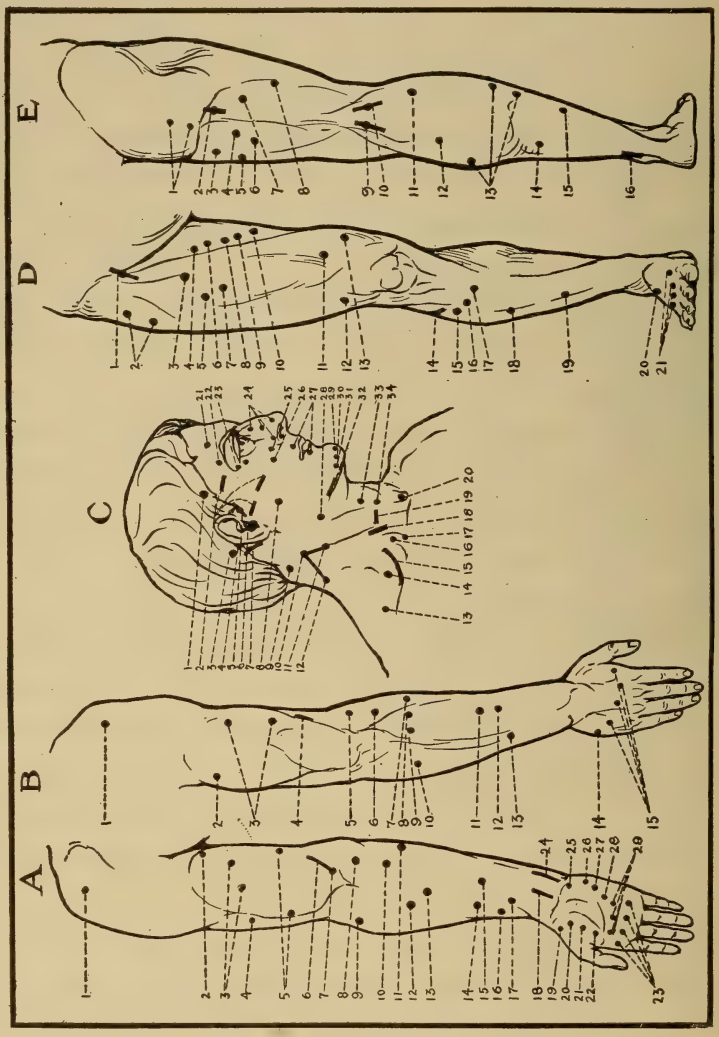


FIG. 596.—The motor points of Erb.

A. Anterior surface of arm, shoulder and hand.

1. Deltoid.
2. Musculo-cutaneous n.
3. Biceps.
4. Musculo-spiral n.
5. Brachialis anticus.
6 & 7. Median (the ulnar lies just internal to 6).
8. Pronator radii teres.
9. Supinator longus.
10. Palmaris longus.
11. Flexor carpi ulnaris.
12. Flexor carpi radialis.
13. Flexor profundus digitorum.
14. Flexor sublimis digitorum.
15. Same (second and third digits).
16. Flexor longus pollicis.
17. Pronator quadratus.
18. Median n.
19. Abductor pollicis.
20. Opponens pollicis.
21. Flexor brevis pollicis.
22. Adductor pollicis.
23. Lumbricales.
24. Ulnar n.
25. Palmaris brevis.
26. Adductor minimi digiti.
27. Flexor brevis minimi digiti.
28. Opponens minimi digiti.
29. Palmar interossei.

B. Posterior surface of arm, shoulder and hand.

1. Deltoid.
2. Triceps (long head).
3. Triceps (external head).
4. Musculo-spiral nerve.
5. Supinator longus.
6. Extensor carpi radialis longior.
7. Extensor carpi radialis brevior.
8. Extensor communis digitorum.
9. Supinator brevis.
10. Extensor carpi ulnaris.
11. Extensor indicis.
12. Abductor longus pollicis and extensor brevis pollicis.
13. Extensor longus pollicis.
14. Abductor minimi digiti.
15. Dorsal interossei.

C. Motor points on the head and neck.

1. Temporalis.
2. Middle branch. (Facial) two lower (branch of upper above).
3. Occipitalis.
4. Retrahens aurem.
5. Upper facial.
6. Facial trunk.
7. Posterior auricular nerve.
8. Masseter.
9. Splenius.
10. Spinal accessory nerve.
11. Sternocleidomastoid.
12. Trapezius.
13. Long thoracic nerve (serratus magnus).
14. Circumflex nerve (deltoid).
15. Brachial plexus.
16. Erb's point (deltoid, biceps, brachialis anticus, supinator longus.)
17. Anterior thoracic nerve (pectoralis major).
18. Phrenic nerve.
19. Omohyoid.
20. Sternothyroid.
21. Frontalis.
22. Corrugator supercilii.
23. Orbicularis palpebrarum.
24. Nasal muscles.
25. Levator labii superioris.
26. Zygomaticus major.
27. Orbicularis oris.
28. Hypoglossal nerve.
29. Levator labii inferioris.
30. Depressor labii inferioris.
31. Depressor anguli oris.
32. Lower branch of facial.
33. Platysma.
34. Sternohyoid.

D. Motor points on the anterior aspect of the leg.

1. Anterior crural nerve.
2. Tensor fasciæ latæ.
3. Sartorius.
4. Obturator nerve.
5. Quadriceps (common point).
6. Pectineus.
7. Rectus femoris.
8. Adductor longus.
9. Adductor magnus.
10. Gracilis.
11. Crureus.
12. Vastus externus.
13. Vastus internus.
14. External popliteal nerve.
15. Peroneus longus.
16. Extensor longus digitorum.
17. Tibialis anticus.
18. Peroneus brevis.
19. Extensor hallucis longus.
20. Extensor brevis digitorum.
21. Dorsal interossei.

E. Motor points on the posterior aspect of the leg.

1. Gluteus maximus.
2. Sciatic nerve.
3. Adductor magnus.
4. Semitendinosus.
5. Gracilis.
6. Semimembranosus.
7. Biceps (long head).
8. Biceps (short head).
9. Internal popliteal nerve.
10. External popliteal nerve.
11. Gastrocnemius (outer head).
12. Gastrocnemius (inner head).
13. Soleus.
14. Flexor longus digitorum.
15. Flexor longus hallucis.
16. Posterior tibial nerve.

In malingering and hysterical paralysis the strength of the downward pressure of the pseudo-paralyzed member is approximately or actually normal.

THE EXAMINATION OF THE MUSCLES.—The tests for paralysis are suggested by the defects noted under "Segmental Lesions," or under the discussion of the special nerves involved.

Lack of tone.

A peculiar lack of tone is noticeable in a completely paralyzed limb even though the patient be unconscious and any existing muscular atrophy is readily detected by observation, careful measurement, and palpation. Loss of power. *With this should be considered the question of power and electrical reactions, as in certain diseases a spurious hypertrophy appears.*

The Reaction of Degeneration.—Faradic and galvanic batteries are required together with the usual appliances for controlling and applying the current.

Faradic test.

The Faradic Current.—*The muscles should first be tested for faradic excitability.* Having wet both electrodes with salt water, a weak current is applied, and with the indifferent electrode over the sternum or between the scapulæ, the smaller electrode is applied successively to the motor points of Erb and the resulting tonic muscular contraction and the increase of current necessary to produce it is noticed. Corresponding points on opposite sides of the body should be tested in alternation.

Galvanic test.

The Galvanic Current.—The indifferent electrode (the larger) should be positive, the normal electrode negative. These are applied as in the faradic test and the current is first closed and after a moment again opened, gradually increasing the current until muscular contraction follows the closure of the circuit, this last constituting *cathodal closure contraction* (CaCC). The switch is then thrown to change the direction of the current, the intensity of which must remain the same and the test repeated, when any muscular contraction will represent anodal closure contraction (ACC). The comparative promptness and strength of contraction must be carefully noted. Further tests are not necessary, but the opening contractions may be noted in the same manner.

Cathodal closure-contraction.

Anodal closure-contraction.

Promptness of reaction.

Significance of Electrical Reactions.—*Normally the response should be prompt and decided to either electrode of the faradic current and should continue during its passage.* If the contraction be sluggish or absent, if it diminishes or disappears, it indicates disease.

CaCC > AnCC is the normal.

In the case of the galvanic current, the reaction following cathodal closure should occur with a less current than is required for anodal closure contraction, but no tonic contraction should occur while the current is passing. Similarly AnCC exceeds, normally, AnOC and this in turn CaOC.

R.D.

The reaction of degeneration is represented by an AnCC which equals or exceeds CaCC. Furthermore, in the case of the galvanic as in the faradic, contraction may be delayed, sluggish, vermiform, or entirely absent.

Sluggish response.

Significance of R.D.

R. D. signifies a loss of nutrition in the motor nerve due either to disease of a trophic center or the nerve itself and is a prominent symptom of the flaccid palsies whether affecting cranial or spinal nerves. When R. D. is present the faradic excitability is lost.

CERTAIN PSYCHIC DERANGEMENTS

Disorders of Memory.—The patient's memory should be noted in the preliminaries of case taking by questions relating to his birthday; the number, names and ages of children, date and place, etc.

Loss of Memory (*Amnesia*).—This may be circumscribed—when only certain things and for a certain space of time cannot be remembered—or *general*. Any disease which impairs the nutrition of the brain, or especially that of the cortex, may result in loss of memory.

It may fail in relation to recent events and remain clear for those of the remote past and both phases must be tested.

It may follow acute infectious diseases, injuries producing mental shock and may be due to organic lesions of the brain, such as apoplexy, tumor, thrombosis, embolism, meningitis, senile degeneration or scleroses, insanity and frequently neurasthenia.

Complete loss of memory is chiefly observed in insanities (dementias) chronic alcoholism and neurasthenia.

Hypermnesia.—An exaltation of memory occurs in some cases of chronic delusional insanity.

Paramnesia.—This is a peculiar illusion of memory in which events which never happened in the experience of the individual are in his belief remembered, occurs in some cases of paranoia and is closely allied to the (normal) conviction that one is repeating at some certain time a former experience.

Defective moral sense, when occurring in an individual whose previous moral status has been above reproach, should excite suspicion of degenerative brain disease, especially dementia paralytica.

Illusions are faulty interpretations of actual visual impressions and are actually visual paresthesias. For example, a strap lying on the floor may be seen as a snake. Illusions are common to nearly all forms of delirium.

Hallucinations are sensory impressions lacking any objective basis. A patient may see a snake on the floor when in reality there is nothing which could be mistaken for a snake. Hallucinations are met with in most all forms of insanity.

Delusions are false beliefs and may be either *sane or insane*—an individual believing that today is Monday when in reality it is Tuesday may be said to have a delusion, or false belief, but it is significant only in so far that he is mistaken. *Insane delusions*, on the other hand, are not only absolutely contrary to facts, but they cannot be corrected by an appeal to reason and are entirely out of harmony with the patient's education and surroundings.

Insane delusions are either *fixed*, *i.e.*, when the insane belief is persisted in, or *changeable* when one belief is constantly being replaced by another, as occurs in delirium.

A delusion is said to be *systematized* when it enters into combinations with other facts of consciousness, as occurs in true *paranoias*, or *unsystematized*, when it is not associated with other facts of conscious experience (delirium, dementia paralytica).

Loss of consciousness occurs as a frequent symptom of organic disease of the brain, but may be a symptom of many conditions depending on nutritional disturbances of the cortex. The degree of unconsciousness varies considerably. The term "*stupor*" is used when the patient can be roused temporarily from his sleep, whereas, if all ordinary stimuli, such as slapping, rubbing, etc., fail to rouse the patient and unconsciousness persists for some time it is termed "coma." (See under "Coma").

Sudden loss of consciousness is usual in syncope, apoplexy, cerebral hemorrhage, embolism or thrombosis of a cerebral artery or may result from concussion of the brain. In epilepsy a temporary loss of consciousness occurs. Uremia, diabetes and cholemia by the production of toxic substances may produce a loss of consciousness, while among the more common and important poisons doing so are alcohol, opium and chloral.

Delirium.—In itself this is divisible into active (wild or maniacal), passive (low, muttering). Furthermore, a delirium may be wholly nocturnal and in all acute diseases tends to assume that form or merge gradually into it during convalescence. Delirium tremens and the fully developed cases of typhoid fever illustrate, respectively, the active and passive type. Delirium, aside from brain lesions, indicates chiefly toxemia or extreme exhaustion, but may be seen in hysteria and occasionally in epilepsy.

Disturbances of Sleep.—Insomnia may occur in acute and chronic diseases, whether cerebral or general, or in neurasthenia or simple overwork. Early waking or frequent waking is quite general in old persons and certain cardiopaths, night terrors and transient somnambulism are common in young children. Tea, coffee, tobacco, cerebral excitement, old age, cardiovascular weakness, and fatigue are common causes of sleeplessness *at night.*

Drowsiness during the day or when attempting to do brain-work is a very common symptom of cardiovascular inadequacy in middle aged and elderly people especially.

"*Psyche* analysis."

Because of the close relation of dreams to delusions and to psychoneurotic symptoms, their painstaking and laborious analysis is believed by Freud to be very important for the interpretation of these symptoms.

The Chief Disturbances of Speech.—*Normal speech depends upon the normal interaction of the senses of sight, touch, smell, taste, sound and muscle sense, together with intact association fibers, a normal memory and an intact motor mechanism.*

Complex associations.

If any link in this chain be lacking a defect in speech results, often taking the form which corresponds to the particular function or subfunction which is deficient. Thus *anarthria or dysarthria* may result from a defective motor tract or from the lack of power to perform the coördinate movement necessary to word formation. This is characterized by imperfect articulation of the linguals l and t if the tongue be involved, or of the labials b, p and n, which are replaced by f and v if the lips are affected. Palatal paralysis causes explosive p's and b's and makes the voice itself nasal.

Anarthria.

Changes in Rhythm.—*Syllabic speech* (division of words into their syllables), *scanning and staccato speech* (explosive word utterance) is another form

frequently observed in multiple sclerosis and occasionally in other conditions. Opposed to this is the *confluent speech* usually bulbar in origin.

In general one may say that lesions involving the tongue and lips give rise to anarthria or dysarthria, whereas speech defects of an entirely different type in which the perceptive centers and higher and more complex degrees of coördination are involved are referable to the cerebrum. The lower mechanism is of course dependent upon and subservient to the higher. Causative lesions.

Disturbances of Speech of Cerebral Origin.—Such are included under the general term *aphasia* which is divided into *motor and sensory types*, the former representing the higher emissive mechanism, the latter the power of receiving and reviving speech concepts, being largely dependent upon sight and hearing (auditory and visual centers). Motor vs. Sensory.

The motor speech center (Broca's center) in the inferior third left frontal convolution, and the articulation center in the inferior anterior central convolution, govern motor speech and either may be affected independently of the other. Broca's center.

A lesion of the left motor speech center causes entire loss of voluntary speech, *i. e.*, true motor aphasia, in right-handed persons, but does not affect automatic and emotional speech, the center for which exists in both hemispheres. It is evident that motor aphasia logically includes the loss of power to utter given words (aphemia), to write words (agraphia), to use gestures (amimia) and the loss of power of musical expression, vocal or written (motor amusia and musical agraphia).* Automatic centers. Amimia and agraphia.

In sensory aphasia the inability to *perceive* and *interpret* language is fundamental and therefore there must be an *auditory aphasia or word deafness* (loss of power to recognize spoken words), *visual aphasia* or *alexia* (failure to recognize or understand written or printed words, *word blindness*), and subdivisions of exactly the same sort as were referred to under "Motor Aphasia." In *paraphasia*, word repetition, misuse and incoherence are the chief features, and *paragraphia* represents the graphic form of the same subdivision. Word deafness and word blindness.

Mind blindness and mind deafness represent an entire loss of visual or auditory memory not only of words, but of common objects and the faces even of near relatives, or in the second variety, all power to recognize musical or other sounds disappears. Here again we find subdivisions, such as *apraxia*, or inability to recognize the use of objects, and *alexia*, representing a failure of comprehension in reading.† *Amnesic aphasia* expresses the difficulty in recalling *voluntarily* special words or classes of words. Apraxia and alexia. Amnesic aphasia.

The Lesions Affecting Speech.—*Motor aphasia*—third *left* frontal convolution in the *right-handed* or a subcortical lesion underlying Broca's center. *Visual speech*—left angular and supramarginal convolutions. *Auditory* Localization.

* "Ataxic aphasia" covers error in form without articulation defects or any evidence of sensory aphasia beyond slight or marked errors in the pronunciation of words, and is a form of aphasia usually seen in attacks that are partial, or, during the process of recovery. The term is sometimes applied to motor aphasia in general.

† Another rare subdivision known as *dyslexia* covers a peculiar mental fatigue accompanying reading, even for short periods unassociated with visual defects or pain.

aphasia—first temporal convolution, auditory speech center or afferent tracts.

Lesions of the association fibers are frequently combined with others, as in *apraxia*. *Anarthria* may result from lesions in any portion of the tract interrupting stimuli from the higher centres to the motor cranial nerves or affecting the essential nuclei in the medulla and pons.

Method of Examination.—Tests for sight and hearing should precede the examination for the detection of the following speech defects: (*a*) **Motor aphasia** (*aphemia*).—The patient cannot speak voluntarily, read aloud, or repeat words. If he cannot write voluntarily or by dictation or copy, he has *agraphia*. If in speaking or writing he uses wrong words or is strikingly incoherent there is *paraphasia or paragraphia*.

(*b*) **Sensory Aphasia.**—The patient cannot recognize words written or printed and read them *silently* even, or, he cannot read them intelligently or correctly. This is determined by writing direct simple questions to be answered by the patient. If he cannot hear spoken words or understand them as indicated by his failure to perform some ordinary action at command, he has *auditory aphasia*.

Mind Blindness and Mind Deafness.—For testing the former one should use common objects, such as a pencil, pen, pocket-knife or coin, and ask the patient to use them, *i.e.*, to write, to open the knife, to open a book, etc. Mind deafness should be tested by the slamming of a door, ringing a bell, coughing, etc., etc.*

INVESTIGATION OF SENSORY FUNCTIONS.—*The eyes of the patient should be bandaged and one should test:* (1) *General sensation.* (2) *Pain.* (3) *Temperature sense.* (4) *Muscle sense.*

Touch.—The tip of a pen, quill, toothpick, or pencil point and a lightly rolled bit of cotton, a feather or a camel's-hair brush are necessary for determining variations in sensibility, the latter articles serving to eliminate the element of pressure. The patient must be instructed to immediately say "touch" when any stimulus is perceived and he must be frequently checked by omitting the touch though putting the question. Exactly similar areas on both sides should be systematically followed to avoid bungling and time-consuming repetition. He should also be made to indicate with his finger the exact points tested, or distinguish between normal, diminished (*hypesthesia*), absent (*anesthesia*), and excessive (*hyperesthesia*) response. Such changes may be unilateral (*hemianesthesia*), bilateral or even general or limited to a single member. As regards perception of stimuli they may be prompt or delayed, and finally we have to deal with various forms of perverted sensory perception (*paresthesia*), among which are many of special sense and the so-called "*aura*" of epilepsy. *A failure to correctly localize sensations* is common in disease and subjective *transference* to the opposite side is called "*allochiria*."

Varieties of sensory defects.

Delayed sensation.

Paresthesias.

* The discussion of this topic is reduced to its most elementary terms. The reader should consult some extended treatise if he desires to enter into the infinite complexities involved.

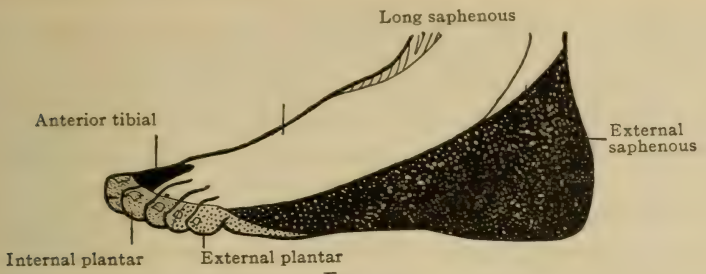


FIG. 597.

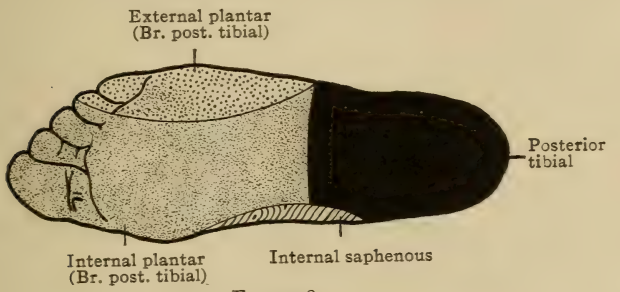


FIG. 598.

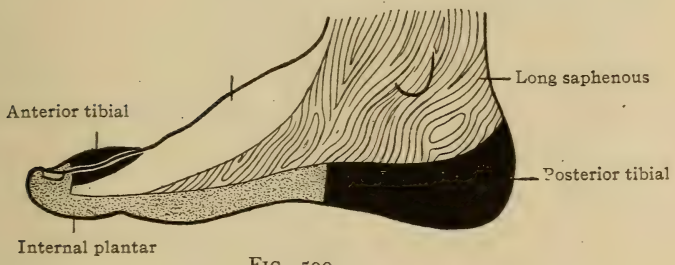


FIG. 599.

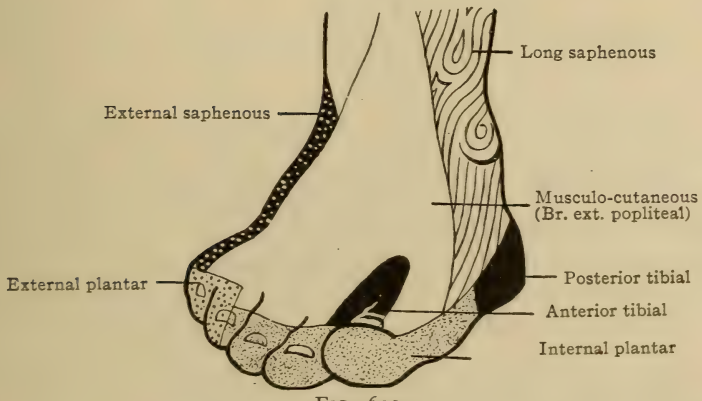


FIG. 600.

FIGS. 597, 598, 599, 600.—Distribution of sensory nerves of the skin of the foot. (G. R. Butler.)

Referred sensation.

Pressure-sense.

Caution.

Tactile Sense.—This may be tested by the esthesiometer or lacking that an ordinary hairpin or pair of blunted or guarded compasses. In this both *pressure and contact* are included and the two points should be separated according to the part examined and in conformity to the summary of normal perception given below,* inasmuch as one point may be felt when two are applied or *vice versa*. The distances necessary for the sensation of two points in actual contact may be abnormally increased. In testing contact or touch the pressure may be determined by placing articles of different weight upon different portions of the body, always seeing that they are supported. *Localization* is readily tested by having the patient place his own

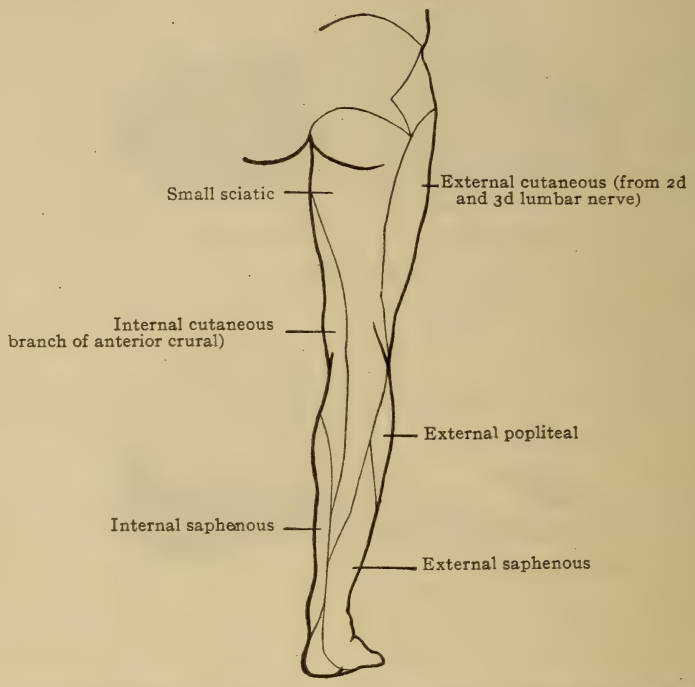


FIG. 601.—Distribution of the sensory nerves of the skin of the leg, posterior aspect. (*G. R. Butler.*)

finger upon any spot touched and the pain sense is determined by using a pen or pin. *Temperature sense* is tested most conveniently with test-tubes containing warm and cold water. Any of the varieties of sensation above described may be increased and diminished, absent or misinterpreted.

Muscle Sense.—For proper testing, articles of various weight but of similar form should be used, the portion of the body tested being *unsupported*. *The tendinous or articular sense* is tested by having the patient imitate

* Lips, 3 mm. (⅛ inch). Tip of toes, cheeks, eyelids, temple, 12 mm. (½ inch). Tip of tongue, 1 mm. (1/25 inch). Tip of fingers, 2 mm. (1/12 inch). Tip of nose, 8 mm. (⅓ inch). Back of hands, 30 mm. (1⅕ inches). Forearm, leg, dorsum of foot, 40 mm. (1⅗ inches). Back, 60 to 80 mm. (2⅖ to 3⅕ inches). Arm and thigh, 80 mm. (3⅕ inches).

any movements of extension or flexion or unusual position in which the other leg is placed and further have him assume positions as described.

Ataxia is chiefly dependent upon the articular and muscle sense but also involves sight, touch and other factors necessary to the harmonious action of muscle groups. It may affect either station, gait or other voluntary movements.

The Test.—The patient should be asked to touch the lobe of the ear of the opposite side, the tip of the nose, a certain finger of the opposite hand, or, in the lower extremities, different points of the opposite members with the great toe. He should then be required to stand with heels and toes together; Static ataxia.

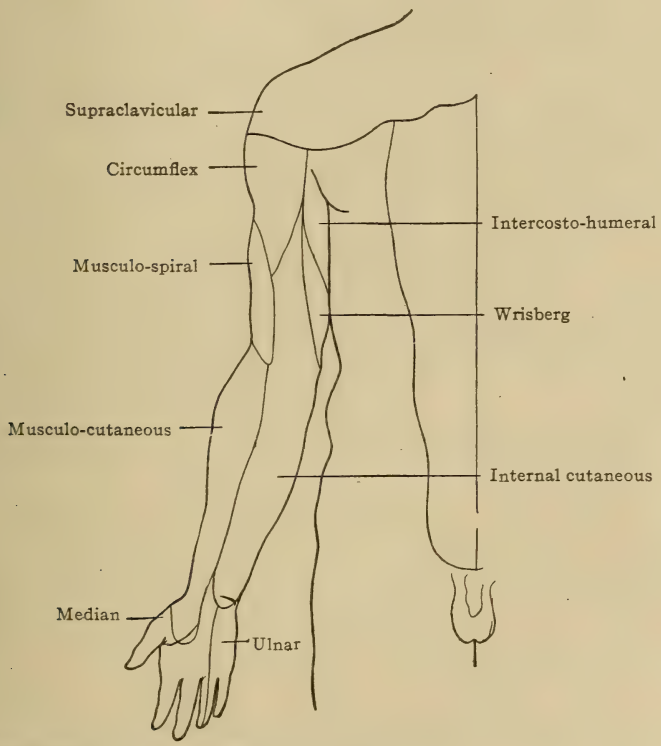


FIG. 602.—Distribution of the sensory nerves of the skin of the arm, anterior aspect. (*G. R. Butler.*)

marked swaying or a fall indicating "static ataxia" (Romberg's sign). If with the eyes open he cannot walk a straight line, he has "motor (dynamic) ataxia." If his gait is reeling and drunken (titubating), he has "cerebellar ataxia" and in this form the patient shows normal muscle sense and coördination if recumbent, the difficulty being one of equilibration. Motor ataxia. Cerebellar ataxia.

If his dyscoördination is associated with motor weakness he may show "paretic ataxia" or spastic ataxia according to the type of motor defect present. Even the "dynamic ataxia" may be found extremely complex if one analyzes the several factors which enter into or intensify it, *e.g.*, lost cen-

tripetal of deep sensory impulses from the muscles, bones and joints, cutaneous anesthesia and impaired muscle-tonus being most important.

Ordinary ataxia indicates cortical lesions or those of the pons crura and corpora quadrigemina, locomotor ataxia, transverse spinal lesions, ataxic paraplegia, Friedreich's disease or syringomyelia.

Stereognosis.—The inability to recognize familiar objects by touch (parietal lobe) usually suggests a lesion of the parietal lobe.

Significance of Sensory Disturbance.—*Hemianesthesia of hysterical origin* commonly affects the left side, is sharply defined and frequently complete even as to the special senses.

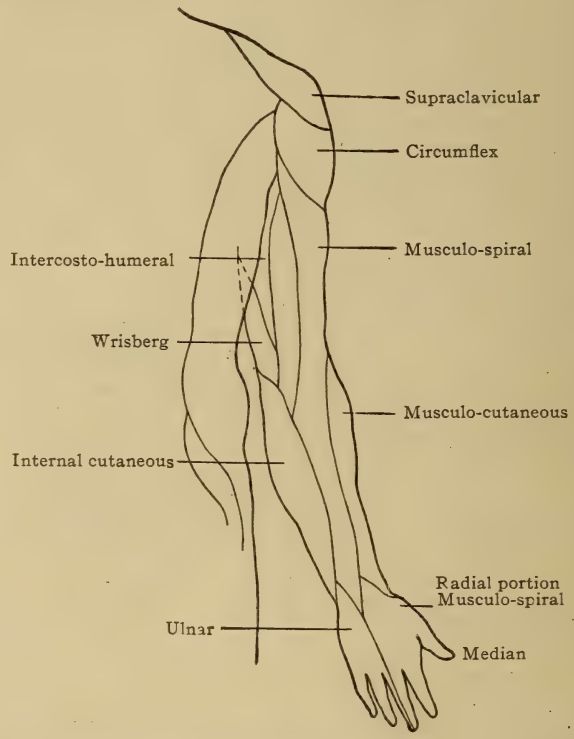


FIG. 603.—Distribution of the sensory nerves of the skin of the arm, posterior aspect. (*G. R. Butler.*)

Cortical hemianesthesia is combined with hemiplegia and usually incomplete unless associated with an unusually large lesion or involving the optic thalamus.

Crossed hemianesthesia associated with hemiplegia of the opposite side has been referred to as present in unilateral lesions of the cord (Brown-Séquard paralysis).

Hemianesthesia and hemiplegia with crossed oculo-motor paralysis indicates a lesion of the *crus*, and finally, combined hemianesthesia and hemiplegia *may* occur as the result of a lesion of the *internal capsule.*

Crus and internal capsule.

Spinal vs. cerebral.

Anesthesias of patchy distribution suggest hysteria or neuritis and as between

those monoanesthesias due to a spinal lesion and the rare cerebral form the former presents a sharp boundary line, the latter an anesthesia diminishing as the trunk is approached. As between hysteria and neuritis the former tends to disregard both segmental and individual nerve distribution.

Neuritis and hysteria.

Bilateral anesthesia without motor paralysis usually affects the lower portion of the body and is particularly common in traumatic neuroses and hysteria. *If the latter, it spares usually the skin of the genitals and a portion of the sacrum*, if from the spinal cord it would almost invariably be associated with paralysis or other evidence of cord disease.

Traumatic neuroses and hysteria.

Hyperesthesia and Hyperalgesia.—The well-known hysterogenic zones of hysteria furnish the best examples.* They may also be encountered in rickets, in brain tumor, as a zone above the anesthetic level in unilateral spinal hemiplegia, or be associated with meningitis, neuralgia and various toxic disturbances of the nerves.

Associated lesions.

Anesthesia dolorosa is encountered in compression of the spinal cord in which it co-exists with analgesia, but is associated with extreme pain. *Analgesia and hypalgesia* may be encountered, the former suggesting hysteria or syringomyelia.

Cord compression.

TOPICAL DIAGNOSIS

CERTAIN CEREBRAL CENTERS.—Certain areas in the cerebral cortex controlling certain bodily functions are known as *centers*. These, in many instances, cannot be sharply circumscribed and must be looked upon as physiological rather than anatomical units.

The Motor Centers.—These lie in the ascending frontal gyrus, the fissure of Rolando and the paracentral lobule. From these the functions of the muscles of the opposite side of the body are controlled and a fairly clean-cut differentiation between individual centers, as, for example, that of the arm and that of the leg, can be elicited by electrical excitation. Stimulation of such centers produces contractions in the muscles controlled, while destruction of them is followed by definite muscle group paralysis. The control of certain muscles which have a bilateral symmetrical action, such as the muscles of mastication and of the larynx, is not merely unilateral, but is undoubtedly exhibited by both cerebral hemispheres.

The part played by subcortical motor centers, though presumably of major importance, remains largely indeterminate.

As stated, the center for **speech** is situated in the inferior frontal and superior temporal gyri. The posterior portion of the left inferior frontal convolution forms the *area of Broca, the motor speech center in the right-handed.*

The Cortical Center for Sight is contained in the calcarine fissure and cuneus of the occipital lobe. The cortical center for the sense of **hearing** lies in the superior convolution of the temporal lobe. In the hippocampal gyrus of the temporal lobe is most probably situated the cortical center for the sense of **smell.** In the performance of the higher intellectual functions the

* These are chiefly, the breast, ovaries (deep pressure), groin, spinal column, and patella.

part played by the cortex of the frontal lobes in particular may be accepted with a fair degree of certainty.

CORTEX.—*In disease certain cerebral tracts are silent (symptomless) or yield only psychic reactions, others permit direct localizing diagnosis.*

Monoplegia is the type which permits most exact localization. The result of a lesion may be limited to the head, an extremity, or, in the case of the former, be confined to a single organ.

On the other hand, the close proximity of motor areas may produce associated monoplegias such as those of arm, leg, or face and arm, or incomplete hemiplegias, but usually with primary or ultimate predominate involvement of one area. Complete cortical hemiplegia is possible, but is rare. All unilateral cortical lesions produce paralysis of the side opposite the lesion.

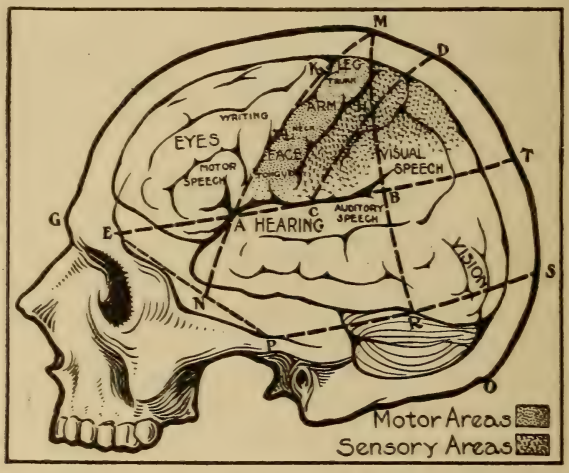


FIG. 604.—Motor and sensory areas of cortex. All lying anterior to the Rolandic fissure and above temporo-sphenoidal lobe are motor. The localization method of Chiene is also shown. This is applied as follows:—Find in the median line of the skull between the glabella (G) and the external occipital protuberance (O), the following points: The mid-point (M), the three-fourths point (T), and the seven-eighths point (S). Find also the external angular process (E) and the root of the zygoma (P) immediately above and in front of the external auditory meatus. Having found these five points, join EP, PS, and ET. Bisect EP and PS at N and R; also bisect AB at C and draw CD parallel to AM. The pentagon (ACBRPN) corresponds to the temporo-sphenoidal lobe, with the exception of its apex, which is a little in front of N. MDCA correspond to the Rolandic area, containing the fissure Rolando, and the ascending frontal and the ascending parietal convolutions. A is over the anterior branch of the middle meningeal artery and the bifurcation of the Sylvian fissures; AC follows its horizontal limb. The lateral sinus at its highest point touches the line PS at R. MA corresponds to the precentral sulcus, and, if it be trisected at K and L, these points will correspond to the origins of the superior and inferior frontal sulci. The supramarginal convolution lies in the triangle HBC. The angular gyrus is at B. (*Sherrington, Chiene and Grünbaum.*)

Silent Areas.—The frontal lobe may be greatly damaged without symptoms as may the *corpus callosum and corpus striatum*, though in right-handed persons *aphasia* may indicate a lesion of the left inferior frontal convolution, or *agraphia* one of the central convolution and it is probable that the *corpus striatum* is concerned in deglutition and contralateral muscle sense. A reference to the *motor areas* will show the lesions that must follow irritation

or destruction of any one of them alone, *i.e.*, *opposite facial paralysis, brachial monoplegia, crural monoplegia, hypoglossal paralysis*, etc., bilateral combined cortical lesions being extremely rare.

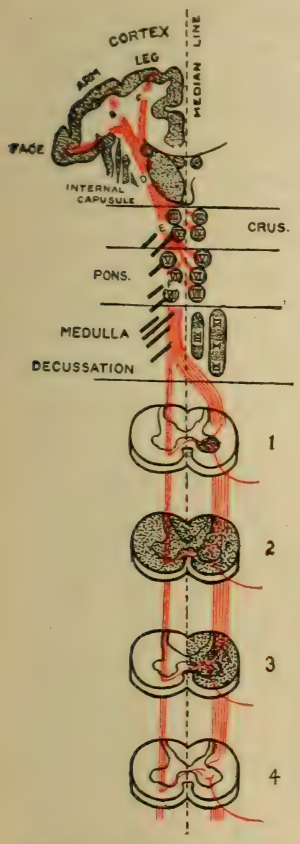


FIG. 605.—Explains symptoms caused by lesions affecting the motor tract in the brain and cord. Lesion at A, B, or C:—monoplegia of opposite side. Lesion at D:—hemiplegia of opposite side. Lesion at E:—oculo-motor paralysis of same side, hemiplegia of opposite side. Lesion at F:—facial and abducens paralysis of same side, hemiplegia of opposite side. Lesion of (1) anterior horns:—causes flaccid paralysis and lost knee-jerks. Lesion at 2:—spastic paralysis of muscles below lesion if pyramidal tracts only are involved or the lesion is incomplete; flaccid paralysis if lesion is complete. Lesion at 3:—causes Brown-Séquard's paralysis. 4.—Normal cord. (*After Van Gehuchten, modified.*)

Mere irritation would produce the peculiar phenomena of Jacksonian seizures, viz., clonic convulsions, initially representing the motor point of maximum irritation and following the motor areas in orderly succession. This is usually unilateral though rarely bilateral through commissural transmission and is ordinarily due to injury, rarely to paretic dementia. If the *parietal lobes* be involved there may be loss of sensation and of stereognostic sense on the opposite half of the body or ptosis (angular gyrus), conjugate deviation of the eyes or even visual aphasia and hemianopsia. *The occipital lobe* is so related to the optic tract as to produce hemianopsia, visual aphasia or even mind blindness. *The temporal lobe lesions* chiefly produce sensory aphasia, or, if bilateral, sound deafness.

Centrum Semiovale.—The structure and anatomical position of this great tract of projection and association fibers makes its lesions present either cortical or capsular symptoms and includes both marked sensory—and the widest range of motor—disturbances, including those of special sense.

Optic Thalamus.—It is said that the diagnosis of an isolated lesion of this ganglion depends chiefly upon the *loss of the facial expression* of *psychical emotion* upon the *opposite* side, the *voluntary* facial innervation being retained, a condition rarely observed. *Irritation symptoms* are sometimes present as in cortical lesions, as is crossed homonymous hemianopsia (pulvinar).

The last symptom occurs in lesions of the posterior third, and, if associated with hemichorea hemiathetosis or hemiataxia forms a definitely localizing syndrome.

Lesions of the Internal Capsule.—Almost all projection fibers between the cortex and the periphery (sensory, motor, and special sense) pass through this narrow and sharply defined area and any lesion entails serious consequences. *The anterior limb* is a symptomless area, but *the angular portion* (knee) is highly motor and

lesions therein cause paralysis in the opposite hypoglossal and lower facial distribution, and, if left-sided, aphasia. If in the posterior one-third of the posterior limb, *patchy hemilateral anesthesia,* and perhaps *hemianopsia* and *auditory disturbances.* If in the anterior two-thirds of the posterior limb, there is *hemiplegia of the opposite arm and leg. In most instances the paralysis is extensive and results in a combination of these lesions, i.e.,* complete hemiplegia. The upper branches of the facial, the trunk muscles, those of the neck, eye, and of mastication are paralyzed only in bilateral lesions.

The Crura.—At this level the mixed type of paralysis commences and extends throughout the pons and medulla, the relation of the pyramidal tract to the cranial nerve nuclei being such that the lesion may produce spastic paralysis of the lower segment (brachio-crural type) and a flaccid paralysis of cranial nerve areas. Thus, lesions of the crus are associated with hemiplegia with *crossed* oculomotor paralysis, sometimes including partial paralysis of the third and fourth nerves of *the other side* through basal exudate. The type of *tegmental* lesion is hemiataxia with crossed oculo-motor paralysis (Osler).

Gendrin's type.

Hemiplegia.

Tegmental lesions.

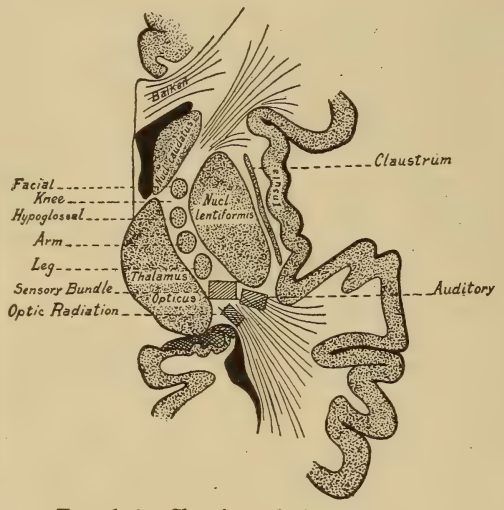


FIG. 606.—Showing relative position of motor and sensory fibers in the internal capsule. (*After Monakow.*)

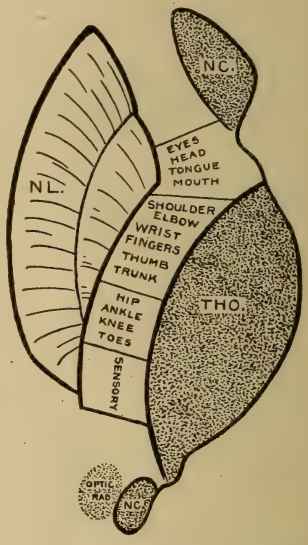


FIG. 607.—Topography of motor tract in internal capsule.

Lesions of the corpora quadrigemina are characterized by *flaccid nuclear or tract oculo-motor paralysis, third and fourth nerves, stumbling gait, and ataxia (cerebellar type).**

Lesions of the Lower Pons.—The picture is usually characteristic, the ordinary type of paralysis being a crossed spastic hemiplegia, with paralysis

* The region is rich in nuclei and lesions may be unilateral or bilateral and involve the tegmentum (incomplete hemianesthesia), the optic tract or lateral geniculate body (hemianopsia), the crusta (paralysis of opposite leg and arm or even hypoglossal and facial hemiplegia) or produce defective hearing through involvement of the median geniculate body (Jakob).

of the facial and trigeminus on the side of the lesion, *i.e.*, paralysis of the muscles of mastication, anesthesia of trigeminal distribution and facial paralysis. If the lesion is extensive, bilateral paralysis of the extremities may result. *Abducens paralysis* (*external rectus of same side, and internal rectus opposite to lesion*), defective taste and articulation (facial and hypoglossa, fibers), brachio-crural hemiplegia of opposite side, and, perhaps, ataxia, vertigo, or trismus are also encountered in the lesions of the *lower pons*.

Lesions of the Upper Pons.—These produce paralysis of the face and extremities of the side opposite the lesion, as in the case of the internal capsule.

Lesions of the Medulla Oblongata.—Here again lesions may be unilateral or bilateral. *The type is: brachio-crural hemiplegia and hemianesthesia of the* Coarse lesion.

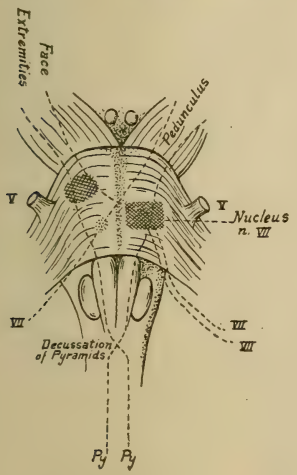


FIG. 608.—Illustrating the mechanism of crossed paralysis in pontine lesions. (*After Hermann.*)

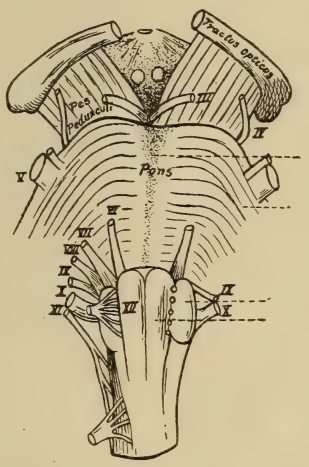


FIG. 609.—Ventral aspect of medulla oblongata. (*After Edinger and Hermann modified.*)

opposite side with the tongue pointing toward lesion, or, if the fillet be involved, bilateral anesthesia. Anarthria or dysphagia results and the type of *glosso-labio-laryngeal paralysis* may be present and is usually bilateral. *Coarse lesions are rapidly fatal.* Involves lower cranial nerves.

Lesions of the Cerebellum.—Though sometimes lacking symptoms if unilateral, the chief and commoner characteristics of involvement of the *vermiform process* are cerebellar *ataxia* (staggering, drunken gait), vomiting, headache, *vertigo* or even movements of forced body rotation. May be symptomless. Ataxia and vertigo.

The ataxia of cerebellar disease is characterized by being almost entirely limited to the muscles of the pelvic girdle and the lower extremities. Nystagmus and optic neuritis are common. Incoördination is coarse and may be greatly diminished in recumbency with closed eyes.

SUMMARY OF SEGMENTAL PARALYSES

Segment Symptomatology.—The paralysis of muscle groups is the basis of localization in diseases of the cord and peripheral nerve lesion, and the

diagrams show the lack of correspondence between the point of nerve emergence, its segmental origin and the segmental sensory areas. So far as possible this topic will be dealt with on the basis of *segment symptomatology* preceded by a discussion of lesions affecting certain of the cranial nerves.

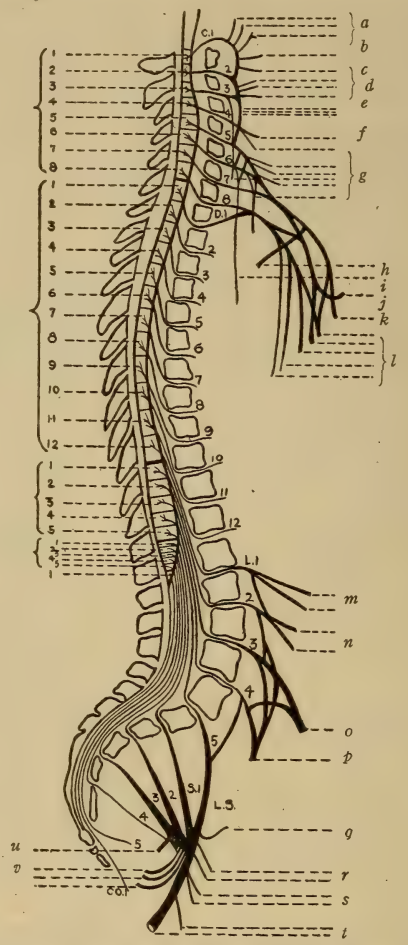


FIG. 610.—Segments of spinal cord and their relation to the vertebral landmarks. The variation between the level of origin of the spinal nerves and their point of exit is well shown. The numerals indicate the segments, those to the right the spinal nerves, and corresponding vertebræ. The lettered nerves when grouped will be named in their order. *a*, nerves to rectus lateralis, to rectus anticus minor, anastomosis with hypoglossal; *b*, anastomosis with pneumogastric; *c*, nerve to rectus anticus major; *d*, to mastoid and great auricular and transverse cervical; *e*, to trapezius, ang. scap. and rhomboid; *f*, supraclavicular and supra-acromial; *g*, phrenic, lev. ang. scap. to rhomboid subscapular and subclavicular; *h*, to pectoralis major; *i*, posterior thoracic, serratus magnus; *j*, circumflex; *k*, musculocutaneous; *l*, median, radial, ulnar internal cutaneous and lesser internal cutaneous; *m*, ilio-hypogastric and ilio-inguinal; *n*, external cutaneous and genito-crural; *o*, anterior crural; *p*, obturator; *q*, superior gluteal; *r*, to pyriformis and gemellus superior; *s*, to gemellus inferior and quadratus; *t*, lesser and great sciatic; *u*, to levator ani; *v*, to obturator int. to sphincter ani and coccyx. (*Dejerine and Thomas.*)

The Tongue (*Hypoglossal Nerve*).—In bulbar palsies and basal processes deviation of the tongue to the paralyzed side (geniohyoglossus), loss of

backward movement (styloglossus) or deviation toward the sound side when retracted or the paralyzed side when protruded, associated perhaps with atrophy, unilateral or bilateral (lingualis), may be encountered.

The Uvula and Velum Palati (*Pharyngeal Plexus, possibly Seventh Nerve*).—Nasal voice and regurgitation of food through the nose indicates bilateral palsy; deflection toward the sound side, unilateral palsy of the *azygos uvulæ*. Food regurgitation and immobility in "ah" intonation (paralysis involving the levator palati). Food regurgitation and nasal speech are also seen in palato-pharyngeus paralysis (fifth nerve).

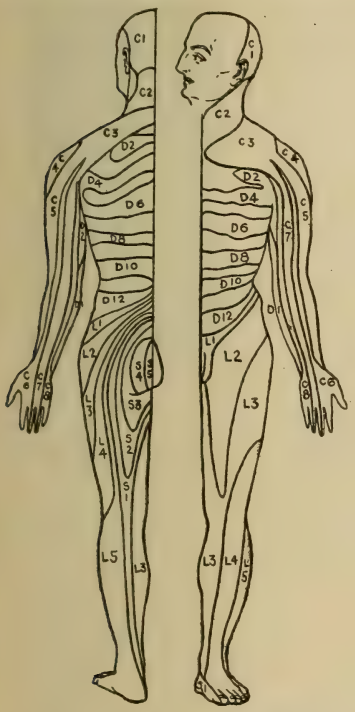


FIG. 611.—Areas of anesthesia following segmental lesions of spinal cord, the numbers shown corresponding to the affected segment. (*After Starr.*)

Pharynx and Larynx.—Choking in deglutition due to the entrance of food into the larynx may arise from the failure of epiglottis closure due to paralysis of the stylo-pharyngeus (*glosso-pharyngeal nerve*), or to deficient downward movement due to paralysis of the constrictors (*pharyngeal plexus*).

Hoarseness, aphonia or paralytic stenosis and the various faulty positions of the cords as shown in the illustration may be due to paralysis of the musculature (recurrent laryngeal nerve, excepting for the crico-thyroid).

Spasm or paralysis of the sterno-cleidomastoid (*spinal accessory, medulla, first, second, third cervical segments*) may cause wry neck (spasm), the head being inclined to one side with the chin raised and pointing to the opposite shoulder. If unilaterally paralyzed, the face cannot be turned to the opposite side; if bilateral, the head cannot be raised from the bed in recumbency.

Flexion and Rotation of the Head.—If the chin cannot be brought to the chest the rectus capitis anticus, major and minor, are at fault. Failure of rotation can seldom be traced to the rectus capitis lateralis and is usually due to sterno-mastoid paralysis.

Deficient thoracic respiratory movement affecting the elevation and lateral movements of the ribs suggests paralysis of the scaleni (*lower cervical segments*).

Deficient Upper Spine Flexion.—This suggests lesions involving the longus colli (*lower cervicals*). Impaired power to raise the chin may be due in part to a lesion involving the clavicular portion of the trapezius (*spinal accessory*); a depressed shoulder to a lesion of the middle portion; abduction of the scapula to a lesion of the lower portion (*spinal accessory, second and third cervical segments*).

Loss of power to approximate the scapulæ indicates a lesion of the rhomboids (*fourth and fifth cervical segments*).

Oblique Position of the Scapula.—The inferior angle approaches the median line, the bone is raised, the arm cannot be raised above the horizontal position and if stretched forward the scapula projects (alar scapula). This indicates weakness or paralysis of the serratus magnus (*posterior thoracic nerve, fifth and sixth cervical segments*).

Inability to Raise Arm though Shoulder Rises.—The latter is flattened from atrophy and a groove appears below the acromiom. This indicates paralysis of the deltoid, any one of the three divisions of which may be separately affected (*circumflex nerve, fourth, fifth and sixth cervical segments*). If the arm cannot be moved outward, the infraspinatus is involved (*supra-scapular*). If writing is difficult the teres minor is involved (*circumflex*), if the arm cannct be moved inward, a lesion of the subscapularis is suggested (*subscapular*). All three represent *the fourth, fifth and sixth cervical segments.*

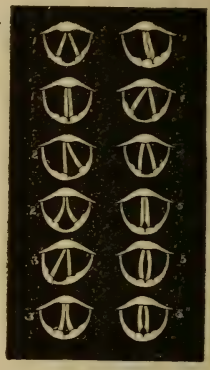


FIG. 612.—Vocal cords. (Diagrammatic mirror picture.)

1. Normal position in breathing and phonation respectively.

2. Adductor paralysis (left) 2′. Bilateral adductor paralysis. Both in phonation.

3. Unilateral abductor (left) and 3′. bilateral abductor paralysis both during breathing.

4. Left recurrent paralysis phonation, 4′. same in respiration, 4″. recurrent bilateral in both respiration and phonation.

5. Arytenoid paralysis, phonation. 5′. Thyro-arytenoid paralysis, phonation 5″. Arytenoid and thyro-arytenoid paralysis.

Impaired lateral trunk movement with failure of dorsal spine extension and backward movement of the arm indicates a lesion involving the latissimus dorsi (*subscapular, sixth and seventh cervical segments, chiefly*).

Inability to firmly approximate the volar surfaces with extended arms suggests a lesion of the pectoralis major (*anterior thoracic nerve, fifth, sixth and seventh cervical segments*).

Inability to extend arm against resistance indicates a lesion of the triceps which is associated with a tendency to subluxation of the humerus, either spontaneous or induced by slight causes (*musculo-spiral, sixth, seventh and eighth cervical segments*).

Impaired flexion of the arm suggests a lesion of the biceps (*musculo-cutaneous, fourth, fifth and sixth cervical segments*), and if flexion and pronation are both deficient the supinator longus is to be considered. The arm is usually spindle-shaped from atrophy of the muscle (musculo-spiral nerve, fourth, fifth and sixth cervical segments).

Deficient supination with arm extended indicates supinator brevis involvement (*musculo-spiral, fifth cervical segment*).

Failure of dorsal flexion and abduction of the wrist with atrophy of the forearm indicates a lesion of the extensor carpi radialis longior and brevior; if adduction also fail from paralysis of the extensor carpi ulnaris, drop wrist is produced; if extension and abduction of the first phalanges fail, a paralysis

of the three extensors of the fingers is indicated (*musculo-spiral and seventh cervical segment*).

Impaired flexion of the fingers and wrist suggests paralysis of the flexors of the wrist and fingers. The median nerve supplies the flexor carpi radialis, palmaris longus, flexor sublimus digitorum, and, in part, the deep flexor of the digits, the ulnar supplying the remainder of the last-named and the flexor carpi ulnaris, all being related to the *eighth cervical segment.*

Failure of abduction and adduction of fingers suggests a lesion of the interossei and lumbricales (*ulnar and median eighth cervical and first dorsal segments*). With extension of the first phalanges, flexion of the second and third and deep interosseus spaces it forms the claw hand (*main en griffe*).

Atrophy of the Ball of the Thumb.—An impairment of its extension and adduction suggests a lesion of the extensor pollicis brevis and thenar muscles, to which is added flexion of the second toward the first phalanx if the extensor pollicis longus is involved, with deficient abduction if the abductor pollicis longus is affected (*musculo-spiral nerve, first dorsal segment*). If flexion of the thumb fails and atrophy is present the "*ape hand*" ("*monkey hand,*" "*Simian hand*") is formed (abductor brevis pollicis, flexor brevis and adductor pollicis). The opposing movement may be absent (opponens pollicis and in part flexor brevis and abductor pollicis brevis) or flexion of the terminal phalanx may fail from paralysis of the flexor pollicis longus. The nerve supply is from the *median and ulnar and first dorsal segment.*

Lower spinal lordosis with deflection toward sound side in unilateral lesions indicates a lesion involving the erector spinæ, sacro-lumbalis and longissimus dorsi (*dorsal nerves, second to twelfth dorsal segments*).

Lordosis with Prominent Nates and Abdomen.—Inability to rise up from a dorsal recumbent position without assistance from the hands indicates a lesion of the abdominal muscle group (*dorsal nerves, second to twelfth dorsal segments*).

Imperfect lateral movement of the lower spine suggests involvement of the quadratus lumborum (*lumbar nerves and segments*).

Loss of thigh adduction indicates paralysis of the adductors (*obturator nerve, great sciatic and crural, third lumbar segment*). The thigh rolls outward.

Imperfect flexion of the thigh suggests involvement of the sartorius (*crural nerve, third lumbar segment*).

Impaired leg extension indicates involvement of the quadriceps femoris (*crural, third lumbar segment*).

Impaired flexion and difficulty in rising from the horizontal position suggests involvement of the ilio-psoas (*crural nerve, fourth lumbar segment*) and the tensor fasciæ femoris (*superior gluteal, fourth lumbar segment*).

Impaired outward rotation, *the leg being turned inward,* suggests involvement of the external rotators, pyriformis, gemelli and quadratus femoris (*sacral plexus, fifth lumbar segment*), as well as the internal and external obturators (*obturator nerve, lumbar plexus*).

Complete loss of thigh extension and abduction with a waddling gait and inability to climb indicates paralysis of the gluteal muscles (*inferior gluteal nerve, sacral plexus, first and second sacral segment and superior gluteal, first and second sacral segments*).

Impaired flexion, perhaps associated with hyperextension through the action of the quadriceps points to a lesion affecting the biceps, semitendinosus and semimembranosus.

Deficient foot extension, *i.e.*, patient cannot stand on tiptoes or raise heel, indicates gastrocnemius, plantaris and soleus paralysis (*internal popliteal nerve, fifth lum.bar segment*).

Drop Foot.—Usually with excessive knee and hip flexion with perhaps pesequinus or equinovarus from contracture points to involvement of the anterior tibial muscles, *i.e.*, tibialis anticus, extensor longus pollicis (*anterior tibial nerve, fifth lumbar and first sacral*).

Deficient Abduction.—Tendency to flat-foot increased by contracture points to involvement of the peroneus longus (*peroneal nerve, first and second sacrals*).

Deficient adduction with resulting deformities suggests involvement of the tibialis posticus (*posterior tibial nerve, first and second sacrals*), and the peroneus brevis (*peroneal nerve, first and second sacral segments*).

Adduction of Toes.—Paralysis of interossei with hyperextension of first phalanges and flexion of the second and third (claw foot) indicate involvement of the interossei and lumbricales (*posterior tibial, first and second sacral segments*).

Deficient Toe Flexion.—Impaired power to push foot off ground indicates a lesion of the adductors, flexor brevis and abductor hallucis.

BRACHIAL PLEXUS PARALYSIS.—Paralysis usually results from traumatism or compression commonly below the clavicle, and may be either partial or complete. If only the upper portion be involved, the lower muscle groups (forearm and hand) may with the exception of the supinator longus escape ("Erb's type"). Complete lesions involve a total motor and sensory paralysis of the arm, and there is a lower arm type in which forearm extension and the use of the hand are lost, the triceps, flexors of the wrist, pronators extensors and flexors of the fingers being involved. Ocular signs indicating involvement of the sympathetic are frequent.

SUMMARY OF LESIONS OF CERTAIN SPINAL NERVES.—Occipital.—Neuralgia, anesthesia.

Posterior Thoracic.—Arm cannot be raised above horizontal and rotated. Projecting scapula (serratus magnus).

Anterior Thoracic.—Inability to adduct arm (pectoralis major).

Musculo-cutaneous.—Loss of elbow flexion power (biceps, brachialis anticus). Anesthesia inner border of forearm.

Circumflex.—Loss of outward rotation and elevation of arm (deltoid, teres minor), third head of triceps.

Suprascapular.—Impaired outward shoulder rotation and elevation.

Musculo-spiral and Radial.—Paralysis from lead, arsenic or alcohol,

tumor and crutch pressure. Traumatism or whatever cause, is common and leads to the characteristic dropping of the wrist from extensor paralysis (*drop wrist*) so commonly seen in clinics. A factor in differential diagnosis is the involvement or non-involvement of the supinator longus which is usually spared in lead poisoning. The triceps and anconeus are paralyzed, extension of forearm lost and there is wasting of the back of the arm.

Median.—Abduction of thumb and flexion of thumb and first and second fingers are lost. Thenar and anterior forearm atrophy is usually marked. If the forearm be flexed pronation is impossible. The thumb and index finger are approximated and there is paralysis of the abductor pollicis.

FIG. 613.—Drop wrist.

Ulnar Nerve.—Impaired abduction and flexion of hand (flexor carpi ulnaris and part of flexor sublimis digitorum) and wasting of ball of little finger and interosseous spaces are present and one cannot cup the palm nor span. The ring and little fingers are "clawed" ("claw hand," "*main en griffe*"). Paralysis of their lumbricales and interossei prevent flexion of the proximal or extension of the distal phalanges and the unopposed muscles exaggerate the position.

Intercostal Nerves.—Violent neuralgic pains or other symptoms of irritation occur, sometimes herpes zoster along the course of affected nerves or anesthesia.

Crural Nerve.—Patient cannot stand or walk and tendon reflex is absent (psoas-quadriceps extensor).

Obturator.—The obturator plexus is a frequent seat of pain, referred to the inner side of the thigh and knee-joint in disease of the mid-lumbar, sacroiliac and hip-joints. Obturator paralysis means loss of adduction.

Sciatic Nerve.—Paralysis of leg flexors, foot and toe, sciatica.

Peroneal Nerve.—Foot-drop from paralysis of flexors of foot or leg by contracture, talipes equinus or varus may appear.

Tibial Nerve.—Paralysis of calf muscles, loss of foot extension or toe flexion. Talipes if contractures occur.

FIG. 614. Dropped foot.

THE CRANIAL NERVES.—Irritation produces excess or perversion of functional activity, *i.e.*, spasm, tremor, pain, itching, formication, etc. Destructive lesions cause ultimate loss of function, muscle atrophy, etc., inasmuch as a peripheral neuron is thus disturbed or destroyed. The nucleus of the cranial nerve corresponds to the nutrient anterior horn cell of the spinal nerve.

Olfactory Nerve.— *Test.*—Apply to each nostril in turn bottles containing well-known aromatic oils (peppermint, cloves, asafetida, bay, etc.) and

have the patient describe the odor. Any inflammation, degeneration, necrosis, traumatism or pressure affecting the terminal filaments, bulb or tract, from the uncinate gyrus and thalamus to the Schneiderian membrane, produces lessened, absent (*anosmia*), or perverted (*parosmia*), function. These constitute also symptoms of hysteria, epilepsy, locomotor ataxia and insanity (*hallucinations of smell*). Trigeminal paralysis and certain catarrhs act no doubt by lessening the secretion.

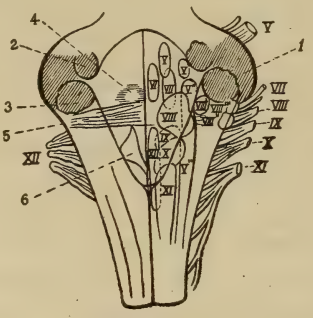


FIG. 615.—Nuclei of cranial nerves in medulla oblongata. (*After Erb.*)

The nuclei by Roman numerals. The nerve roots are similarly shown at the side. 1, Brachium pontis. 2, Brachium conjunctivæ. 3, Cerebellar peduncle. 4, Eminentia teres. 5, Striæ acousticæ. 6, Ala cinerea.

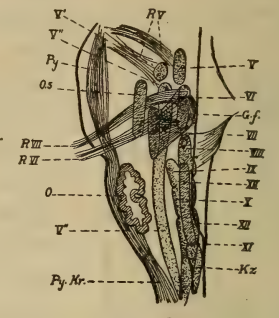


FIG. 616.—Lateral aspect cranial nerve nuclei within medulla oblongata. (*After Erb.*)

THE EYE, ITS REFLEXES AND THE OPTIC NERVE.—The Pupil.—*The shape, equality or inequality on the two sides, and the mobility or response to light and accommodation should be noted.*

Irregularities in outline suggest chiefly adhesions from past iritis. In acute iritis the color of the iris is turbid and greenish, the corneal circumference showing an hyperemic zone. Difference in the color of the iris of the one eye as compared with the other is especially important in detecting iritis in eyes normally dark. The commoner causes of iritis are gout, rheumatism, trauma and syphilis and the first two are usually of the unilateral relapsing type, the last is bilateral, belongs to the period of secondary infection and seldom recurs.

Oculo-motor and sympathetic.

Inequality.—Even in healthy persons, slight inequality occurs, *but decided pathologic* irregularity is important and may represent either unilateral contraction or dilatation.

Contraction (miosis) may be due to iritis, oculo-motor irritation, or paralysis of the sympathetic, but occurs normally with suspended reflexes, e.g., in sleep.

Dilation (mydriasis) indicates conversely *sympathetic irritation* (aneurysm of the arch, goiter, enlarged glands or other swelling, etc.) *or oculo-motor paralysis.* Furthermore, glaucoma, cataract, disease of the optic nerve and the retinal hemorrhages of albuminuria may cause a dilated pupil, unilateral or bilateral.

Local disease.

Persistent mydriasis is frequently noted in cases of hysteria, high myopia, neurasthenia, severe dyspnea or actual asphyxia, cerebral abscess, hemor-

rhage, thrombosis and tumor, exophthalmic goiter, epileptic coma, catalepsy melancholia, mania, active delirium, late meningitis, aortic regurgitation, shock, fear or other strong emotion, nausea, and poisoning by such mydriatics as tobacco, alcohol, nitrous oxid gas, cocain, chloral, chloroform, ether, duboisin, hyoscin, scopolamin, stramonium, conium and belladonna. Certain cases of locomotor ataxia show as an early symptom dilated pupils and lid-ptosis, with beginning atrophy of the nerve head in many instances, *and such cases run a course marked by long delay in the development of ataxic and paralytic symptoms.*

Early eye changes in tabes.

Persistently Contracted Pupils.—The following conditions may be associated with persistent bilateral miosis. Locomotor ataxia, general paresis, high hypermetropia or astigmatism, disseminated sclerosis, tumor, hemorrhage, meningitis, inflammation or degeneration of the brain or cord, uremia, sunstroke (early stage), photophobia, congestion of the iris, etc.

The primary action of ether, chloral, opium, eserin, and pilocarpin is miotic and general venous congestion, unless associated with marked dyspnea, may produce bilateral pupillary *contraction.*

Unilateral Contraction.—Marked *unilateral* contraction aside from adhesions, iritis, paretic dementia and apoplexy is an important suggestive sign of a serious lesion of the brain, cervical cord or any region involving the motor oculi or sympathetic. Mediastinal tumors aneurysmal, glandular or malignant, with their pressure effects, account for many cases, and should be constantly borne in mind. In not a few cases *an artificial eye* has puzzled temporarily the careless physician. Tuberculous cases often present persistent slight dilatation, usually unequal and sometimes merely transient.

Often a serious symptom.

Trivial.

Hippus.—An oscillating contraction and dilatation of the iris on sudden exposure to light is of little clinical importance, though often associated with nystagmus and sometimes present in disseminated sclerosis.

EYE REFLEXES.—Light response: *Proper testing demands that the consensual light reflex be remembered and involves both light and accommodation reactions,* that of convergence being of no special importance. The best methods demand the use of the ophthalmoscope in a darkened room and the use of such a + lens as will enable the observer to see the magnified pupil as the mirror flashes the light upon the eyes. Each must be separately tested and the patient should look slightly to the right or left of the observer to avoid the accommodation reaction. As a matter of fact there is normally a sympathetic reaction of the other pupil (*consensual reaction*).

Direct and indirect.

Reaction to Accommodation.*—*Ordinarily contraction of the pupil attends accommodation for near vision. In locomotor ataxia and general paralysis especially, this reflex persists though that for light is lost (Argyll-Robertson pupil).*

Argyll-Robertson pupil.

In general, such a pupil indicates a lesion of the optic tract, or Meynert's fibres, whereas fixed contraction indicates a lesion of the centre or of the *motor oculi.*

THE OPTIC NERVE.—The primary optic neurons† receive the visual

* Probably inseparable from the convergence reaction.

† "The true optic nerve" (Church and Peterson).

DESCRIPTION OF PLATE XII

FIG. 1.—NORMAL FUNDUS OF LEFT EYE.

Direct Method.—Disc is round, having light-colored center, distinct margins and yellowish-red intermediate zone. Choroidal ring almost complete and slightly more pigmented to the temporal side. Two cilio-retinal vessels on the lower outer edge of disc passing toward the macula. The crescentic fovea centralis, with surrounding blood-red area, is unusually well shown and most typical in youth. Veins and arteries are slightly to the nasal side of the disc. The arteries cross the veins on the disc, but in the periphery the veins cross the arteries.

FIG. 2.—EMBOLISM OF THE CENTRAL ARTERY OF THE LEFT EYE. DIRECT METHOD.

Large, oval-shaped, blanched area, which includes the disc and macula. Cherry-red spot at the macula. Intermediate zone of the disc shows apparently normal except for a faint fogginess. The arteries are almost empty and the smaller ones have the blood stream broken in different places. The arteries have lost their light streak. The veins are slightly distended, but not tortuous; the light streak is almost lost in the veins, but can be faintly seen.

FIG. 3.—THROMBOSIS OF THE CENTRAL VEIN OF THE LEFT EYE. (APOPLEXY OF THE RETINA, HEMORRHAGIC RETINITIS.)

Fundus Changes.—Left Eye. Acute papillitis. Disc very much swollen and apex seen with + 4 D. and fundus without any lens at the sight hole. Arteries small and very few of them in view. The veins are very tortuous, looking like half hoops or serpentine, hence the name, "Medusa Nerve." The light streak in the veins is conspicuous at the top of each loop. The hemorrhages are of all sizes and shapes and shades of red.

FIG. 4.—ALBUMINURIC RETINITIS. BRIGHT'S DISEASE. RIGHT EYE. DIRECT METHOD.

Fundus Changes.—Swollen disc, striated edges; exudation ("snow banks") about its edges with two areas above and one below the disc. Macular figure unusually well marked. Many scattered and flame-shaped hemorrhages seen in the periphery and about the disc. Vessels about the disc show effusion into their sheaths by the white edge at each side of the vessel. The disc resembles that of choked disc in brain tumor, but the "snow banks" and macular figure are almost too conspicuous for such a diagnosis.

PLATE XII

Fig. 1. Normal Fundus

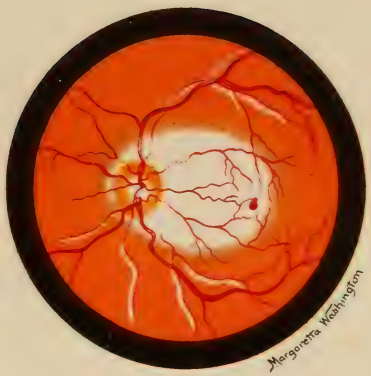


Fig. 2. Embolism of the Central Artery

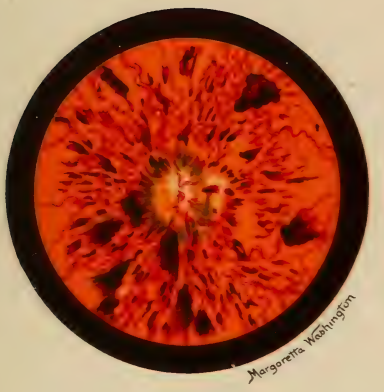


Fig. 3. Thrombosis of the Central Vein (So-called Hemorrhagic Retinitis)

Fig. 4. Albuminuric Retinitis

(From Thorington's "Ophthalmoscope and How to Use It")

impressions through their dendrites, the retinal rods and cones, the axons passing to the visual centers in the optic thalamus (pulvinar), corpora quadrigemini and geniculate bodies, through the posterior portions of the internal capsule and finally, in the optic radiation, to the cuneus.

The axons from the nasal sides of the retina, which represent the outer and larger portion of the visual fields, cross and pass to the centers of the opposite side. Those of the temporal portion (inner and lesser visual field) run direct to the centers of their own side. The point of decussation (chiasma), therefore, represents all fibers.

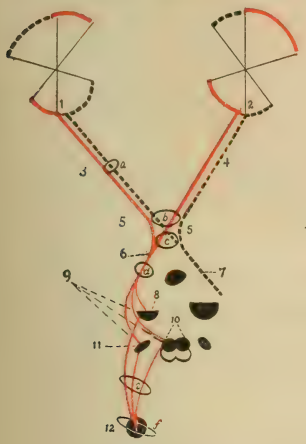


FIG. 617.—Course of optic nerve fibers from cortex to retina showing relation of visual fields. (*After Sahli, slighly modified.*)

Explanation. A lesion at *a* produces unilateral blindness, at *b* and *c* a temporal hemiopia by interruption of the nasal fibers, at *d*, *e* or *f* a lesion will produce right homonymous hemiopia. 1 and 2 represent the retina; 3 and 4, optic nerves; 5, the chiasm; 6 and 7, optic tracts; 8, the pulvinar; 9, the primary optic center; 10, the anterior corpora quadrigemini; 11, the external geniculate body, 12, the occipital lobe. The right and left tracts, their respective retinal distribution and visual fields are indicated by the solid red (left tract) and dotted black (right tract) lines.

Anterior to it lie the mixed fibers for the respective halves of each eye; posteriorly, those for the outer segment of the eye of the same side (nasal visual field) and the inner portion of the opposite eye (temporal visual field).

Interference with these fields *causes hemianopsia* and the *variety of the hemianopsia* and the *naming of the affected visual field* represent the *patient's viewpoint;* hence *the variety of hemiopia is the reverse of the retinal field affected, i.e., temporal retinal change = nasal hemianopsia,* etc.* *The condition may be unilateral or bilateral, total, partial or concentric,* affecting *corresponding halves* (homonymous), *both temporal or both nasal fields* (heteronymous), or even *both upper or lower halves* (superior or inferior altitudinal). **Hemiopia.**

A lesion may occur at any point from and including the retinal surface to the cortical centers. If unilateral and in front of the *chiasm,* blindness of that eye follows. *If at the chiasm* either the anterior or posterior angle (nasal half of retina) is usually affected causing *bitemporal hemianopsia. If the outer portion only be involved,* unilateral or bilateral nasal hemianopsia follows. A lesion behind the chiasm if on the right side causes left hemianopsia; if on the left, right hemianopsia.† Precisely the same phenomena occur if a lesion affects the more central tracts, save that the difficult "hemianopic" (Wernicke's) pupillary reaction is to be considered. **Effect of lesions.** **Wernicke's localizing reaction.**

Test.—Carefully note size of pupils by plane mirror illumination in a dark room, then with the ophthalmoscope direct a narrow, strong beam of

* Hemiopia, hemianopsia, hemianopia are synonymous terms.

† For example, a lesion of the right tract cuts off the retinal impressions of its own temporal half and the opposite nasal half of the retina, hence left visual fields are lost for both eyes (left homonymous hemianopsia).

DESCRIPTION OF PLATE XIII

FIG. 1.—ALBUMINURIC RETINITIS OF PREGNANCY. NEURO-RETINITIS. PAPILLO-RETINITIS. LEFT EYE. DIRECT METHOD.

Fundus Changes.—Disc hidden and slightly swollen. Punctate dots above and at the macula (neuritic dots) are conspicuous. Areas of exudation some distance from the disc Hemorrhages numerous, small and flame-shaped. The veins full and tortuous, some arcovered by the swollen retina.

FIG. 2.—RETINITIS DIABETICA. RIGHT EYE. DIRECT METHOD.

Fundus Changes.—Arteriosclerosis showing in the upper and lower temporal vessels where they cross. Small flame-shaped and round hemorrhages scattered irregularly in the fundus. The disc edges are foggy and the membrana cribrosa is indistinct. The intermediate zone and in fact the entire disc has a canary-yellow color appearance.

FIG. 3.—RETINITIS PIGMENTOSA. RIGHT EYE. DIRECT METHOD.

Fundus Changes.—The periphery of the eye ground is characteristic of many myopic eyes, the choroidal vessels being very conspicuous. The retinal vessels are not very numerous and the smaller ones can be traced with difficulty in the periphery. The disc is quite yellow. The retinal vessels narrow. The reflex immediately around the disc approximates the normal, but beyond this the condition is atrophic. A few stellate pigment spots, together with irregular pigment massings on the vessels, are scattered throughout the fundus. This is an unusual variety of the disease. The patient is color blind for red and has night blindness.

FIG. 4.—PARTIAL DETACHMENT OF THE RETINA. RIGHT EYE. DIRECT METHOD.

Fundus Changes.—Retina detached downward and forward. The wavy condition of the retina, the course of the dark-colored vessels without their usual light streaks, as they appear on the detachment, are all quite characteristic. This disc appears foggy because it is out of focus as compared with the detachment. The disc is seen with — 7 D., whereas the detachment is best seen with a + 1 D. The white streaks are very likely congenital and possibly obliterated vessels. A rupture of the choroid would be crescentic in shape and situated elsewhere in the fundus.

PLATE XIII

Fig. 1. Albuminuric Retinitis of Pregnancy

Fig. 2. Retinitis Diabetica

Fig. 3. Retinitis Pigmentosa

Fig. 4. Detachment of the Retina

(From Thorington's "Ophthalmoscope and How to Use It")

light upon the *blind portion* of the retina. If *pupillary contraction* occurs, the lesion is posterior to the corpora quadrigemina.* It should be remembered that (*a*) *tract lesions ordinarily produce hemianopsia*, (*b*) *nerve lesions total blindness* and that (*c*) *central vision is retained in unilateral tract lesions* because *the macula of each eye receives fibers from both optic tracts.*

General laws of localization.

Diagnostic Significance.—*Disturbances of vision due to tract lesions are usually associated with gummata, new growths and syphilitic and tuberculous meningitis.*

Causes of tract lesions.

Associated Lesions.—Incomplete hemianopsia with aphasia and mind or word blindness indicates a *cortical lesion.* *Angular gyrus lesions* are suggested by impaired vision in one eye associated with a contracted visual field of the opposite eye. *Athetosis suggests a pulvinar lesion; altitudinal hemianopsia, a lesion of the upper or lower part of the chiasma, or if unilateral, a lesion of the cuneus.*

Angular gyrus.

Pulvinar.

Chiasma and cuneus.

Hysterical hemianopsia is associated with the usual stigmata, hemianesthesia, insensitive conjunctiva, and *more often takes the form of contracted fields and alteration of color vision areas. Fugitive hemianopsia may accompany migraine. Total unilateral blindness may be due to a destructive lesion of the occipital lobe or optic nerve. Bilateral lesions of the cuneus or optic nerves or total destruction of the chiasma will cause bilateral blindness. High refractive errors of long standing may be associated with unilateral loss of vision.*

Hysteria.

Total blindness.

Amblyopia, Amaurosis.—*Amblyopia and amaurosis* describe respectively, the one, impairment, the other, total loss of vision without actual lesions, but in the latter associated with various neuroses and toxemias. *Amaurosis* is alarming but usually *transitory*, lasting from a few hours to several days. It suggests uremia, diabetes, severe and *especially acute* anemias, spasmodic contraction of retinal vessels, hysteria and migraine, and has followed the administration of quinin and the salicylates, the overuse of alcohol and tobacco, cerebral trauma and lead poisoning.†

Transitory blindness.

Testing Vision.—*Acuity.*—If total blindness be suspected the patient should be asked to count the fingers of the physician as held before him at varying distances; failing in this, he should be tested for light perception in a dark room‡ (see also "Simulated Blindness").

The Visual Field.—Rough Test.—Squarely face the patient at a distance of 18 inches. If the left eye is to be tested the right should be covered and the patient told to look squarely and fixedly at the right eye of the examiner whose left is closed. The latter then holds his open hand well off to the side but on a level with the eye and brings it inward, moving the fingers constantly until the patient sees them *without moving the head or eyeballs;* the same procedure tests all portions of the field and checks the patient's range with the

Simple methods.

* As the pupillary reflex sensory fibers run in the tract nearly to the corpora, any lesion anterior means an interrupted reflex arc.

† Unilateral amblyopia suggests either the "amblyopia exanopsia" of existing strabismus or of a pre-existing deviation which has become straightened, or lesions that may produce complete blindness, or affect the angular and supramarginal convolutions.

‡ Minor disturbances are tested by the use of the well-known Snellen's test types.

DESCRIPTION OF PLATE XIV

FIG. 1.—ATROPHY OF THE OPTIC NERVE (POST-PAPILLITIC ATROPHY). ALSO MEDULLARY NERVE FIBERS. RIGHT EYE. DIRECT METHOD.

Fundus Changes.—Disc is bluish, the edges not well defined, lamina cribrosa not present As a coincidence there are medullary nerve fibers present. The whole fundus is mottled (map-like). The retina is atrophied and the macula cannot be distinguished. Arteries are straight and some of the larger veins slightly tortuous.

FIG. 2.—PRIMARY OPTIC ATROPHY. RIGHT EYE. DIRECT METHOD.

Fundus Changes.—Disc bluish and glistening. Membrana cribrosa at center of disc. A cilio-retinal vessel is seen on the temporal edge of disc passing toward the macula. Fundus reflex apparently normal. Vessels are not particularly narrowed at the present time. Arteriosclerosis evident at crossing of vessels. Disc has "saucer"-shaped excavation.

FIG. 3.—RETINO-CHOROIDITIS (SPECIFIC). LEFT EYE. DIRECT METHOD.

Fundus Changes.—Nasal edge of disc hidden and cannot be distinguished from the neighboring retina. Temporal edge of disc is clear and reveals a narrow crescent. A few yellowish-colored spots seen in the choroid. Other spots of choroiditis have become absorbed and white areas (atrophy) have taken their places with irregular pigmentations. The choroidal circulation is exposed in the periphery. A large patch of retino-choroiditis is seen close to the temporal side of the disc.

FIG. 4.—GLAUCOMA. LEFT EYE. DIRECT METHOD.

Fundus Changes.—Edge of disc seen with + 6 D. and the bottom of the cup is seen with a −5 D. Vessels of the retina disappear as they pass into and around the edge of the disc, and are out of focus when they reach the bottom of the cup, where they appear indistinctly at the nasal side. The nerve is bluish or pearly white in color and atrophic (glaucoma atrophy). The edges of the disc have a distinctly yellowish color and the pigment is broken into fine particles. There is a peculiar redness showing at the macula.

The cupping embraces the entire disc.

PLATE XIV

Fig. 1. Atrophy of the Optic Nerve (Post Papillitic Atrophy) Also Medullated Nerve-fibers

Fig. 2. Primary Optic Atrophy

Fig. 3. Retino-Choroiditis

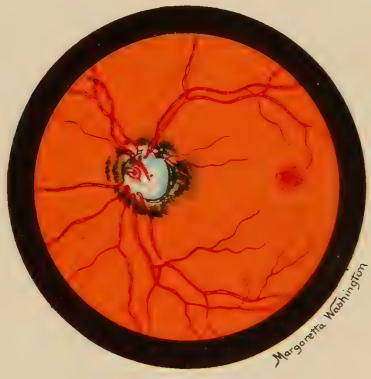


Fig. 4. Glaucoma

(From Thorington's "Ophthalmoscope and How to Use It")

presumptive normal of the physician. For more accurate or graphic delineation the various perimeters may be employed.

Central Scotomata.—*Loss of central vision or central amblyopia if unilateral is usually due to choroiditis or retinitis, if bilateral, to chronic toxemias or syphilis.* Scotomata of color vision are best detected by the perimeter.* In migraine and certain conditions of cerebral meningeal irritation the so-called "flittering" scotomata appear. These are "like a sunset cloud."

Unilateral vs. Bilateral.

Scotomata.

Muscæ Volitantes.—Little floating motes, specks or thread-like figures are common in overstrain, hysteria, anemia, dyspepsia, eye-strain, and various other conditions.

Muscæ volitantes.

Color Fields.—The limits of color vision may be indicated by concentric circles, the inner and least being for green, then red, blue, and finally white. Aside from actual nerve lesions color perception is greatly modified in toxic amblyopias (tobacco, etc.) and the neuroses.

Vary with color.

Modifying conditions.

Optic Neuritis (*"Choked Disc," "Papillitis"*).—This important clinical condition occurs in from 70 to 90 per cent. of all brain tumors, is usually found in meningitis, especially if that be basal and is not infrequently present in cerebral abscess. *In all cerebral lesions it is likely to be bilateral*, though more advanced in one eye than in the other. If in such cases it is unilateral it is on the side of the brain affected. As regards the location of brain tumors in relation to the frequency of optic neuritis, it may be said that it is invariable in tumors involving the corpora quadrigemina, occurs in 90 per cent. of those at the base, parieto-occipital region and cerebellum, and with scarcely less frequency in those of the crura and frontal lobes. In the case of no region does it fail to appear in much less than 50 per cent. of the lesions. Among other causes of optic neuritis are chronic nephritis, diabetes, hydrocephalus, sinus thrombosis, syphilis, lead poisoning, cysts, meningeal hemorrhage and trauma; rarely it is noticed in severe acute infections and sunstroke. In these diseases it does not assume often the high grade (choked disc) encountered in the conditions previously described and in many instances is unilateral.

Extremely important.

Unilateral vs. bilateral.

Brain tumor.

Other associated lesions.

The Wassermann test has shown that *primary* optic neuritis is frequently syphilitic.

OPHTHALMOSCOPY.—Every physician should be able to use the ophthalmoscope at least so far as its findings relate to the common lesions, other than actual refraction errors. It often proves the master key of diagnosis in obscure cerebral lesions, syphilis, tuberculosis, locomotor ataxia and arteriosclerosis, and of prognostic value in diabetes and chronic nephritis. Any good form of ophthalmoscope will suffice, the difference being rather in the skill and cerebration of the physician than in any marked advantages possessed by one over another instrument. The best of all, nevertheless, for

Often the master key.

* A rough test consists in holding before the patient's eyes at a distance of 1½ to 2 feet a black square of cardboard with a central white spot. While his eyes are fixed on the white spot a black strip carrying a green or red spot or wafer at its extremity is placed quickly at the outer side of the white. If not seen, it indicates central color scotoma.

the *practitioner* is the electric ophthalmoscope, simple, compact, portable, yet for illumination of the background almost automatic.

Oblique Illumination.—A beam of light is focused obliquely upon the corneal surface by an indirect examination lens, which is held 2 to 4 inches above and to the side of the affected eye, slightly in front of the patient; a second lens may be used for magnification. *Opacities of the cornea* appear as small (*nebulæ*) or larger (*leucomata*), cloudy or opaque areas. *Deep-seated opacities such as occur oftenest in syphilis yield a reflection, superficial areas are dull.* The presence or absence of the *light reflex* should be noted and the *consensual reflex* tested. *Hippus, photophobia* and the *pupillary contractions of accommodation and convergence, are readily noted and the pupillary outline and its bilateral uniformity observed.* The aqueous humor, iris, lens, and vitreous may be observed if the lens is moved or the eyes of the patient turned as becomes necessary.*

Corneal opacities.

Pupils.

FIG. 618.—"Choked disc." (extreme optic neuritis). Brain tumor. The swelling of the disc exceeds two diopters. (*After Gowers.*)

Iris.—The iris may show inflammation, adhesion (synechiæ), or the coloboma of a past iridectomy.

Adhesions, and scars.

Iritis.—*A delicate pink injection, maximal at the corneal margin and not moving on sliding pressure upon the conjunctiva,* a darkened turgid greenish iris, photophobia, and a sluggish, contracted, sometimes irregular pupil, indicate iritis.

Cardinal symptoms.

Conjunctivitis.—This shows a vivid and tortuous general hyperemia most marked in the *cul de sac,* the vessels being readily moved by sliding pressure. Common in all catarrhal affections and eye strain, it may in severer suppurative forms be gonorrheal, diphtheritic or meningeal, or, if associated with pain, a symptom of corneal ulcer or even glaucoma. *Ecchymoses* occur as the result of traumatism, violent physical strain, asthma, epileptic seizure or in connection with hemophilia, purpura, leukemia, malignant endocarditis or pernicious anemia. *In several cases observed by the author an apparently causeless hemorrhage has preceded for weeks or months a cerebral apoplexy.*

Simple vs. Specific.

Ecchymoses and apoplexy.

Retinoscopy.—Sitting 1 meter from the patient, whose vision should be directed straight ahead, the source of light being slightly higher than the head, a beam from the perforated mirror is thrown upon the pupil. The red reflex appears *if the media are transparent.* The mirror should then be tilted in all directions, the fugitive central reflex produced being carefully noted as it gives an exact clinical indication of the nature of the refraction.†

Light.

Red reflex.

Fugitive reflex, and refraction.

* It is best to dilate the pupil for inspection of the lens margins and anterior layers of the vitreous. (Thorington.)

† If the source of light be vertical (Argand burner, not electric light) and the mirror concave, a straight pupillary shadow moving quickly in a direction opposite the tilting of the mirror indicates either a normal, hypermetropic or less than 1 diopter myopic eye. Movement of the shadow in the same direction as the mirror tilt indicates myopia exceeding

A subtraction of 1 diopter is always made for the 1 meter distance, or a + 1 spherical lens may be placed in a trial frame before the eye, in which case the subtraction need not be made. The eye must be completely under the influence of a mydriatic in doing retinoscopy, if accuracy is desired.

THE FUNDUS OCULI.—Indirect Methods of Ophthalmoscopy.—Proper dilatation having been secured,* the patient with head slightly bent forward sits close to the source of light which is on a level with the ear lobe.† The examiner sits from 1½ to 2 feet distant, the left shoulder advanced, a convex lens (13 diopters) in the left hand, the ophthalmoscope in the right. The perforated center of the larger (concave) mirror should be exactly opposite the eye-hole of the instrument in which a + 3 diopter lens is placed. When a beam of light is thrown (from the mirror) upon the pupil a rosy reflex entirely fills it. The patient's gaze should be fixed upon the examiner's left ear if the left eye is under examination, upon the right little finger-tip as extended while holding the ophthalmoscope if the right eye is under inspection. This reveals the optic disc, and slowly shifting the patient's gaze from right to left and up and down brings the periphery into view and the macula should be seen when the patient looks directly at the center of the examiner's forehead. The large convex lens should be held between finger and thumb at about 2½ to 3 inches from the eye or at such distance as is indicated by its strength and the clearness of the image. It may be steadied by resting the little finger upon the patient's brow.‡ *In indirect ophthalmoscopy the image is inverted, all relations being exactly reversed but the image is large and the method easy.* Astigmatism is indicated by shrinkage or expansion of the image in any *meridian* attending the slow withdrawal of the lens, and in general the *contraction of the field upon withdrawal indicates hypermetropia; its enlargement, myopia,* so that simple, compound, mixed or irregular astigmatism may be thus detected. These conditions are merely suggestive, not accurate.

Dilating pupil.

Position of patient and examiner.

Red reflex.

Observing disc.

Accessory lens.

Gives inverted image.

Refractive errors revealed.

The Direct Method.—The advantages of this method lie in the magnified (cornea and crystalline lens) *direct* image obtained and the better showing of details though a smaller surface is brought into view in any one position. The light should be close to the patient but behind and slightly above the level of the ear corresponding to the eye under examination. The examiner must be able to *slowly* bring his own eye within 2 inches of the one under

Magnified direct image.

Technic.

---

1 diopter and the higher the error the more crescentic and slow moving is the shadow. Astigmatism is evident if the shadow edge moves differently or with different rapidity in opposed meridians. Simple astigmatism is present if there is movement of the shadow in only one meridian, the other being normal; compound astigmatism, if the movement of the shadow is similar in each meridian but of different degree. Mixed astigmatism is evidenced by different directions of the shadows in opposite meridian.

* A drop or two of 2 per cent. homatropin solution, or better, a fresh 4 per cent. cocain or euphthalmin solution is dropped into the eye a half hour or more previously and followed after examination by a drop of eserin solution (gr. ¼ to 1 ounce of water).

† A light directly above the head may be used for this indirect method.

‡ Bed-ridden patients may be examined whether recumbent or sitting up by proper adjustment of the light and correct position on the part of the physician.

examination and the eye used should correspond to the one examined (examiner's right, patient's right). The ophthalmoscope is applied flat to the operator's cheek, the elbow at the side, the small mirror so adjusted as to give maximum reflection, and the patient told to look off into space over the shoulder of the examiner and under no circumstances to look at a near object (relaxation of accommodation). The examiner must relax his own accommodation or if necessary use a -1^{D} or -2^{D} lens* if the patient's refraction is normal. The proper lens must be inserted in the ophthalmoscope when the patient's refractive error does not permit a distinct view of the retinal structures which might lead to serious misinterpretation. If the examiner's refraction is faulty, the proper glasses should always be worn. As the patient must look directly in front of him to bring the macula into view and as the impingement of light upon that point will surely cause pupillary contraction, preliminary dilatation is often necessary.

Relaxing accommodation.

Use of mydriatics.

MOTOR NERVES OF THE EYE.—Motor Oculi (*third*), **Patheticus** (*fourth*), **Abducens** (*sixth*). These three nerves, closely related in origin and course and connected by an elaborate system of association fibers, control the movements of the eye. *The third supplies the ciliary muscle, levator palpebræ superioris, inferior oblique and superior, inferior and internal recti. The fourth supplies the superior oblique, the sixth the external rectus.* The *multiple nuclei of the third* and *that of the fourth* lie beneath the floor of the Sylvian aqueduct, while *that of the abducens* lies lower beneath the floor of the fourth ventricle.

Muscles supplied.

Nuclei.

All are much exposed to pressure in basal lesions, *the third emerging at the inner surfaces of the crura, the sixth between the medulla and the pons*, while *the fourth is the only nerve emerging on the dorsal surface*, springing from the roof of the fourth ventricle immediately after it has *decussated* with its fellow. All three nerves may be affected in *gumma, meningitis, tumor, or hemorrhage*, by *syphilitic* or other forms of neuritis or by direct injury affecting the base, and *one may be affected alone*, the patheticus most rarely. Furthermore, *isolated paralysis of the fourth indicates cerebellar tumor or an exudate on the anterior inferior aspect of the cerebellum.* As the unilateral lesions of the base usually affect cranial nerves of the same side with the exception of the patheticus, while producing hemiplegias of the opposite side, the value of crossed paralysis as a localizing symptom is readily seen. Partial paralysis of motor oculi innervation may result from lesions involving individual nuclei or the roots springing from them and passing through the knee of the internal capsule. *Complete uncomplicated unilateral paralysis of the motor oculi indicates a lesion of its trunk and the effects of orbital and sinus lesions or pressure at the sphenoidal fissure should be held in mind.* Hemiplegia of the opposite side co-existing suggests in the case of the *motor oculi, a mid-brain lesion or a tumor involving pressure upon a crus*, in which latter case the hemiplegia precedes the eye involvement. If the paralyses are coincident and simultanous a lesion above the corpora quadrigemina is suggested.

Often involved in basal lesions.

Isolated paralysis of the 4th.

Crossed paralysis.

Isolated paralysis of motor oculi.

* It is entirely unnecessary and wholly unwise to close one eye in using the ophthalmoscope and the observer should *imagine* that he is looking at a distant object.

TESTS FOR LESIONS OF THE THIRD, FOURTH AND SIXTH NERVES.—These are essentially tests of pupillary activity and muscular strength, the latter being best indicated by diplopia (double vision) inasmuch as marked impairment may exist without strabismus (squint).

PARALYTIC SYMPTOMS.—Motor Oculi.—Paralysis involves abolition of pupillary reflex and fixed moderate dilatation due to unopposed sympathetic influence, the eyeball swings outward and slightly downward (unopposed influence of abducens and patheticus) and any attempt to overcome the *ptosis* present is accompanied by excessive contraction of the occipito-frontalis and a characteristic predominance of forehead wrinkles on the affected side.* Irritant lesions will of course cause contracted pupils and various ocular spasms, such as nystagmus, nictitating or various non-rhythmic movements. Blepharospasm or hippus may replace ptosis.

Pupillary dilatation.

Strabismus and ptosis.

Irritative symptoms.

Strabismus.—*The "cross-eyed" person cannot bring the visual axis of both eyes simultaneously to the same point* and ordinarily the defect is lateral, more rarely vertical. In a *divergent strabismus* the defective eye swings outward, in *convergent squint* inward. If the squint be spasmodic (*concomitant*) it is always made evident by asking the patient to look straight ahead; furthermore, the eye moves in any direction in direct ratio to the movement of the sound eye, though retaining its lack of parallelism, and diplopia is usually absent. *Paralytic strabismus*, on the other hand, shows either total loss of movement in the affected muscles or marked weakness, which, however, may be apparent only when the patient looks in a certain direction, or under continued fixation of the gaze upon one object in the weak axis.

Divergent vs. Convergent.

Concomitant.

Paralytic.

The abducens, *supplying the external oblique muscle is the nerve oftenest affected.*

Diplopia.—In double vision the image of an object is received on the macula lutea of the fixing eye while in the other eye it strikes some other portion of the retina. *Double vision occurs only in paralytic squint except when developed by special tests.* It is a frequent symptom of the toxic irritation produced by certain drugs such as alcohol and belladonna or of any irritative or destructive lesion at the base of the brain. Having the patient follow with his eyes the finger or pencil moved laterally and vertically before them and noting their convergence as it is brought gradually close to his nose measures roughly the extent and indicates the specific muscles affected. He should be asked to tell the number of fingers held before him in each position, no rotation of the head being permitted. To find the eye and

Causes.

Simple test.

* Firm downward pressure over the brow by cutting off the action of the frontalis enables one to estimate the actual degree of ptosis. Ptosis or pupillary signs without other symptoms of oculo-motor involvement are not infrequently seen in locomotor ataxia, as in a case under the author's observation for a long time. Ptosis may be incomplete and is simulated by (*a*) hysterical orbicularis spasm, (*b*) a transient ptosis upon waking seen in neurotic or overworked persons, (*c*) or by paralysis of the Müllerian fibers of the sympathetic in the orbital tissue, a paralysis which differs from true ptosis in the accompanying pupillary contraction and the symptoms of inflammation and edema of the corresponding lower lid. Furthermore, the inhibiting action of the association fibers of the trigeminus (fifth) may cause ptosis in trigeminal lesions.

muscle producing double vision requires in many instances a resort to special tests.*

Looking "toward" or "from" the lesion.

Conjugate Deviation.—Due either to a tonic contraction of the associated muscles which rotate both eyeballs to the same side or to a paralysis of the antagonists. If both eyes deviate to the same side they are said to "look toward" the lesion, if it be paralytic, and "away from it" if the condition be spasmodic (irritation), but in lesions of the pons the reverse is true.

Sixth Nerve.—Abducens.—This has the longest course of any cranial nerve and is frequently involved in basal lesions, *producing internal strabismus.* Its associations in disease are essentially the same as those of the third nerve. It supplies the external rectus.

TRIFACIAL.—The fifth consists of two roots, one sensory and one motor. The sensory root bearing the Gasserian ganglion divides below the ganglion into three divisions, the third of which, the inferior maxillary, is joined by the fibers from the motor root.

Hence the ophthalmic and superior maxillary divisions of the trigeminus are sensory; the inferior maxillary, both sensory and motor.

Lesions of the fifth nerve produce the following symptoms:

Ophthalmic Division.—Anesthesia of the conjunctiva, skin of brow and nasal bridge, the upper lid, forehead and anterior portion of scalp.

Taste affected.

Supramaxillary.—Anesthesia of the alæ nasi, cheeks, temples and nasal mucous membrane, upper pharynx, tonsils, soft palate, roof of mouth and upper teeth, upper lip, diminished taste and lost palate.

Inferior Maxillary.—Taste impaired in anterior portion of tongue, salivary flow checked; anesthesia of the mucous membrane of the mouth, lower part

Neuralgia.

of the face and lower lip, trifacial neuralgia, paresthesia, and, *as it receives the motor root,* trismus or paralysis of the muscles of mastication. Unless the paralysis be bilateral little actual disability occurs but in complete dis-

Trismus or jaw-drop.

ability the jaw drops and even swallowing is difficult. Dryness of the nasal, buccal and conjunctival mucous membrane of course follows a lesion of the fifth and partial deafness may result.

Substances used for tests.

Taste.—This sense is tested by placing upon the tongue successively, sugar, salt, quinin and tartaric acid, the tongue being protruded continuously throughout the procedure and the anterior and posterior portions separately tested. *Paresthesia* (taste perversion) may be evident or admitted.

Jaw deviation.

Motor Action.—The deviation of the jaw is toward a unilateral lesion, and when the teeth are clenched the affected muscle fails to contract forcibly

Tensor tympani.

under the palpating finger placed over the masseters. The perception of muscle notes of different pitch tests the field of hearing.

* *The Candle Test.*—In the usual test-spectacle-frame of the test-case place in front of one eye a red lens. Standing several meters away move about a lighted candle until two images (one red, one plain) are reported by the patient who also states where the colored image lies with relation to the other. Then the colored image represents the visual axis of the covered eye, the plain one that of the uncovered. If now by moving the candle in the direction of the colored image the double vision increases, it proves the eye covered by the red glass to be affected and the weak muscle is that which turns the eye in the direction of the false image.

Miscellaneous Symptoms.—*Loss of the corneal reflex* is an early and important sign. No sneezing follows tickling the nose, irritating substances do not affect the tongue or nostrils, a cup placed to the lips feels as if broken, the patient may support the jaw with the hand to aid mastication and swallowing, and atrophy and loss of reactions may follow the paralysis. Herpes zoster, dental decay and ulceration of the nasal, buccal, and corneal surfaces may occur as expressions of lost trophic function.

Nasal and labial.

Masseters.

Trophic changes.

Trismus is the result of infective tetanus or general convulsions, a convulsive tic occasionally results from reflex irritation. Tonic spasm also occurs, rarely also in hysteria, and after long-continued and enforced stretching of the muscles. *Clonic spasms*, slow or fast, are seen in the fear, chill and "teeth grinding" so common in children and indicative of overwrought nerves, worms, gastro-intestinal disorders, adenoids, impacted cerumen, etc. The trifacial, while frequently the seat of neuralgia, is well protected from ordinary injuries and pressure; but may, nevertheless, be involved in certain bulbar palsies and in focal and gross lesions at the base.

Spasm.

Clonic spasms.

Neuralgia.

THE SEVENTH NERVE.—The Facial.—*An accurate knowledge of the origin and connections of the facial nerve is of the utmost importance to the clinician. It is almost purely motor and essentially the nerve of facial expression.*

Origin and course.

The cortical fibers decussate in the raphé of the tegmentum and enter nuclei at the junction of the pons and medulla, external to that of the sixth nerve.

From its origin in the pons nucleus it sweeps about the nucleus of the sixth, which lies just external, and *emerges* with the eighth nerve *between the restiform bodies and the olive* at which point *any lesion must include both nerves* (*seventh and eighth*) as they run together into the internal auditory meatus. In the Fallopian aqueduct it runs with its branch to the stapedius and the fibers of the chorda tympani and, emerging from the stylo-mastoid foramen it passes forward in the substance of the parotid gland, dividing behind the ramus of the lower jaw into branches which supply the superficial muscles of the face and upper part of the neck. Though its *cortical center* lies below the motor areas of both leg and arm (*inferior portion of the anterior frontal convolution*) it lies between the two in the internal capsule, crus, and pons.

Cortical center.

Facial Spasm and Paralysis.—*It is evident that a lesion may be either supranuclear, nuclear or infranuclear, and that the last may be (a) within the aqueduct, (b) external.*

Three varieties.

In supranuclear paralysis, usually associated with hemiplegia, the orbicularis palpebrarum is not affected, R. D. is absent and the paralysis is on the same side as that of the leg or arm muscles, save in lower pontine lesions.

Supranuclear.

Nuclear paralysis is essentially that of the infranuclear type, but seldom exists alone. *A lesion within the aqueduct* involves the chorda tympani and the stapedius branch, *hence there is a loss of taste over the anterior two-thirds* of the tongue, and often *hyperacusis* (exaggerated sound sensation).

Nuclear and infranuclear.

General irritative symptoms may be present, such as muscle spasm, blepharospasm and convulsive tic.

Usual type.

Paralysis of such muscles as control the expression results. Facial expression lines are obliterated and the victim cannot whistle, draw the corners of the mouth outward, close the watering eyes tightly or wrinkle the forehead of the affected side.

A lesion involving the nerve at its point of emergence involves the auditory nerve, hence is associated with the various symptoms of irritation or paralysis of that structure. If the fibers are involved between the nuclei and their decussation (*lower pontine lesions*), *crossed* paralysis results.

Bilateral palsy is rare as an independent lesion. Monoplegia is the type and in hemiplegia with alternating facial paralysis we deal with the nuclear form.

AUDITORY NERVE.—The dorsal root contains the auditory fibers, the ventral those of equilibration, and passing out on either side of the restiform body, they join and within the internal meatus pass to the cochlea for audition and to the vestibule and semicircular canal for equilibration.

Audition and equilibration.

The cortical center for hearing lies in the opposite temporal lobe, that of *equilibrium* in the cerebellum.

Cochlea.

Symptoms.—*Deafness and paresthesia are present alone if the cochlea only be involved. Vertigo, vomiting, tinnitus aurium, sibilant or roaring sounds* ("escaping steam"), with *disturbance of equilibrium,* are present in varying degrees if the labyrinthine branch be affected (*Menière's syndrome*).

Tinnitus aurium.

That common and ordinarily trivial symptom, *tinnitus aurium,* like deafness, may be due to the many common causes, such as catarrh of the middle ear and Eustachian tube, or psychasthenia, inflammation or impacted cerumen in the external meatus, full doses of salicylate or quinin, or, to anemia, digestive disorders and various toxemias, tympanites, meningitis, syphilis, or even brain tumor. *Tinnitus cerebri, i.e.,* the similar sound seeming to arise in the brain itself, is frequent in arteriosclerosis and is also found in meningeal cases.

"Nerve deafness."

Deafness.—"*Nerve deafness,*" *i.e., that arising from cortical, nuclear, or peripheral nerve lesions, is rare; that due to catarrh of the middle ear and impacted cerumen common.*

Tests.—Hearing is ordinarily tested roughly by the watch which should be held at varying distances from the ear and the range of audition of the patient compared with the known normal for the particular watch used. If deafness is evident the tuning fork should be employed to test by bone conduction the integrity of the cochlear nerve and centers.* If the vibrating fork or watch is best heard when placed directly upon the mastoid the *nerve* is not at fault. In most instances *sudden deafness* is due to impacted cerumen, hysteria, or syphilis of the internal ear. As common causes of deafness, nasopharyngeal catarrh, usually associated with Eustachian involvement, adenoids and polypi must be remembered as well as growths in the auditory

Sudden deafness.

Ordinary form.

* *Rinné's Test.*—Set tuning fork in vibration, place handle against mastoid or zygoma. When the sound becomes inaudible hold the still vibrating fork close to the meatus. Normally, the sound should still be heard at the meatus; abnormally, bone conduction is the greater.

meatus. In headache, mental strain or fatigue, or pronounced mental irritability, sounds may be exaggerated (*hyperacusis*) or actually painful. Hyperacusis.

Though rarely bilateral, cortical lesions involving the auditory center may cause deafness. Such deafness is usually progressive and affects one ear chiefly in most instances. The direct cause is usually an irritation of the vestibular nerve but its basic etiology is still unknown.

A similar condition may result from gross disease of the ear or attend or follow syringing or unduly inflating the ear drum, and a lesion of the left cortical center may cause word deafness, *i.e.*, inability to interpret the sounds heard. Word deafness.

Tumor, hemorrhage, meningitis, abscess and syphilis may produce deafness by involvement of the internal capsule or corpora quadrigemina.

The nerve itself may be injured or compressed by tumors and inflammatory or hemorrhagic exudates, or be involved in diphtheritic paralysis, locomotor ataxia, and traumatism. Deaf mutism is not an unusual sequence of cerebro-spinal meningitis. Deaf mutism.

Certain forms of deafness, usually transient, follow the administration of quinin and the salicylates, or arise from violent concussion or constant and repeated lesser sounds (occupational deafness). Transient deafness.

In hysteria decided or complete deafness may come on suddenly, is manifested chiefly in defective range for notes of varying pitch and shows relatively equal loss of both aërial and bone conduction. It may as suddenly disappear and usually follows emotional shock. Hysteria.

MÈNIÉRE'S DISEASE.—(*"Labyrinthine Vertigo"*).—*This combines progressive nerve deafness with sudden attacks of vertigo, lasting but a few moments or for several hours, persistent and exaggerated tinnitus aurium (hissing, buzzing, throbbing, roaring or loud reports), and, sometimes, a peculiar incoördinate jerking of the eye ("nystagmoid movements") and head muscles.*

Nausea, extreme pallor, vomiting, and, in rare instances, even transient unconsciousness or temporary collapse may accompany the attacks which may occur several times daily or be separated by months of immunity. Simple vertigo is usually present more or less persistently in these cases.

*Labyrinthine disease is usually a primary disease of the labyrinth or secondary to middle ear suppuration and necrosis and mastoid disease.**

GLOSSO-PHARYNGEAL NERVE.—The ninth nerve is seldom paralyzed alone and a lesion is recognized only through its sensory disturbances, through its motor for the stylo-pharyngeus and middle pharyngeal constrictors. The sensory fibers run to the brain with the fifth nerve and a loss of taste over the posterior surface of the tongue, and absence of the pharyngeal reflex are the only sources of information. Root involvement does not affect taste on account of the peculiar course of the sensory fibers. Seldom paralyzed alone.

THE VAGUS.—*The tenth nerve has most interesting and complicated functions. It is motor for the pharynx, larynx, and soft palate and both motor and* A marvelous nerve.

* The exquisitely delicate and difficult tests of Bárány must be sought in the special monographs.

sensory for the heart, respiratory passages and most of the abdominal viscera, in connection with which the sympathetic plays an important part.

Spinal accessory association.

Its motor fibers, including the cardio-inhibitory group, are contained in what was formerly regarded as the bulbar portion of the spinal accessory which joins it outside the cranium. The pharyngeal plexus is formed by the vagus and glosso-pharyngeal nerves and supplies the pharyngeal muscles and mucosa, hence a lesion of the nerve nuclei or trunk gives rise to marked dysphagia.

Dysphagia.

Laryngeal supply.

The Laryngeal Nerves.—The superior and inferior laryngeal nerves supply the musculature and mucosa of the larynx. *The recurrent laryngeal* which supplies the mucosa of the lower portion and all muscles except the crico-thyroid, is especially liable to pressure paralysis by reason of its relation to mediastinal structures, aneurysm being one of the commoner causes of paralysis of the left recurrent. Bilateral paralysis of the abductors causes imperfect approximation and separation of the vocal cords in respiration and voice production. The symptoms are inspiratory stridor and whispering voice without aphonia, the cords being nearly approximated. It is encountered in cases of hysteria and occasionally follows a simple laryngitis though usually toxic. Pressure upon one recurrent nerve, usually aneurysmal or due to pleural adhesion, produces unilateral abductor paralysis, immobility of the affected cord in inspiration and hoarseness. *Adductor paralysis is usually hysterical and the separation of the cords produces aphonia.*

Chiefly recurrent branch.

Common types.

Laryngeal Spasm.—The croup of children, locomotor ataxia (*laryngeal crises*) and hysteria are the commonest sources; the last form being chronic and usually associated with speech production.

Laryngeal Anesthesia.—This, due to diphtheria, neuritis, bulbar paralysis, or hysteria, makes feeding difficult and inhalation-pneumonia common.

Laryngeal Hyperesthesia.—This is rare, but occasionally seen, especially in sexual psychasthenia.

As affecting heart action.

Cardiac Plexus.—Formed by the vagi and sympathetic nerves, its branches control the activity of the heart. Stimulation of the vagus slows or temporarily arrests the heart action, and in rare instances the power of voluntary self-inhibition of the heart beat has been observed. In complete paralysis the accelerator fibers act unhindered causing rapid heart action. Palpitation and obtrusive auto-audible cardiac heart action are probably appreciated through the vagus path.

Gastric Branches.—The vagi control the activity of both stomach and esophagus, conveying sensory impressions as well as motor stimuli and are probably concerned in some degree with so-called nervous dyspepsia and the gastric crises of locomotor ataxia. In bulbar paralysis the nuclei of the accessory nerve and the hypoglossal are involved with those of the vagus.

Bulbar paralysis.

SPINAL ACCESSORY.—*The chief function of the eleventh nerve is the supply of the sterno-mastoid and trapezius muscles, and, through the vagus, the pharynx and larynx.*

Sterno-mastoid and trapezius.

Tests.—Laryngeal paralysis has already been covered. Paralysis of the sterno-mastoid prevents wholly or in part rotation of the chin toward the

sound side, and if the trapezius be paralyzed, shrugging of the shoulders is interfered with.

Palatal paralysis.

Complete paralysis of the palate is indicated by regurgitation of fluids through the nose in swallowing and thick nasal speech. If the palate be examined while the patient says "eh" and "ah," defective upward movement is perceived. If the lesion is unilateral, immobility of one side with dragging of the median raphé toward the sound side will be manifest.

Hypoglossal Nerve.—The twelfth nerve is entirely motor and supplies the tongue and depressors of the hyoid bone. It rises from a nucleus nearly median in the lower portion of the floor of the fourth ventricle and its cortical center lies in the lower frontal convolution.

Tests.—Note evidence of any wasting, tremor, or fibrillary contraction. Wasting indicates a nuclear or trunk lesion; its absence, a supranuclear or cortical lesion. Deflection of the tongue toward its paralyzed side is evident on protrusion (see also "The Tongue" under "Segmental Paralyses").

Cortical vs. peripheral palsy

THE SYMPATHETIC NERVOUS SYSTEM.—The anterior horns of the cord carry fibers of unknown cortical origin to the sympathetic ganglia through a special set of axons running in the anterior nerve roots, and sympathetic ganglion cells, with their axons, in turn constitute the motor sympathetic neuron.

A similar set of fibers (sensory) traverse the posterior roots and there is a free communication between the cranial nerves and the sympathetic system. The vasomotor fibers are especially plentiful in the anterior roots of the dorsal region. The visceral fibers are chiefly in the cervical region where they accompany the accessory and vagus nerves and ultimately pass to the alimentary canal.

The remarkable structure of the so-called "*chromaffin system*" is considered elsewhere under the diseases related to the internal secretions.

The wonderful automatic control exercised by these nerves cannot be dealt with here, but though their activity may continue when all central nervous connections are cut, they are to a certain extent governed and regulated by the higher centers.

The cilio-spinal fibers are important in diagnosis, irritation causing pupillary dilatation; section, contraction. Other symptoms of sympathetic origin are: vasomotor spasm or relaxation, abnormal sweating in the involved areas and various disturbances of glandular secretion and cardiac and visceral activity.

DISEASES OF THE BRAIN AND SPINAL CORD

HEMORRHAGIC PACHYMENINGITIS (*"Internal Meningeal Hematoma"*).—This may be encountered in either the brain or the cord. *The cerebral form* is almost exclusively confined to the insane (*especially paralytic dements*, 20 per cent.), and the aged (90 per cent.), occurs in those over fifty years of age, but is occasionally encountered in vascular degeneration, profound anemias, alcoholism, tuberculosis, syphilis, heart disease, cachexias, diseases of the purpuric type and even in acute infections.

Chiefly in the insane, alcoholic and aged.

It may occur in infants as a result of forceps injury, or contracted pelvis in the mother, and, in rare instances, may complicate chronic Bright's disease, the acute infectious diseases, pernicious anemia, scurvy or hemophilia.

Three forms.

Pathologically three forms of pachymeningitis may be seen, (a) simple subdural hemorrhagic exudate, (b) an inflammatory exudate converted into a delicate vascular membrane by granular tissue and vascular extension, (c) the two conditions combined. In the insane and aged it is supposed that the cerebral atrophy and arteriosclerosis so frequently encountered are contributive factors.

Pressure symptoms.

Symptoms of the Cerebral Form.—If any exist they express intracranial pressure, headache, choked disc, slow and irregular pulse, recurrent convulsions, coma, and a paralysis, usually but not invariably unilateral, and often associated with aphasia. Fever and albuminuria may be present.

The pupils are sluggish, that opposite the lesion being dilated usually in the earlier stages. Nystagmus, conjugate deviation of the eyes and head, and forced attitudes may or may not be present. Recurrences are frequent and complete recovery is rare.

Symptoms of Spinal Form.—This may involve any portion of the dura mater of the cord (most commonly the cervical enlargement) or be accompanied or preceded by cerebral hematoma.

Irritative pressure symptoms occur accompanied by secondary degeneration and producing *intense peripheral neuralgic pain* chiefly brachial and cervical with hyperesthesia and paresthesia, slowly progressive muscular weakness, paralysis and muscle atrophy.

Chronic course.

Prognosis.—The disease is chronic, usually fatal within two years, and, during the later months, is often associated with *spastic paraplegia* of the lower extremities.

Differential Diagnosis.—It cannot be distinguished from *tumors of the cord. Amyotrophic lateral sclerosis* lacks the severer neuralgic pain, and *syringomyelia* may be differentiated by its characteristic sensory symptoms.

EXTERNAL PACHYMENINGITIS.—*Both the cerebral and spinal forms yield the symptoms of compression and result ordinarily from traumatism, caries, or the extension of some suppurative process. It is most frequently secondary to syphilis and tuberculosis, tumors, or suppuration, involving the adjacent structures.*

Pseudo-membranous and purulent pachymeningitis are not worthy of detailed consideration.

INTRAMENINGEAL HEMORRHAGE.—This is more often a complication of acute febrile toxemia, convulsive seizures and the rupture of basilar vertebral aneurysms, less often it results from traumatism.

Symptoms.—These are usually *localized pressure symptoms* of sudden development, and cases secondary to acute febrile toxemia and traumatism furnish the most favorable prognosis.

Extrameningeal Hemorrhage.—Aneurysmal rupture or traumatism involving the peridural plexus may cause hemorrhage usually with pressure symptoms.

HEMATOMYELIA.—Hemorrhage into the cord itself, *usually associated with hematorrhachis,* may result from the causes of intrameningeal hemorrhage, but is more often due to *direct injury* involving fracture or dislocation of the vertebræ, and is *undoubtedly one of the causes of the disturbances noted after injuries involving violent flexion or torsion of the spine.*

Hematorrhachis.

Usually directly follows injury.

The hemorrhage varies greatly in extent and position and the paralysis may be paraplegic or unequally affect the two sides, but is frequently unilateral, producing the well-known Brown-Séquard symptom-complex, viz., unilateral motor paralysis with anesthesia of the opposite side.

Pain is usually slight or absent, *paresthesias* common and the lumbar or cervical regions most frequently affected. *The onset is sudden,* and in low-seated lesions urinary retention or incontinence is often observed.

Sudden onset valuable sign.

Prognosis.—Partial recovery is the rule and a fatal termination the exception, both primary and residual paralysis are chiefly of the spastic type.

CAISSON DISEASE (*"Divers' Paralysis"*).—This obscure ailment is encountered almost exclusively in caisson workers and divers subjected to extreme and sometimes sudden changes of atmospheric pressure, and, in submarine workers similarly exposed. The changes seem to be strictly limited to the cord, taking the form chiefly of punctate hemorrhage, myelitis or laceration.

Decompression the factor.

It is believed to be due to nitrogen saturation and its sudden release, which gives rise to *gas emboli,* for the most part affecting the spinal cord, but also occurring in the liver, spleen, lungs and even the superficial tissues.

Symptoms.—Immediately after leaving the caisson or perhaps not for forty-eight or seventy-two hours a series of symptoms develops, varying in severity from excruciating pain in the lower extremities to spastic paraplegia with anesthesia and urinary retention. Coma is sometimes observed and sudden death or *exitus* after a few hours exceptionally occurs.

The upper extremities are seldom affected and many minor symptoms such as temporary albuminuria, vertigo, deafness and *tinnitus aurium* may be encountered. The disease is rarely fatal and the symptoms ordinarily disappear in a few weeks or days.

Transient.

"MILIARY," "DIFFUSE," AND "TUBERCULOUS," SCLEROSIS.—This may be dismissed as of little practical importance, all three varieties being associated with mental disease and seldom encountered save in the autopsy rooms of asylums.

MULTIPLE SCLEROSIS (*"Insular" or "Disseminated" sclerosis*).—*This is a disease of unknown causation,* but usually follows an acute infection and occurs chiefly in young male adults* (90 *per cent. under the thirtieth year*).

Male adults chiefly.

It is characterized anatomically by an extensive, irregular dissemination of reddish-gray areas of sclerosis *involving both the gray and white substance of all portions of the cerebro-spinal tract,* the white being chiefly affected.

The cerebral lesions ordinarily predominate and, in the cord, the cervical region and the *cauda equina* are usually chiefly affected. In the former

* In nearly half of a large series recently reported a positive Wassermann was present.

the brain stem and the basal ganglia suffer most, the cortex being but slightly involved and the cerebellum sometimes escaping.

Indeterminate symptoms.

The disease is remarkable for its lack of ascending or descending degenerative changes and the inconclusiveness of its symptoms, both facts being explained by the extraordinary persistence of the axis cylinders in the tissues involved.

Characteristic complex.

Diagnosis.—*Intention tremor, lessened in recumbency, nystagmus, scanning speech, muscular weakness and transient palsies, are the important symptoms.*

Varied findings.

Vertigo, optic atrophy (temporal side), spastic weakness of the lower extremities and a stiff awkward gait are common. Enfeebled mentality and sudden attacks of coma or of epileptoid convulsions may occur and the symptoms may be extraordinarily varied and both slow and insidious in development. The sphincters may be affected in the terminal stage.

Differential Points.—Paralysis agitans lacks nystagmus and shows its tremor during rest. Hysteria lacks nystagmus and usually presents distinctive stigmata.

No concrete picture.

It is obvious from the pathology that no concrete picture is possible and the very multiplicity and variability of its symptoms is diagnostically helpful.

A mere slowing up of movements, paresis of universal distribution, muscular rigidity and spasticity make a striking melange—aside from scanning speech (60 *per cent.*), *intention tremor* (75 *to* 80 *per cent.*) *and nystagmus* (50 *to* 60 *per cent.*).

Bulbar symptoms such as anarthria and dysphagia may be present in rare instances and causeless laughing or crying may strongly suggest hysteria in some cases.

The eye backgrounds should be examined carefully for optic nerve changes. Partial or, more rarely, complete, temporal atrophy or neuritis and central scotoma may be present.

Marked chronicity.

Prognosis.—It is markedly chronic and terminates by intercurrent disease, usually after the patient has become bed-ridden.

Septic phlebitis usually.

SINUS THROMBOSIS.—This may arise in the course of wasting diseases or even a chlorosis but usually represents a septic phlebitis resulting from disease of the adjacent structures.

Lateral Sinus Thrombosis.—This usually results from suppurative disease of the middle ear and mastoid cells.

If pyemic symptoms developed during a middle ear and mastoid suppuration persist after apparently successful operation, or arise following such a surgical procedure, suspicion amounting almost to certainty results.

If tenderness, swelling and persistent emptiness, of the jugular of the affected side, is present the diagnosis is practically certain and further surgical procedure is demanded.

Cavernous Sinus Thrombosis.—This may follow suppuration, necrosis or operation, within the nasal passages or orbital tissues.

The most striking symptoms are orbital and conjunctival edema with unilateral exophthalmos and septicemic or pyemic symptoms.

Longitudinal Sinus Thrombosis.—This condition occurs usually in connection with a pachymeningitis and is seldom recognized ante-mortem.

Epistaxis and orbital and conjunctival edema are suggestive symptoms.

Reduced in importance.

CEREBRAL CONGESTION.—*Cerebral congestion has lost its place as an important clinical entity, most of the symptoms formerly classed under that head being due to toxemia rather than mere congestion and now referred to underlying disease to which congestion is purely secondary.*

Mental excitement, indeed any mental effort, means temporary congestion and certain drugs such as alcohol, quinin and strychnia undoubtedly produce it.

The arterial hypertension of nephritis may be associated with evidences of active cerebral congestion so plainly foreshadowing an apoplexy as to be of clinical interest and prophylactic value.

Passive congestion cannot be distinguished from relative cerebral anemia, according to the author's experience.

Familiar symptoms.

The symptoms of active congestion are headache of varying degree, often associated with a subjective sensation of pressure and throbbing, chiefly frontal but often occipital or temporal, vertigo, tinnitus aurium, mental irritability and impaired concentration.

CEREBRAL ANEMIA.—Relative cerebral anemia may be (*a*) simply a part of general anemia; (*b*) due to temporary vasomotor changes; (*c*) the actual loss of or impoverishment of the blood; (*d*) arteriosclerosis; (*e*) myocardial weakness, with or without valvular lesions.

Symptoms common to all types.

Nausea, vertigo, and faintness or actual syncope, mental depression, impaired concentration, drowsiness during the day and particularly after meals, frequent dull or severe headache, disturbed sleep, early waking or actual insomnia, are some of the chief symptoms, but there is no sharp line of division between the various groups, and the complete picture is seldom present.

Unrefreshing sleep and bad dreams are more or less common manifestations and all of those mentioned assume special importance as relatively early signs of cardiovascular inadequacy or decompensation.

Insomnia.

The headaches associated with marked general anemia are usually dull but may assume a very violent type. *The insomnia* usually comes on after a period of sleep, the patient lying awake the balance of the night or merely catching short naps. The early morning awakening, bad dreams and unrefreshing sleep, usually associated with advanced age is not uncommon in the cardiac decompensations of younger persons.

CEREBRAL EDEMA.—This condition is chiefly of interest in connection with the uremia of Bright's disease, the unilateral paralysis or spasm sometimes observed therein being due, possibly, to a localized cerebral edema.

Presumptive only ante-mortem.

Congestive edema may be a part of general edema, especially that of nephritis or a local congestion due to obstruction, cerebral growths or abscesses. The "wet brain" is considered under alcoholic meningitis.

CONGENITAL HYDROCEPHALUS.—These cases cause much difficulty in labors and the children usually die within the first five years. Rarely, even somewhat extreme degrees of change do not prevent full mentality and reasonably long life; but ordinarily, children are backward, mentally

* In some instances the dominant symptoms are of cerebellar origin.

defective, and subject to spastic weakness of the lower extremities and perhaps convulsive seizures.

HYDROCEPHALUS IN ADULTS.—This is ordinarily secondary to tumor though possibly in rare instances primary. There may or may not be enlargement of the skull through separation of the sutures, the symptoms are variable and the disease is not positively diagnosticable.

During life the most common symptoms are headache, recurring attacks of coma, optic neuritis and occasionally, ataxia.

TUMORS OF THE BRAIN.—About one-half of the tumors of the brain are tuberculous, and of these 80 per cent. occur under the age of twenty-one, whereas cysts and syphilitic tumors occur almost exclusively in adults.

Sarcomata, usually primary constitute about 20 per cent. of which 70 per cent. occur in adults; carcinomata of the softer sort and usually secondary 70 per cent., of which 75 per cent. occur in adults; gliomata 15 per cent., of which 60 per cent. are in adults; gliosarcoma 5 per cent., of which 80 per cent. are in adults.

Tuberculosis, rarely primary, is usually secondary to some preëxistent lesion and basal in location.

Syphilitic tumors, always acquired and seldom developed later than three years after the primary infection, constitute but 3.7 per cent., 99 *per cent. of which are in adults.*

*These show a predilection for the arteries, meninges and ocular nerves, are usually small, may be multiple, and tend to extend to the adjacent cortex.**

Cysts are ordinarily the result of hemorrhage or softening, rarely parasitic.

Gliomata and neurogliomata may be dense, but more often are soft or myxomatous and the seat of slight reactive inflammation or perhaps cystic changes; they occur most often in the substance of the cerebrum, cerebellum or brain stem.

Osteomata, fibromata and lipomata are rare.†

This tumor exists only in the nervous system; is essentially infiltrating in type, remaining throughout limited to the tissue in which it originates and varies greatly in size and extent.

It is peculiarly vascular and hemorrhages are easily induced.

The color is usually gray or reddish-gray but may be exactly that of the main tissue.

Symptoms.—*The most suggestive symptoms are: (a) Persistent or paroxysmal, often violent, headache, usually at its maximum in the early morning. (b) Apparently causeless vomiting without antecedent nausea, and oftentimes without relation to meals, most constant in children. (c) Optic neuritis, gen-*

* Starr states that gummata are not only rare in children but are never inherited and may appear within one year after infection or as late as twenty. "Organic Nervous Diseases," 1903, p. 592.

† Starr's table shows that of 600 cases of brain tumor, 193 were tuberculous (152 in children), 120 were sarcomatous (86 in adults), 91 were gliomata (54 in adults), 30 were gliosarcomatous (25 in children), 32 were cysts (adults 30), 41 carcinomatous (31 adults), 22 were syphilitic (20 adults), 71 miscellaneous. Of all these the sarcomata most rapidly invade the adjacent tissues.

erally double. (d) Mental changes (usually apathy). (e) Vertigo. (f) General or slight convulsions or epileptiform seizures closely resembling grand mal, petit mal, Jacksonian epilepsy, or even showing the psychic equivalents. (g) Pressure symptoms, irritative or destructive. (h) Syncopal attacks. (i) Emaciation. (j) Polyuria. (k) Insomnia. (l) Slowed pulse.

Large tumors in important locations may produce no characteristic localizing symptoms whatever, or, the order and type of the symptoms may be suggestive or definitive. Unlike hemorrhage they are usually of gradual onset and progressive in type though inflammatory reaction may intensify them and periods of intermission are common.

TUMOR IDENTIFICATION.—Tuberculosis.—*Three-fourths of all cases occur in persons under twenty, 50 per cent. in those under ten years of age. One finds usually a personal or family history of tuberculosis, predominance of basal signs, or, if the lesion is cortical, "irritative" rather than "destructive" symptoms. Retinal tubercle is both rare and late in appearing. Early progressive symptoms followed by prolonged intermission and pontine or cerebellar localization are common. Single and multiple lesions are about equally balanced. By fusion they may attain the size of an orange, but such large growths are unusual. Their effects are chiefly those of thrombus formation and pressure, for they do not invade the brain tissue itself.*

Syphilis.—Syphilis of the brain is almost without exception a disease of adult life. There is usually a curious indefiniteness, shifting quality, and lack of completeness in the symptoms present. The chief diagnostic factors are the following: (*a*) *History or symptoms of acquired, never a congenital, syphilitic lesion, usually of not more than three years' standing.* (*b*) *Cortical localization and irritative lesions are the rule.* (*c*) *Rapid advance is often followed by temporary arrest.* (*d*) *Headache may be extreme and is usually worse at night.* (*e*) *Disappearance or striking betterment under antisyphilitic medication.** (*f*) *Positive luetin or Wassermann test.*

Varied Forms.—One must consider the isolated gumma, which yield tumor symptoms, meningoencephalitis, or gummatous leptomeningitis, with or without decided symptoms of high intracranial pressure and, with especial frequency an involvement of cranial nerves oculo-motor, auditory etc.

Paretic dementia and tubo-paresis are syphilitic and the various conditions are dealt with separately in this section. See also "Cardiovascular Syphilis.)

It should be remembered that the Wassermann reaction is much more constant in these cases if the spinal fluid is used in sufficient quantity (1.0 c.c.) than is the case if the blood is employed. Increased globulin (actual turbidity) and a decided lymphocytosis are present in cerebrospinal lues.

It should be remembered that normally the cerebrospinal fluid is poor in cells.

The average for ten fields with a magnification of 300 diameters should not exceed eight cells in normal fluid.

If desired a special counting chamber may be obtained (Fuchs-Rosenthal)

* It should always be tried in doubtful lesions.

or, more conveniently the simple white blood cell counter and pipette may be employed. More than eight or ten cells to the cubic millimeter of fluid represents a pathologic finding. As stated elsewhere, it is not certain that the complicated "*colloidal gold test*" is of any decided clinical value.

Glioma.—There is usually *sudden coma* followed by accession of symptoms, irritative cortical phenomena. Long periods of latent and symptomless development are not uncommon.

Sarcoma.—*The existence of a primary growth, and the rapid, progressive and diffuse symptoms are suggestive.*

Carcinoma.—The patients are usually over fifty and a primary growth is present. *Local tenderness may be present rarely in cortical tumors.*

Terminal Symptoms of Tumor.—Rapid pulse, dementia, persistent continuous vomiting and Cheyne-Stokes respiration are sometimes present, stupor and coma the common phenomena.

The symptoms indicative of increased cranial pressure should be held clearly in mind.

These include headache, optic neuritis, vomiting, vertigo, convulsions and more or less marked and profound mental disturbance.

Such are not peculiar to brain tumor of course nor need they all be present in the given case.

Optic neuritis is the most constant sign in brain tumor, vomiting is a common event and headache rarely fails to appear at some time. Convulsions arise in a considerable proportion of the cases.

SUMMARY OF FOCAL SYMPTOMS.*—*All lesions may involve other areas by contiguity, transference of irritation, actual pressure, or invasion and incomplete clinical pictures are common.*

Cerebral Cortex.—*Frontal.*—Impairment of mentality, change of disposition, irritability, loss of memory, apathy and somnolence, childishness, fits of passion, maniacal outbursts or non-delusional dementia, motor aphasia and agraphia (left lower frontal).

Parietal.—Word blindness (left inferior parietal), hemianopsia (if occipital lobe is affected), disturbed muscle sense and sensation of opposite side of body, incoördination, homolateral tremor.

Motor (Rolandic) Area.—Jacksonian epilepsy (tonic or clonic convulsions) followed by transient, perhaps ultimately permanent paralysis and accompanied or preceded by localizing numbness or tingling (the origin and course of the spasm indicate the area of involvement); *spastic paralysis, i.e.*, increased reflexes, preservation of muscle tonus, nutrition and electrical reaction.

Vertex.—Monoplegias with heightened reflexes or rigidity, are the rule, often preceded by an extremely localized weakness (fingers, toes, etc.). Median growths may produce hemiplegia of the spastic type.

Occipital.—Mind blindness, *lateral homonymous hemianopsia.*

Temporal.—Intercortical sensory aphasia and word-deafness (left side).

Sylvian Fissure and Island of Reil.—Superimposed growths may involve

* Largely based upon the admirable descriptions of Starr, Nothnagle and Ziehen.

adjacent lobes and with the following results: *internal capsule, paraphasia* or hemiplegia; inferior parietal or superior temporal, *sensory aphasia;* third left inferior frontal, motor aphasia; operculum, facial palsy.

Cerebellum.—*Median Lobe.*—Optic neuritis and severe headache appear early, staggering gait or propulsive movements, vertigo (when erect) or actual falls. *Inferior Surface.* Associated cranial nerve involvement. *Middle Peduncles.* Unilateral swaying *in direction opposite to growth,* or propulsion and cranial nerve involvement on same side as lesion. *Superior Peduncles.* Same as corpora quadrigemina.

Corpora Quadrigemina.—Partial, usually unequal and bilateral oculomotor palsy, nystagmus, staggering gait, superior and inferior recti chiefly affected, incoördination of eye movement, unsteady vision. These symptoms are pathognomonic.

Crus Cerebri.—Oculo-motor paralysis same side, hemiplegia opposite side with perhaps bilateral hemianopsia (optic tract).

Pons.—*Upper Pons.—Paralysis of third and fifth nerves, hence anesthesia of face, external strabismus, corneal dryness and ulcer, dilated pupil, ptosis, etc. Knee-jerks often inhibited.*

Lower Portion.—Sixth, seventh and eighth nerve paralysis, internal strabismus, with alternating hemiplegia.

Medulla.—Hemiplegia, hemianesthesia, hemiataxia, paralysis of related cranial nerves including the hypoglossal, vagus and spinal accessory. The wealth of vulnerable structures in this region may yield a multitude of diverse symptoms. Ataxia, vertigo, projection movements, dysphagia, unilateral or bilateral sweating, vomiting, lingual paralysis, cervical retraction, etc.

Internal Capsule.—If this be affected there is hemianopsia, hemiplegia and hemianesthesia.

Optic thalamus, awkward positions of the body and athetoid movements, marked sensory disturbance and, in posterior lesions, homonymous hemianopsia; voluntary facial expression present, involuntary emotional expression absent. In most cases no characteristic symptoms.

CEREBRAL HEMORRHAGE, EMBOLISM AND THROMBOSIS.—

Age.—Cerebral hemorrhage is rare before forty and the tendency increases with age though cases are occasionally seen in children and in young men or women who have a precocious arteriosclerosis. It is doubtful whether there is much difference in its incidence as between men and women.

Family History.—*Hereditary predisposition* certainly is of the highest importance but relates to the basic cause.

Associated Diseases.—Chronic alcoholism, lead poisoning and *syphilis* are important antecedent conditions as are gout and *nephritis* because of the associated high pressure and vascular changes. Traumatism and the hemorrhagic diseases such as hemophilia, leukemia and pernicious anemia must be considered. The acute infectious diseases exert little influence.

Syphilis and nephritis chief factors.

Two factors are essential to the ordinary type of cerebral hemorrhage, namely, weakened vessels and high vascular tension, local or general. Hemorrhages may be large and single or punctate and multiple, cortical or "capsulo-ganglionic."

Hypertension.

Miliary aneurysms.

One of the commonest of immediate pathologic causes is the rupture of such miliary aneurysms as may frequently be found in the medulla, pons, caudate and lenticular nuclei, and optic thalamus. The lenticulo-striate artery, being unsupported, tortuous and without collateral circulation, is the chief offender (75 per cent.). Other cases may be due to congenital hypoplasia or acute toxemic degeneration.

The cases associated with "renal cirrhosis" are said to constitute about 35 *per cent., but this figure must be enormously expanded if modern methods of diagnosis are employed and the term "renal inadequacy" is substituted.*

The author's experience leads him to believe that relatively few cases occur without hypertension due to renal inadequacy. In this condition extreme lability of the blood pressure, uremic exacerbations of general and local arterial hypertension and associated arterio-capillary fibrosis usually coexist.

No common condition is so frequently overlooked as is renal impermeability and high arterial tension, because of the lack of focal subjective symptoms.

Absorption vs softening.

Absorption may be prompt in minor hemorrhages but ordinarily the brain tissue suffers permanent damage and the clinical symptoms depend both upon the position of the clot and its extent. Any hemorrhage that reaches the fourth ventricle will of course produce almost instant death. By extension from the neighborhood of the internal capsule exuded blood may reach and fill any of the ventricles. Cortical lesions result from lesions of the Sylvian artery and occasionally the *pons* or even the *cerebellum* may be the site of the rupture.

Hemorrhage into ventricles.

Cause evident or absent.

Exciting Causes.—Violent muscular strain, uremic arterio-capillary spasm, the sexual act, labor, defecation, fear, anger, joy and similar physical strains or emotional states frequently *determine* an attack, but more often no definite cause can be assigned and it often occurs during sleep.

Signal symptoms.

Premonitory Symptoms.—*Usually none are present, occasionally numbness, prickling or formication in the lower extremities, transient visual disturbance, irritability, throbbing frontal and occipital headache, vertigo, drowsiness during the day, or insomnia may be noted before the attack.**

They may also be present when no cerebral hemorrhage follows. These are for the most part merely symptoms of general arterial hypertension, toxemia, and local shortage of the cerebral blood supply and may be present in cardiac decompensation, arteriosclerosis and hypertension with chronic nephritis.

The reported experience of life insurance companies shows the frequency and early fatality of moderate grades of arterial hypertension, even when associated with no lesion discoverable by the usual tests.

* In a case recently observed two attacks of momentary unconsciousness, mental confusion and sensory aphasia with barely perceptible transient tremor of the left hand were followed in a few days by an apoplectic seizure producing instant death. In another, on two occasions, the patient fell suddenly to the floor without stumbling or tripping, arose immediately, showing neither loss of consciousness, mental confusion or motor weakness, only to die instantly a few weeks later as in the preceding case. Such occurrences are extremely common.

They are significant, therefore, and demand attention, but do not necessarily mean an *impending* cerebral hemorrhage.

Unilateral choreiform movements and spasm of the ocular muscles are rare, and in many instances such symptoms undoubtedly indicate a slight antecedent hemorrhage.* There are two distinct types of seizure, *the sudden* and *the gradual*, the former furnishing the typical cases of "*apoplectic insult*" or "*stroke.*"

"Insult" or "stroke."

Symptoms of Attack.—*Loss of consciousness is immediate and complete, the face congested or ashen and cyanotic. All voluntary muscles are relaxed, the pupils may be moderately dilated, irregular, or, in ventricular or pontine hemorrhage, contracted. The pulse is slow, full, and of high tension. There may be conjugate deviation (head and eyes) toward the lesion because of the paralysis of the muscles on the side opposite the lesion. Conjugate deviation toward the sound side is spasmodic if due to irritation only and the temperature is normal or more usually subnormal. Respirations are slow and stertorous, the cheeks are blown outward with each expiration, and facial paralysis may be indicated by the greater excursion of the paralyzed side. The lips are relaxed, spluttering and foam-covered. In cases of gradual onset, paralysis and loss of consciousness may not appear for several hours. In lesions of the pons, medulla or central ganglia, fever may be present at the onset.*

Striking signs.

Eye symptoms.

Larval cases.

Second Stage.—After a period varying from a few hours to one or two days there is a febrile inflammatory reaction lasting from one to seven or eight weeks. A preagonal hyperpyrexia or sudden drop to subnormal usually indicates extending hemorrhage, rupture into the ventricle or involvement of the medulla or pons.

Reaction.

Hyperpyrexia.

After a variable period consciousness returns, and, usually, to some extent, motor power, while in the limbs that remain paralyzed *early rigidity* develops. *Trophic changes*, chiefly in the form of bed sores, and congestion of the lungs, may add to the gravity of the case. *It is important that paralysis should be recognized in the cases of coma*, as it often at once differentiates apoplexy from other conditions. Ordinarily, it is only necessary to raise the arms and legs and note the comparative resistance offered to passive movement and the change in the tonus as indicated by the dropping of the lifted arm or leg. Bilateral comparison of the passive drop of the arms, hands, or legs is usually most effective and produces sharply defined contrasts between the sound and paralyzed members. Facial paralysis is readily noted and eye symptoms such as conjugate deviation at once suggest the cause.† *Loss of power may be complete* and include the opposite half of the body including the area of the facial and hypoglossal nerve; or, *incomplete*, the face being unaffected. If the *ventricles* be affected *tonic rigidity or marked convulsive seizures* may occur.

Remission.

Trophic changes.

Signs of paralysis.

Ventricular symptoms.

* The author has seen a number of cases in which ocular hemorrhages shortly preceded an apoplectic seizure.

† As stated, the most frequent source of hemorrhage is the lenticulo-striate region, a crossed hemiplegia being produced rather by pressure than actual destruction which accounts for the marked diminution in extent and degree of paralysis so frequently following apoplectic insult.

Reflexes.

The reflexes are entirely abolished during coma or longer periods, though greatly increased usually when the first shock of the attack has passed, and the theory of suspended function through shock seems a sufficiently reasonable explanation of this departure from the law that governs reflex action.

Superficial reflexes.

Residual Symptoms.—*The superficial reflexes* are absent permanently or for long periods over the affected side. *Conjugate deviation is usually transient* but occasionally persists for weeks or months and in these cases

Paralyses.

there is probably a direct lesion of the inferior parietal lobule. In *residual paralyses* we ordinarily find *crossed hemiplegia, usually most marked in the leg and least in the trunk, with increased patellar reflexes. There is unilateral lagging of the thorax.* In respiration the shoulders drop downward, yet the muscles react normally to the electric current and their atrophy is that of

Aphasia and dysphasia.

disuse. *Speech is* restored either entirely or to a considerable degree, often very early, but *dysphasia* may persist for some time. The well-known *hemiplegic gait* is due to the patient's throwing the balance of the body

Hemiplegic vs spastic gait.

constantly toward the sound side, *the affected leg swinging outward and striking the ground in a flail-like manner.* Later flaccidity is replaced by spasticity, when, as a rule, predominating extensor contraction and a typical spastic

Arthritis.

gait appears. Usually improvement in the leg is more rapid than in the arm and in the latter spastic contraction produces flexion of both elbows and

Secondary degeneration.

fingers. A low-grade arthritis from disuse is frequent with pain and partial ankylosis, symptoms of vasomotor paralysis may appear and a secondary

Hemianesthesia.

descending degeneration may intensify the deep reflexes even of the sound side. Hemianesthesia affecting pain, temperature and touch is present though imperfect and ordinarily transient. *If marked and persistent* it constitutes a direct focal symptom. Muscle sense is less often affected but disturbances of vision and aphasia may be indirectly produced, and be transient or focal, persistent, or even permanent. Facial paralysis usually affects

Relative involvement.

only the lower branches; the muscles of the neck and trunk are relatively less affected than those of the extremities and the hypoglossal paralysis is

Athetosis.

seldom complete. *Choreic or athetoid* movements may appear during the regenerative stage and usually indicates a focus in the posterior part of the internal capsule or the adjacent optic thalamus.

Indefinite in early stage.

Localizing Symptoms.—*As regards the localizing symptoms it must be remembered that correct localization is seldom possible in the earlier stages of an apoplexy.*

Important local signs.

The persistence of a hemianesthesia suggests a focal lesion in the posterior portion of the internal capsule, the central convolutions or the intermediate medullary tissue.

Monoplegia indicates a lesion of the centrum ovale, the cortical motor center, or a central paralysis of such nerves as the facial and hypoglossal if these are alone affected.

If aphasia complicate a monoplegia a cortical lesion is most probable, if aphasia is absent the centrum ovale is suggested.

If hemiplegia is associated with cranial nerve paralysis of the opposite side from the fifth backward, a lesion of the pons or pons medulla on the

side of the cranial nerve paralysis is probable. If *ocular motor alternating paralysis co-exists with hemiplegia* a lesion of the peduncular tract is suggested. Hemorrhage into the internal capsule generally produces complete hemiplegia.

Differential Diagnosis.—*Symptomatic apoplexies*, those pseudoapoplectic seizures that occur in the course of such diseases as cerebral tumor or abscess and paralytic dementia, may present the greatest diagnostic difficulty, though in most instances such a case history is known or obtainable as at once suggests their true nature. Absence of choked disc helps to exclude brain tumor. If hemianesthesia appears, it is usually transitory. Certain epileptic seizures resemble apoplexy, but the subsequent course and previous history usually suffice.

Case history helps.

Condition of disc.

Uremia.—An immediate diagnosis is often impossible even in the presence of urinary signs of renal disease. Convulsions are more likely to occur as an initial symptom of uremia and the subsequent course is usually distinctive, "uremic paralyses" without actual vascular rupture being rare.

Uremic paralyses rare.

Meningitis.—This condition often offers serious difficulties particularly as certain apoplexies are associated with painful contraction and rigidity of the neck muscles. The persistence of convulsions, their bilateral incidence, the general hyperesthesia, the possible demonstration of optic neuritis and the history and course of the case must be depended upon as indicating meningitis.

Acute sepsis may produce pseudo-apoplexies but the clinical picture soon becomes distinct.

Hemorrhagic pachymeningitis if unilateral cannot be differentiated from a cortical hemorrhage of slow onset.

Hysteric Hemiplegia.—*Hysteric paralyses are usually shifting and transitory and seldom involve the facial nerve or produce hemiopia. Sensation is markedly and usually atypically affected as compared with apoplexy. There are usually visual, olfactory and auditory disturbances or hallucinations and as a rule the well-known hysteric stigmata make the diagnosis.*

As regards the secondary changes it cannot be decided positively by any known diagnostic test whether in a given case these are due to cerebral softening or to cerebral hemorrhage.

Finally it cannot be too emphatically repeated that focal disturbances cannot be correctly localized during the apoplectic seizure, and judgment must in every case be suspended until the various and confusing indirect primary symptoms have had an opportunity to subside.

INTRACRANIAL ANEURYSM.—This extremely rare condition usually affects the basilar artery and probably is almost invariably syphilitic in origin.

Symptoms.—Unless associated with a distinct *bruit* these may closely resemble or be identical with those of cerebral new-growths.

Choked disc is said to be rare.

EMBOLISM.—*The diagnosis depends almost entirely upon a consideration of the factors of antecedent disease and sometimes the age, as the symptoms of an*

actual attack are so nearly like cerebral hemorrhage as to render positive differential diagnosis impossible in most cases.

Antecedent disease of the heart.

Diseases of the heart and blood vessels are most important, whether rheumatic, arteriosclerotic, myocardial or aneurysmal.

Associated conditions.

Nine out of every ten cases of cerebral embolism are due to preëxisting heart disease, less frequently perhaps in the acute cases of endocarditis than in the recurrent inflammations attacking valves already diseased.

This is especially true of mitral stenosis in the young and luetic cardiovascular lesions in persons above forty years of age.

In acute septic endocarditis the embolus may be capable of exciting abscess instead of the mere softening due to the blocking of the area supplied by one of the cerebral arteries.

In any form of severe decompensation such emboli may result, not necessarily from particles detached from the valves, but from fragments of a clot which may exist either in the auricles or ventricles.

The attack may come on during quiet sleep, be transient and quickly recovered from and not infrequently there is a history of *antecedent embolism* in other portions of the body.

Heart murmur.

In certain cases a marked and sudden change in the character of the heart murmur may suggest the detachment of a particle of vegetation or auricular clot.

A large number of differential symptoms are given by different authorities, but neither the author's experience nor his knowledge of the conditions attending the two lesions lead him to give them any important place.

The fact that 90 per cent. of all cases of cerebral embolism depends upon an antecedent lesion of the left heart points the way to differential diagnosis.

THROMBOSIS.—This lesion may be entirely symptomless even when not occurring in the so-called "silent" regions. The localizing symptoms and resulting paralysis are so nearly alike in thrombosis, embolism and hemorrhage as to permit no accurate differentiation from this standpoint.

Mode of onset.

The mode of onset of the paralysis in thrombosis is more suggestive, more or less distinct premonitory symptoms usually being present.

Premonitory Symptoms.—Headache, vertigo, numbness, formication, slight weakness of a limb or of one side of the body, defects in memory or speech, irritability are some of the many symptoms, suggestive if progressive and not removed by treatment directed to sustain the heart, *the symptoms being far less violent, coma seldom present and consciousness being, as a rule, either wholly or partially retained.*

Mental state.

Mental symptoms are much more pronounced than paralytic symptoms after the attack and usually assume the form of general mental impairment with marked irritability and a tendency to emotional excitement. Slowing of the pulse is seldom present.

Finally, the differential diagnosis of these diseases is assisted by the fact that 80 per cent. of the so-called "apoplectic" attacks are chargeable to cerebral hemorrhage, and the physician's mind is also comforted by knowledge that an immediate differential diagnosis is of little importance to the patient.

CEREBRAL ABSCESS.—It might be thought that cerebral abscess

would present striking general symptoms which differentiated it from cerebral tumors and other confusing lesions.

Such, however, is not the case save in exceptional instances, and the diagnosis ordinarily depends more upon the recognition of one of the well-known causative factors than upon any specific or exclusive symptomatology.

Of such causes we may enumerate acute or recurrent septic endocarditis, traumatism with or without fracture, suppurative diseases of the scalp, empyema, pulmonary cavities and most important of all chronic suppurative otitis media, a condition frequently associated with mastoiditis and caries.

Abscesses caused by middle ear disease and mastoiditis are most frequently in the cerebellum, whereas, when from other causes, they chiefly affect the cerebrum, abscesses of the basal ganglia or brain stem being rare.

The accumulation may be superficial or within the substance of the brain, and 80 per cent. are single; multiple abscesses resulting usually from more remote foci, such as empyema. In diameter they vary usually from 2 cm. to 8 cm.

Symptoms.—*Extraordinarily latent cases have been reported which for several months or years yielded few or no indications of their presence. More often the symptom-complex is essentially that of brain tumor and consists largely of pressure phenomena.* Larval cases.

In acute cases following operation or injury or in those secondary to an otitis media, the symptoms are primarily those of a *meningeal inflammation, and there may be fever, chills, vomiting, delirium and severe headache,* perhaps preceded by symptoms that are irritative in character. Primary vs. secondary. Meningeal form.

In the later stages of abscess, *mental torpor, drowsiness and optic neuritis* are common, *a period of latency* is the rule in cases which arise from distant foci and not uncommonly *from those due to direct injury.*

The localizing symptoms are, of course, less distinct than in tumor but follow the same rules. Brain tumor symptoms.

Fever is very often entirely absent and overlooked when present if the primary disease be febrile. Not infrequently, there is subnormal temperature.

It is evident, therefore, that it is only by a combination of suggestive etiological factors, the symptoms of intracranial pressure and perhaps of septic absorption, that a differential diagnosis is made. MacEwen's method of skull percussion is more likely to yield results in abscess than in any other condition producing intracranial pressure. *The increased resonance* obtained depends upon a distention of the lateral ventricles and compression of the venæ galeni. *Optic neuritis* is relatively common but choked disc is far less constant than in brain tumor or meningitis. Seek causative factors. Skull percussion. Optic neuritis.

DEMENTIA PARALYTICA.

("General Paralysis of the Insane"), ("General Paresis")

Etiology.—The etiologic factor in general paresis is identical with that of locomotor ataxia and the diseases seem to differ only in the seat of the morbid process. *Both are caused by the vascular ravages of Treponema pallidum of syphilis and they may occur in combination (tabo paresis).* A form of syphilis.

Morbid Anatomy.—Amongst the multitude of diverse findings are: a small brain in the late stages, with shrunken convolutions, sclerotic arteries, hyperemia, and edema in the active early periods, hypertrophic pachymeningitis, thickened and adherent pia, an increase of cerebrospinal fluid with dilatation of the fourth and lateral ventricles, and loss of the fine tangential fibers in the superficies of the cortex. (See Cardiovascular Syphilis.)

Microscopically there is general neuroglia overgrowth (sclerosis) with degeneration of cells and fiber atrophy, and changes are often found in the spinal cord identical with those of locomotor ataxia. The whole brain is involved though in varying degree.

Symptoms.—As in locomotor ataxia we deal with a *syphilitic-progressive degeneration ending in general exhaustion and palsy*, but, unlike its purely spinal congener, associated with marked mental symptoms and terminal dementia.

It may be divided into two stages, to which some add a prodromal stage, and varies considerably in type.

Neurasthenic symptoms.

First Stage.—The early symptoms of this stage are significant but not distinctive, being identical with those of certain forms of so-called "neurasthenia." *A change of character* is soon evident, carefulness, good nature, self-restraint, frugality, good judgment, high ethical standards, concentration, vigor, and modesty, give place to carelessness in matters large and small, peevishness, irritability, or violent outbreaks of temper, periods of extravagance and foolish investment, coarseness and perhaps brazen immorality. The victim is inattentive to occupational and domestic duties and obligations, tires easily and lacks concentration and ability to perform sustained work. He is garrulous, egotistic, yet jocose and loud in voice and manner. *Delusions of grandeur* shortly appear and the patient deals only in superlatives. His enormous wealth, marvelously simple yet grandiose schemes, his house, his wife, his children, are all wonderful beyond description and he, incomparable.

Change of character.

Garrulity.

Delusions of grandeur.

Violent outbreaks may occur in this later period of the first stage, often associated with drinking bouts and with sexual excesses marked perhaps by extreme perversion.

Impaired memory.

Writing and speech.

Knee jerks.

Pupils.

In the later period of this stage or even during the earlier exaltation period lapses of memory may occur, *the handwriting may be uncertain, letters lack coherency*, errors in figuring occur and one may *note tremor of the facial muscles and tongue, difficult enunciation of consonantal words such as "British Constitution,"* the *speech being hesitant, slurring or even stuttering. The knee-jerks are increased or lost, but always abnormal and the pupils miotic, often sluggish and unequal or of the Argyll-Robertson type.*

The Argyll-Robertson pupil is practically a pathognomonic sign of syphilis of the nervous system, occurring very rarely in any conditions other than paretic dementia and locomotor ataxia.

Various symptoms.

Attacks of vertigo or syncope, pseudo-apoplexies with hemiplegia, or transient epileptiform seizures may occur and *insomnia* may be troublesome and intractable but *true apoplexy is uncommon* in this stage.

Sexual weakness and *loss of vesical* power may be manifest but the vegetative function may be long preserved and the appetite is often gluttonous. Either the Wassermann or the luetin test is usually positive and the spinal fluid yields a lymphocytic picture.

Second Stage.—*This is the stage of decided and increasing dementia.* Dementia.
Memory, even for familiar faces and common events, becomes fitful and almost lost. *Personal cleanliness* is neglected, there is *slobbering at meals* and a *gradually increasing dependency. Apoplectiform attacks* are followed *by permanent paralysis, hemiplegic in type, and true apoplexy may supervene and terminate the case.* Primary cerebral palsies.

The duration of this form is extremely variable, the stage of dementia being usually reached in a few months after the appearance of pronounced mental symptoms. Death occurs sometimes within a year, usually within two or three, but in tabo-paresis may be postponed for fifteen or twenty. Duration.

Remissions are not infrequent but entire recovery usually means an error in diagnosis. Remissions.

Variation in Type.—*Dementia is sometimes primary, the period of exaltation and excitement being entirely lacking.* Primary dementia.
In such instances the premonitory symptoms *are almost precisely like those of pronounced "neurasthenia,"* in other cases a *marked hypochondriasis is substituted for exaltation.* Hypochondria.
In some of these cases there is much complaint of dull pain in the head, trunk and extremities. *Other cases are of a primary exudative type* evidenced by primary ocular palsies or cephalalgia with convulsive seizures. Cephalalgia.
These cases are of especial interest because of the fact that terminal symptoms of dementia are often postponed for five or ten years. Tabo-paresis.
Of equal importance are *those cases of tabo-paresis presenting a combination of locomotor ataxia and paretic dementia.* In these the onset may be primarily that of a locomotor ataxia, or, such as to immediately suggest the combined lesion. *When the cord symptoms are pronounced the brain symptoms are usually delayed and vice versa. It must be remembered that various forms of mental disease other than general paresis may complicate tabes dorsalis.* Cord vs. Brain symptoms.

Differential Diagnosis.—*The earliest stages of paralytic dementia in its ordinary form cannot be made the basis of an absolute diagnosis without risking a serious and dangerous error in diagnosis unless lues is proven.* Early accurate diagnosis impossible.

With the appearance of pupillary symptoms and marked tremor of the face and tongue, or marked and characteristic exaltation, the clinical picture is greatly cleared.

Furthermore, cases of pure cerebral syphilis of the curable type exactly simulate one form of paresis and lead to error. A differential diagnosis may be impossible in such instances for a considerable length of time, pending the result of active and radical specific medication. Curable cerebral syphilis.

All cases resembling a suggesting paretic dementia should be carefully examined for lead poisoning (lead line, occupation, etc.) and alcoholism must be given consideration. Hemorrhagic pachymeningitis actually complicates about 20 per cent. of the cases of paretic dementia.

Senile dementia runs a very slow and relatively passive course and occurs

after the sixtieth year almost invariably. Disseminated sclerosis lacks the mental symptoms and is a disease of youth.

A positive Wassermann or luetin test may be determinative and the spinal fluid mononucleosis and globulin increase are of great differential value in many instances.

It should be remembered that general paresis represents a very considerable proportion of the insanities and must be borne in mind when dealing with dubious cases.

ANEMIA OF THE CORD.—Only in the most profound acute and chronic anemias do symptoms from the cord manifest themselves.

In *pernicious anemia* hemorrhage may occur or even a true postero-lateral sclerosis. Cord symptoms of severity may follow a great loss of blood and on the other hand marked changes may be found at autopsy which have yielded no symptoms during life. The diagnosis can seldom be made save tentatively and is based upon the presence of anemia as an etiological factor together with localizing symptoms in the cord itself.

THROMBOSIS, EMBOLISM, ENDARTERITIS OF THE CORD.—However important these conditions may be as the primary factor in certain degenerative changes, they are by themselves of no clinical importance.

LOCOMOTOR ATAXIA

(*Tabes Dorsalis*), (*Syphilitic Posterior Spinal Sclerosis*)

Definition.—A disease characterized by a gradual onset and prolonged course, associated with loss of the deep reflexes and normal pupillary light reaction, visceral crises, incoördination, lightning pains and ultimate paralysis.

Adult males.

Etiology.—*It is essentially a disease of the male, more common in the white than in the colored race, occurs most frequently between the ages of thirty and fifty, and is caused by the Treponema pallidum of syphilis.*

Syphilis the cause.

Alcoholic and sexual excess, exposure, fatigue and injuries are occasional associated conditions, but are probably not causative and usually not even contributory. Aside from hereditary syphilis, which occasionally causes the disease in children, heredity plays no part.

Degeneration of sensory structures.

Morbid Anatomy and Pathology.—*It is essentially a posterior spinal sclerosis* involving primarily the larger dorsal root fibers, though changes in the blood vessels of the nerve roots, pia mater and cord may appear early. The disease is a progressive degeneration and even though the primary lesion is not in the cord itself, the major changes are ultimately to be found there, and even macroscopic examination reveals the morbid condition in most instances.

Pupillary signs.

SYMPTOMS. (*Incipient Stage*).—**Eye Symptoms.**—*The Argyll-Robertson* pupil* showing a loss of the iris reflex to light, with normal reaction to accommodation, is one of the earliest symptoms. *The pupils are often contracted,*

* As stated previously this pupil reacting to accommodation and not to light, wherever found, single or double, with or without other symptoms, almost invariably means *syphilis of the nervous system.*

perhaps unequally so, and there may be *optic atrophy, ptosis, paralysis of the external recti or even total blindness.* **Dysuria.** This is an early symptom, whereas incontinence occurs later in the disease.

Dysuria and incontinence.

Loss of Deep Reflexes.—*Early in the disease the knee jerk is diminished and finally lost, the response being often unequal on the two sides until both are abolished, and the Achilles jerk is usually first to disappear.*

Knee- and Achilles-jerk.

Pain.—*The characteristic lightning pains are acute, darting, stabbing, follow dorsal root areas,* are irregular in occurrence, variable in intensity, likely to be initiated by excesses of any kind and leave as suddenly as they appear. They most often affect the legs, but may occur in the arms, head or trunk, and sensitive areas and herpetic eruptions may follow them.

Often misinterpreted.

Ataxic Stage.—*Romberg's sign* is one of the earliest, the patient being unable to stand steadily with the feet together and the eyes closed. Furthermore, he cannot stand on one leg or start off promptly when ordered, his turns are made laboriously and cautiously and descending stairs is difficult. It may be present in the first stage and antedate the loss of knee-jerks.

Static ataxia.

The characteristic gait (ataxic) develops later, the patient throwing out the leg with jerky, uncertain action, keeping the feet far apart to secure a wider base, and bringing them down heel first with a sort of stamp; being obliged ultimately to use one or even two canes or becoming totally disabled.

Ataxic gait.

Incoördination is less prominent in the arm movements than in those of the leg and muscle power is remarkably preserved, as is nutrition, until the later stage of the disease. There is unusual mobility and relaxation of the joints which occasionally appears at a comparatively early stage.

Incoördination.

Muscular power.

Joints.

Various sensory symptoms develop even during the pre-ataxic period, most marked in the lower extremities, the patient feeling often as if the feet were muffled in cloths or cotton and losing the sense of the resistance of the ground. Sensations of numbness and tingling are prominent and *lightning pains* may or may not exist in varying degrees of severity.

Anesthesia.

Paresthesia.

Not only is the *tactile sense* impaired but the *sense of pain* as well, and this is associated with a marked retardation of pain transmission so that several seconds may elapse before the prick of a pin is perceived.

Analgesia.

Oddly enough ataxic symptoms are unusual or at least greatly delayed in those cases in which optic atrophy is an early sign (one in ten).

Significance of optic atrophy.

Visceral Crises.—*These remarkable symptoms constitute a frequent source of error in diagnosis and may be gastric, intestinal, rectal, renal, urethral, clitoral or laryngeal and associated with marked pain in and functional disturbances of the parts affected.*

Misleading.

At this stage incontinence of urine may displace simple dysuria or retardation, cystitis and even pyelo-nephritis may occur; sexual power, diminished in the early stage, is now usually lost, and simple constipation may be replaced by *critical rectal pain and spasm and ultimately by relaxation of the sphincter.*

Urinary and rectal symptoms.

Trophic Changes.—*Perforating ulcer of the foot and the so-called Charcot's joint* are the most prominent of these changes. The former appears usually on the sole of the foot beneath the great toe, the latter is a *painless disinte-*

Charcot's joint and perforating ulcer.

gration of the joint associated with effusion, deformity and tendency to dislocation. The bones are brittle in this disease and spontaneous fracture may occur.

Cutaneous symptoms.

Various skin rashes, areas of sweating, herpes and edema may appear in connection with lightning pains. Muscular atrophy seldom appears until later in the disease but the nutrition of the nails may be greatly affected.

Brain Symptoms.—Paranoia, melancholia, and dementia may occur, and paralytic dementia *is* "cerebral tabes," the difference being merely one of localization.

Loss of power delayed.

Long duration

Duration.—Paralyses are seldom marked until from five to eight years have elapsed; even then progress is slow, periods of apparent arrest not uncommon, while actual arrest sometimes occurs. The chronic course of the ailment is remarkable, the patient may live for twenty-five or thirty years, though rarely cases occur in which the progress of the disease is rapid. Death results from *intercurrent disease* in nearly every instance.

Typical cases simple.

Differential Diagnosis.—Tabes when established, need seldom be confounded with any other disease though it may be combined with general paresis, *tabo-paresis* and its separation from that ailment is clinical rather than pathological, *the combination of loss of deep reflexes, the Argyll-Robertson pupil* and lightning pains making a distinct clinical picture. In true ataxic paraplegia the knee-jerks are increased, in chronic multiple neuritis with ataxia ("pseudo-tabes") there is marked muscular atrophy and tenderness over the affected nerves.

ATAXIC PARAPLEGIA (*"Gower's disease," "postero-lateral sclerosis"*).—*A combination of marked incoördination and slowly developed spastic paraplegia with retained and increased reflexes and frequently ankle clonus, making it a clean-cut clinical picture.*

It occurs chiefly in males between thirty and forty years of age, and is associated with degenerative changes in the dorsal and lateral columns of the cord and is of unknown causation. The sphincters are usually involved and there is a late development of mental symptoms similar to those of paretic dementia. Cranial nerve symptoms are seldom pronounced. There may be "dull aches," rarely lightning pains.

The combination of spastic paraplegia with ataxia is striking and lacks all important tabetic symptoms.

Friedreich's Ataxia occurs only in children and young adults, shows scanning, syllabic speech and lost reflexes.

PRIMARY COMBINED SCLEROSIS.—*This name has been given by Putnam to cases presenting clinically progressive weakness, numbness of the extremities, exaggerated tendon reflex, spastic contraction and paraplegia with pronounced anemia.*

HEREDITARY ATAXIA (*Friedreich's Ataxia*).—This disease of childhood or young adults it is not *invariably* hereditary. It is associated with degeneration of the dorsal and lateral columns of the cord.

Symptoms.—*The chief features are marked incoördination involving first the legs, but later the arms, and these to a marked degree. The gait is uncertain, stumbling and staggering (cerebellar), rather than stamping, as in locomotor*

ataxia. Arm movements are jerky and irregular and grasping involves pouncing upon the object. Tendon reflexes are diminished or lost though a Babinski is sometimes present, there is early talipes equinus, the big toe becomes flexed dorsally and nystagmus occurs late. Pupillary reactions and the optic nerve are unaffected. Paralysis and mental impairment usually come on late. The speech is indistinct, slow and scanning, and both scoliosis and kyphosis are common.

Both choreiform and athetoid movements may occur. Speech is peculiarly slow and disorderly.

CEREBELLAR HEREDITARY ATAXIA (*Marie*).—*This later-life form of hereditary ataxia, presumed to be associated with a congenital cerebellar defect, is characterized by impaired pupillary reaction, exaggerated tendon reflex, clonus, ocular palsies, optic atrophy, and, by its development in the third decade.*

Incoördination, nystagmus and scanning speech are present as in Friedreich's ataxia, but the gait is more distinctly reeling and drunken.

It is doubtful if any line should be drawn, as in the two diseases the same general tracts are affected, the spine in Friedreich's ataxia, the cerebellum in that of Marie, the actual tracts being structurally continuous.*

PRIMARY LATERAL SCLEROSIS (*Erb-Charcot Disease*) (*"Spastic Paralysis of the Adult"*).—This is a rare disease of early adult life, between the ages of twenty and forty, no definite etiologic factors have been proven and the pathologic change is essentially a degeneration of the pyramidal tracts, either primary or secondary.

Symptoms.—*Muscular rigidity and weakness are often associated with dull pain and a persistent sensation of fatigue. Rigidity and impairment of locomotion slowly increase and are out of proportion to the loss of power.*

Spasticity and lost power.

Tension and stiffness of the legs are first symptoms in most instances and the constant "stubbing" of the toe may wear away the shoe in a significant way.

"Toe stubbing."

The spastic gait is marked early in the disease and later becomes extreme, the patient shuffling along with toes dragging, tripping over every obstacle, the legs being closely approximated or even so crossed (adductor spasm) that locomotion is lost.

Spastic gait.

The knee-jerk is extremely active, Babinski's sign, rectus and ankle clonus are easily elicited, superficial reflexes are increased and in some instances clonus appears whenever the foot is placed upon the ground and the slightest touch may produce clonic spasm of the legs. The latter may be equally or unequally affected and the arms are usually free until the disease is far advanced.

Increased reflexes.

The ailment is one of long duration, yet nutrition is remarkably preserved, sensation is not affected and the sphincters are rarely involved until late in the disease. Ultimately an extreme state of disabling contracture and atrophy from disuse develops. Diagnosis is never positive, as *primary* sclerosis is excessively rare and cases usually prove to be secondary.

Atrophy terminal only.

* L. F. Barker working with the clinical collaboration of Sanger Brown, established the fact that *congenital hypoplasia of the cerebellum and spinal cord "with outspoken degenerations of the spino-cerebellar paths" occur in this disease.*

Secondary Spastic Paralysis.—It is hardly possible to separate secondary from primary spastic paralysis which may result from any condition producing a lesion in the pyramidal tract, *i.e.*, compression, tumor, caries, etc.

Heredity and age important factors.

HEREDITARY SPASTIC SPINAL PARALYSIS (*Hereditary Spastic Paraplegia*), (*Family Form of Spastic Spinal Paralysis*).—*This variety is distinguished from spastic paralysis chiefly by its distinctly familial nature and the fact that it commences several years after birth, usually at about the age of five.*

Spastic type.

Two groups are distinguished, viz., those with the symptomatology of a *cerebral spastic paraplegia* developed in infancy and childhood, but *lacking all other cerebral symptoms*, and those developing in early adult life insidiously with primary spasticity and late paralyses.

Begins in Infancy.

THE SPASTIC PARALYSIS OF INFANTS (*Birth Palsy*), (*Spastic Cerebral Paraplegia*), (**Little's Disease**), (*Spastic Diplegia*).—This condition is essentially a *general spastic paralysis occurring at or shortly after the time of birth*, more rarely following a convulsive seizure or an acute disease.

Normal sensation.

The legs are chiefly affected without marked *sensory disturbances or wasting and the reflexes are increased. Athetosis and ataxia are extreme and the mental condition markedly affected.*

Increased reflexes.

The condition is no doubt most often due to meningeal hemorrhage and the interference with mentality will depend upon the extent and location of the effusion. In cases of premature birth, spastic paraplegia without mental symptoms is thought by some to be due to an imperfect development of the pyramidal tracts. *In most instances, however, the disease is associated with instrumental delivery and seems to be due to actual injury.*

Fig. 619.—Little's disease. Diplegia. (*Gordon.*)

Backwardness and mental defects.

Backwardness of the child may first call attention to the condition, or there may have been *repeated convulsive seizures. The head may seem badly supported, the child may not be able to sit up and shows no tendency to walk or creep at the proper age. Leg rigidity and adductor spasm* are usually marked.

Oscillation.

Symptoms *are commonly either absent or less marked in the arms; when marked it constitutes a* **spastic diplegia.** *Constant irregular larger movements* of a choreic type may be present in the extremities, sometimes most marked

Athetosis.

when coördinate movement is attempted, and pronounced bilateral athetosis is often present.

Slight rigidity

Erb's Syphilitic Spinal Paralysis.—*The leading features are slight muscular rigidity, exaggerated deep reflexes, pain and sensory disturbance trifling,*

involvement of the rectum and bladder, impotence, a slow onset and a certain amenability to treatment.

Involvement of sphincters.

HYSTERICAL SPASTIC PARAPLEGIA.—Hysterical persons can accurately simulate true spastic paraplegia and differentiation is sometimes extremely difficult, but a few positive points can be laid down.

Such are: spurious clonus, more marked and irregular disturbances of sensation, variability in the site and intensity of paralysis, unsustained or irregular resistance to passive movements, frequently associated hysterical stigmata and absence of the Babinski.

Spurious clonus.

Peculiar resistance.

FIG. 620.—Little's disease. Paraplegia. (*Gordon.*)

AMAUROTIC FAMILY IDIOCY.—The chief characteristic of this form of infantile paralysis affecting children during the first or second year of life is the constancy of blindness, first, partial, later, complete, associated with optic atrophy. The children become idiots and either spastic or flaccid paralysis may develop. It is a family disease, kills usually before the end of the second year, and is sometimes associated with auditory disturbances, strabismus and nystagmus.

Blindness and idiocy.

Early death.

SYRINGOMYELIA.—*Gliosis Spinalis.*—Pathologically, this disease consists in the formation of embryonal neurogliar tissue which undergoes hemor-

rhagic or degenerative changes, resulting in an enlargement of the central canal of the spinal cord quite distinct from ordinary dilatation; it is a disease of early adult life, almost invariably involves the cervical region, sometimes assumes a family type and affects men much more frequently than women.

Cervical region chiefly.

Dissociated anesthesia.

A most pronounced diagnostic feature is a loss of thermal and painful sensation without marked involvement of the tactile or muscle sense. This is usually accompanied by pain in the extremities and cervical region, followed by peripheral muscular atrophy of the hands, slowly extending centrally. Scoliosis is usually present, the legs may, late in the disease, present spastic paraplegia and cutaneous disturbances both trophic and vasomotor are common.

Spinal symptoms.

Such are dry or sweating skin, congestion or edema of the extremities, especially the hands, bulbous or herpetic eruptions, atrophy and disintegration of the nails, erosions, fissures and ulceration of the terminal phalanges. *It should be noted that these cutaneous lesions are painless.* Spontaneous fractures and arthropathies may occur and late in the disease the vesical, rectal and genital centers may be involved and bulbar symptoms appear. Reflexes are usually increased and frequently the general aspect is that of amyotrophic lateral sclerosis, though if the dorsal columns are involved, tabetic symptoms may be marked.

Painless cutaneous lesions.

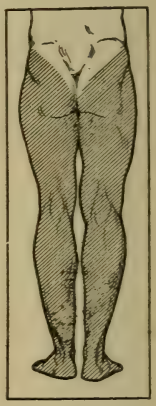


FIG. 641.—Anesthetic area common in hysterical paraplegia. The genitals are spared. (*After M. Allen Starr.*)

Mixed types.

Clinically, one may encounter a straightforward type lacking the predominance of confusing motor or sensory changes, or the picture may be that of hematomyelia, cervical pachymeningitis, progressive muscular atrophy, amyotrophic or spastic paralysis, tabes, hysterical hemiplegia or a type in which trophic changes predominate. (*"Morvan's syndrome."*)

The last is the only one which often can confuse diagnosis if the peculiar sensory symptoms are present. In Morvan's syndrome tactile sense is faint and trophic changes dominate the clinical picture.

Varieties.

MYELITIS.—This may be acute, *subacute or chronic* and the term is usually confined to a lesion involving both gray and white substance, *poliomyelitis*, which involves the gray matter only, being considered separately. As regards the position and extent of the lesion we distinguish cases that involve a considerable vertical area as "diffuse," those affecting the whole transverse area as "transverse," and those with foci scattered throughout the cerebrospinal tract as *disseminated*.

COMPRESSION MYELITIS.—This type is associated with traumatism, spinal caries, aneurysm, new growths and parasites, more rarely with large syphilitic growths.

Seek cause.

Diagnosis.—The diagnosis depends somewhat upon the recognition of an *antecedent caries, aneurysm, malignant growth or syphilitic infection.* This form of myelitis is as a rule *much more painful than acute myelitis* and in malignant disease especially most excruciating suffering may be witnessed from compression of the nerve roots, either directly by the bones or a tumor, or by

an exudate. *In unilateral compression* the Brown-Séquard's syndrome may be evident. *Paraplegia* in these cases is usually slow in development and preceded by weakness and paresthesia, the girdle sensation is usually marked and sensation persists longer than motion. If the cord be involved above the lumbar region, the paralysis is spastic with exaggerated reflexes; if below, the reflexes are usually diminished or lost and the sphincters involved. *Recovery may be complete* in cases of pressure due to deformity if the latter be corrected. Brown-Séquard type.

ACUTE MYELITIS.—*An inflammation of the cord substance may follow or more rarely directly complicate acute infectious diseases*, chief of which are variola, severe dysentery and acute intestinal toxemia, diphtheria, typhus, acute rheumatism, measles, septicemia and pyemia, gummatous or *endarteritic syphilis*. Less important are erysipelas, pneumonia, malaria, puerperal fever, scarlatina and influenza and certain acute chronic intoxications such as alcoholic and gas poisoning and gout. Traumatism, with or without fractures (sometimes mere concussion, violent torsion or flexion), caries, and not infrequently a combination of fatigue and exposure to wet and cold may initiate an attack. It may also be due to the extension of meningitis or even neuritis, or develop secondarily in tuberculosis and syphilitic processes and *it occurs chiefly in men* and *between the ages of twenty and forty*. Causes. Sex and age.

Symptoms.—*Premonitory malaise, sense of weakness and numbness, chill or chilliness and a variable degree of fever accompany the attack. One must first diagnosticate a myelitis, secondly, determine the position and extent of the lesion; and finally, determine the probable cause as a factor in prognosis.* Premonitory.

The essential symptoms are: (*a*) *Hyperesthesia, absence of reflexes and a girdle pain* (*painful constriction*) *at the level of the lesion.* Localizing.

(*b*) *Rapidly developing, not immediate, motor paralysis* below the lesion chiefly affecting the flexors, partial or complete, according to the degree of transverse involvement.

(*c*) *Increased reflexes* below the lesion.

(*d*) *Involvement of the sphincters*, rectal and vesical.

(*e*) *Vasomotor phenomena, i.e.* congestion, sweating.

(*f*) *Trophic changes*, especially pressure necrosis over the heel, hips and other dependent portions.

(*g*) *Changes in muscle tonus, nutrition and electrical response varying with the level of the lesion.*

The actual level of the lesion is best indicated by the upper margin of anesthesia, the hyperesthetic level being just above it. It will be seen at once that the region involved markedly affects the motor symptoms.

If in the lumbar enlargement the paralysis is typically flaccid, with atrophy, loss of knee-jerks, R.D. and urinary incontinence. Lumbar type.

If mid-dorsal or cervical, the paralysis is spastic and the reflexes are usually lost only in the areas corresponding to the actual level of the lesion. The picture is ordinarily that of spastic paraplegia with retention of urine, increased muscle tonus, Babinski's sign, exaggerated ankle clonus and patellar reflex. Mid-dorsal. Ordinary type.

In the cervical form flaccid paralysis may be marked and relatively extensive at the level of cord involvement and if the fourth cervical roots be involved death may result from paralysis of the diaphragm, and vomiting, bradycardia, syncope, hiccough and dyspnea may occur. **Disseminated foci must be recognized by regional diagnosis.**

Upper boundary zone.

The upper level of a transverse or diffuse lesion is clearly indicated by the girdle pain and zone of hyperesthesia, which may be emphasized by passing a cloth or sponge over the spine. Optic neuritis and miosis may occur in cervical

Unusual signs.

myelitis. Symptoms of irritation (spasm or convulsions, pain) if present are usually of short duration, but the diseased area tends to extend. *If diffuse there is acute ascending paralysis.*

Hemorrhage and meningitis.

Differential Diagnosis.—*Hemorrhage* into the cord is characterized by absolute suddenness of onset, and frequently lacks fever. *Abscess, by suppurative foci;* in *meningitis* the irritative, painful and spasmodic symptoms predominate, increasing with disease duration until stupor or coma supervenes, whereas in *myelitis* they are relatively slight or of brief duration. The diseases may of course co-exist. Only a dorsal myelitis could be confounded with hysteria, and here girdle sensation, sphincteric involvement, true ankle clonus with perhaps extension spasm and the trophic changes are

Landry's paralysis.

distinctive. In *Landry's paralysis* the lesion is an *ascending motor paralysis, flaccid throughout,* with normal electrical reactions, lost reflexes, and usually,

Neuritis.

good sphincteric control. *Multiple neuritis* presents at times great difficulty, but ordinarily the *more gradual onset* of paralysis, *marked pain and local tenderness over peripheral nerve areas,* and the *less frequent sphincteric disturbance* makes the diagnosis clear. The combined cases offer more difficulty.

Poliomyelitis.

Acute Anterior Poliomyelitis. This shows flaccid paralysis throughout, seldom involves the sphincters, and lacks the girdle pain and other sensory

Subacute myelitis.

and trophic phenomena of myelitis. *Subacute Myelitis.* Differs from the acute only in its gradual onset (three to six weeks).

Compression myelitis is suggested by the history or presence of spinal caries, traumatism or new growths, aneurysms, syphilis, echinococcus or cysticercus cysts or syphilitic infection, combined with the symptoms of a slowly developing myelitis, persistent and perhaps agonizing pain if the nerve roots be involved (malignant growths and metastatic cancer causing the most severe type), and *such pain may be marked over areas anesthetic to painful*

Anesthesia dolorosa.

and thermic stimuli (anesthesia dolorosa). The relation of pain *to spinal flexion may be suggestive, as is its relief in properly treated cases of caries and deformity.* Aside from the areas directly involved spastic motor paralysis predominates, save in the lumbar region, and sensory paralysis varies with

Brown-Séquard syndrome common.

the transverse area of the lesion. The Brown-Séquard syndrome may of course be present, indicating unilateral involvement. This symptom-complex is more common here than in any other spinal cord lesion save hematomyelia.

Determining factors.

Primary Chronic Myelitis.—The only essential factors are its slow and irregular development, affecting at random the different cord functions, motor, sensory and trophic, but lacking the nerve root symptoms of compression myelitis.

COURSE.—As any region may be involved and any type or grade exist, the course varies from early death to complete recovery or the establishment of chronic myelitis and permanent deformities. *Sensation*, last to go, is first to return. In unfavorable cases *bed sores* may hasten the end, sometimes being associated with *hyperpyrexia;* descending and ascending sclerosis may occur; the legs may become rigid or the seat of flexor or extensor spasm (*i.e.*, "*jack-knife rigidity*"), and the *reflex responses extreme. Urinary decomposition* may produce *cystitis or pyelitis* and hasten the end.

Chronic.

"Jack-knife rigidity."

It should be remembered that cases of slow onset and a history of antecedent acute infectious disease yield the best prognosis. Bed sores, high degrees of anesthesia, sudden severe onset and an unusual degree of early pain and spasm are bad omens.

Good habits, relative youth, a vigorous physique, and an absence of tuberculous or malignant disease offer some encouragement. In chronic cases, after two years no hope of recovery may be entertained.

LANDRY'S PARALYSIS.—This rare disease of unknown causation chiefly affects *young adult males* and is characterized by a *progressive symmetric ascending paralysis*, and an abrupt onset suggesting an acute infection.

Symptoms.—*The sharply febrile onset is succeeded almost immediately by a complete paralysis beginning in the legs and involving in a few days or even hours the trunk, upper extremities, muscles of respiration and usually of deglutition and articulation. Enlargement of the spleen may be noted, the sphincters are usually not involved, the reflexes are lost, but sensation is ordinarily preserved, or but slightly affected, and the muscles do not atrophy or show abnormal electrical reactions unless the ailment is of unusually long duration.*

Ascending paralysis.

Sensation preserved.

Atrophy and R.D. usually absent

Prognosis.—Death in from forty-eight hours to two weeks is the almost invariable result.* Nothing definite can be said of the pathologic changes save that the lower motor *neurons* are the point of attack. *Acute myelitis* shows anesthesia, bed sores, reaction of degeneration and atrophy at the level of the lesion and early *involvement of sphincters. Multiple neuritis* cannot be differentiated positively in certain rare instances (see "multiple neuritis").

PROGRESSIVE MUSCULAR ATROPHY

Definition.—This is a disease of unknown causation, affecting chiefly adult males beyond the age of thirty, said to be influenced etiologically by the usual factors, such as traumatism, excessive mental or physical strain, exposure to wet or to cold, and, to a slight degree, hereditary influence.

Adult males

Usual factors.

* The author very foolishly and quite vainly kept a young girl alive for forty-one days by artificial respiration, breathing having ceased early in the attack. Muscle atrophy was well marked only in the last stages. The patient ate and slept during this period, but if artificial respiration was suspended during sleep, a wave of intense cyanosis would immediately follow and the girl would wake as it deepened with a look of utter terror, unable to utter a sound, and feeling the pangs of dissolution. Upon resumption of the movements her cyanosis would almost wholly disappear. She died very suddenly of acute pulmonary edema on the forty-second day.

It is essentially a gradual atrophy of the motor neurons, either lower or upper being first involved, the lower in the greater degree and sometimes exclusively.

The degenerative process affects the gray matter of the anterior horns and the fibers of the anterior nerve roots within and without the cord, together with the intermuscular branches. The degeneration of the gray matter extends to the medulla and the antero-lateral white tracts and lateral pyramidal tracts are affected in varying degree, in some cases even to the motor cortex. There is of course marked muscular atrophy.

Symptoms of the Aran-Duchenne Type.—*Following a period of vague and indefinite pseudo-rheumatic pain, muscular atrophies appear in the following order. The muscles of the ball of the thumb* (the patient first noticing an inability to adduct the thumb, separate the index from the middle finger, write, button his clothes), *the interossei and lumbricales, forearm flexors* and *extensors and the deltoid*, followed by other muscles of the shoulder girdle group. One hand is usually first affected and the muscles of the legs and face and the trapezius are usually attacked late. The platysma myoides *entirely escapes and indeed may hypertrophy, and fibrillary contractions are marked.*

Order of involvement.

Fibrillary contractions.

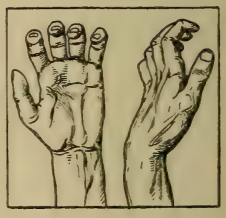


Fig. 622.—Claw hand.

The atrophy of the muscles of the hand and forearm produces the so-called "*claw hand*" and the wasting of intercostal and abdominal muscles reduces the patient to a helpless skeleton. Later the neck and even in some instances those of the face are involved and in prolonged cases those of the legs. The electric excitability of the muscles is early diminished and finally lost, the reaction of degeneration occurring in rapidly advancing cases. *Muscular power disappears pari passu* with the wasting. *Subjective sensory symptoms* may be present yet sensation is unimpaired and the sphincters unaffected, but the knee-jerks are lost *when the quadriceps extensor atrophies.*

Sensation unaffected

Knee jerks.

Amyotrophic Lateral Sclerosis.—*In certain cases of a progressive muscular atrophy described as amyotrophic lateral sclerosis* a primarily spastic paralysis is associated with atrophy following the Aran-Duchenne order quite closely. In these the reflexes are increased and the picture is one of spastic paraplegia with the typical gait but with gradual atrophy. *Jaw clonus* and Babinski's sign are present but the sphincters are unaffected. The tendon reflexes are seldom increased in the flaccid type, markedly increased in the amyotrophic type. *Fibrillary contraction* is marked and there is a generally exaggerated motor response to muscle or tendon percussion, yet there is usually a partial reaction of degeneration. Bulbar involvement is often early and may precede the leg involvement.

Fibrillary contraction.

The Bulbar Type (*Glosso-labio-laryngeal Paralysis*).—*Bulbar symptoms may precede or follow spinal symptoms but no actual pathological distinction exists to justify making of the primary bulbar type a separate disease. Bulbar involvement is indicated by difficulty in the pronunciation of the linguals and dentals, atrophy and weakness of the tongue leading to dysphagia, dysarthria,*

Characterist symtom

the accumulation of saliva and drooling. With involvement of the lips, labial pronunciation becomes difficult and succeeding paralyses of the pharyngeal and laryngeal muscles increase the dysphagia and enfeeble the voice. This type may be associated with progressive atrophy of either the spastic or flaccid type and cases of acute onset occasionally occur. A similar condition affecting the eye muscles is known as *progressive ophthalmoplegia.* Acute form.

Muscular Atrophy of the Peroneal Type.*—*This differs from the preceding form in three particulars viz., first, that it is an hereditary or family disease.*

Second, in that the atrophy commences in the muscles of the feet and peroneal group, often unilaterally at first, reversing the usual form.

Third, sensory disturbances are prominent; it especially affects young children, leading to various forms of club-foot, and the sphincters are not involved.

Differential Diagnosis.—The chief difficulty arises in distinguishing between a progressive muscular atrophy of the spastic type and syringomyelia, differentiation being only possible when the peculiar sensory symptoms of the latter disease are pronounced.

The bulbar type must be distinguished from the *pseudo-bulbar palsy* of cortical origin which results from bilateral lesions in the facial area of the cortex or between these areas and the cranial nuclei, *and lacks the fibrillary tremor, rapidly advancing atrophy and R.D. of bulbar palsy.*

A *"Werdnig-Hoffmann Type"* of infantile, familial, central, muscular atrophy shows its primary effect in the muscles of the back, thighs, and pelvis.

Gower's Type.—The atrophy is limited to the muscles below the knee.

Eichhorst's Type.—In this the *femoral* and *tibial* muscles are first affected.

Zimmerlin's Type.—The initial atrophy affects the proximal muscles primarily of the thorax and arm, etc.

MUSCULAR DYSTROPHIES.*—There are various clinical forms of muscular wasting, with or without initial hypertrophy, classed under diseases of the nervous system but lacking definite pathologic findings. Atrophy, palsy and marked hereditary tendency usually co-exist in all forms.* No pathology.

Etiology.—The disease may exist in a family for generations and is most frequent in the male. Atavism similar to that of hemophilia is evident, the mother transmitting the disease even though herself unaffected. Heredity.

Classification.—No sharp dividing line can be drawn, but for clinical purposes cases may be divided into (*A*) *those occurring in youths and adults,* (*B*) *those of childhood.* Two classes.

Class (A).—The disease may assume (1) *atrophic and* (2) *hypertrophic forms, the former including* (*a*) *cases without involvement of the facial muscles,* (*b*) *those in which they are early involved.* The latter includes both cases of genuine hypertrophy and those of muscle lipomatosis.

PSEUDO-MUSCULAR HYPERTROPHY.—(*Progressive Muscular Dystrophy*).—**Symptoms.**—*The mechanical motor impairment calls attention to apparent enlargement of the muscles, first usually of the calves.* The leg ex-

* The tendency to multiply these diseases by the discovery of more or less unimportant variants is indicated by the fact that this might be called the "Marie-Tooth-Dejerine Sottas type."

Prominent weak muscles.

tensors, glutei, the lumbar muscles, the deltoid, the triceps and infraspinatus are excessively involved and become prominent and resistant yet *weakness co-exists and is progressive.** Placed upon the floor the patient passes through a succession of characteristic movements to regain an erect posture.

Posture.

When standing there is lordosis, the shoulders are thrown back, belly prominent and feet widely separated. The shoulder girdle, being greatly weakened and extremely movable, makes the arms abnormally long and if an attempt is made to raise the child by catching him under the armpits, one feels that the body is slipping down between the shoulders.

Lifting test.

Terminal stage.

The terminal picture is that of extreme atrophy and helplessness without fibrillary twitching or R. D. but associated with mental degeneration.

Duration of the disease is from a year to two decades or more, death occurring from intercurrent disease or inanition, and prolonged remissions may occur.

Scapulo-humeral Muscular Atrophies.—The chief difficulty in diagnosis arises in differentiating between certain cases of the disease which present overlapping symptoms, it being sometimes impossible to assign a case definitely to a single division.

Tapir mouth.

In the facial type *the first changes appear in the muscles of the face, particularly the orbicularis oris and extend to the muscles of the cheeks and forehead, affecting both expression and speech and producing what is known as the "tapir mouth." The extension to the general muscular system is downward.*

Erb's type

Erb's juvenile muscular dystrophy is charaterized by being distinctly a disease of youth, having its incidence usually at the age of puberty and within the age limit of twelve and twenty years (twelve to sixteen usually) and the fact that the muscles first involved are those of the shoulder girdle. In all forms late contractures are, of course, common, with the usual deformities. Fibrillary contraction of muscle, reaction of degeneration, disturbance of sensation and reflexes are lacking until extreme atrophy and weakness have developed.

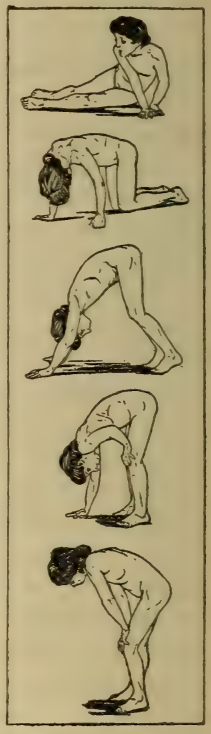


FIG. 623.—Characteristic postural changes in pseudo-hypertrophic paralysis accompanying act of rising from floor. (*After Gowers and Starr.*)

MYOTONIA (Thomsen's Disease).—This is a disease of childhood, of the familial type, affecting chiefly the male, and very rare in this country. Occasionally cases of the acquired type may be encountered.

Peculiar complex.

Symptoms.—The characteristic symptom is a peculiar slowness of muscular contraction and relaxation, most marked in prehension and locomotion. The hand closes slowly and awkwardly around the object, and relaxation is equally deliberate. So in walking, the leg is slowly advanced and halts for a second or two, yet the awkward gait lasts for but a few steps.

*In many of the muscles the enlargement is due merely to peculiar fatty deposits.

The laryngeal, ocular and facial muscles are rarely affected, the general musculature is well or unusually developed and out of proportion to the actual strength. As might be expected the electrical reaction of the involved muscles is deliberate, persistent, and often associated with vermicular contractions between the poles. The disease is essentially chronic and incurable.

Erb's myotonic reaction.

MYASTHENIA GRAVIS (*Asthenic Bulbar Paralysis. Erb-Goldflam Syndrome*).—This is a progressive loss of muscular power associated with rapid exhaustion under interrupted tetanizing faradic stimulation though not under galvanic (*Jolly's "myasthenic reaction"*). The loss of power affects first the muscles of the eye, face, jaws and neck, but may involve the entire body. The essential symptom is the rapid fatigue of the affected muscles, restored by rest, and this in connection with the peculiar electrical reaction, the transient nature of the symptoms, simulating paralysis, and the absence of true atrophy or fibrillary contractions makes the diagnosis easy. The disease is essentially chronic, but the mortality probably exceeds 50 per cent.

"Myasthenic reaction."

Simulates bulbar paralysis.

NEURITIS

Etiology.—*Inflammation of the nerves may be single or multiple and due to toxemia, extension of inflammation, actual germ invasion (leprosy), traumatism involving wounds, tears, concussion and torsion, pressure, and finally, to cold.*

Infection, toxemia, traumatism.

The toxemias, whether due to mineral poisoning or acute and chronic infections, are often associated with polyneuritis, the acute infectious diseases most frequently observed being diphtheria, erysipelas and leprosy, and of minerals, lead, arsenic and mercury. Beri-beri is an acute disease of which polyneuritis is usually the predominant symptom.

Fatigue with chilling or prolonged exposure to cold and wet is a common cause of both local and poly-neuritis; alcohol is also a potent etiologic factor.

Fatigue and chilling.

Alcohol.

Symptoms.—*Whether the neuritis be local or general, the symptoms are essentially the same, the differences being those of anatomic distribution.*

Pain is severe, boring or stabbing, and is associated with **tenderness on pressure.** *It is usually most marked at the terminal points, but may be felt along the whole course of the nerve. Edema and congestion may be present over the same area, muscular power is impaired, motion is painful.*

Pain.

Tenderness.

Tactile sense is impaired or lost, formication and numbness are common, and actual paralysis follows, as does muscular atrophy, the skin becoming shiny, and the nails brittle and deformed.

Trophic changes and reflexes.

Where the larger trunks are involved the reflexes may be lost and the paralysis be typically flaccid. It must be remembered that all degrees of inflammation are encountered as is illustrated by the behavior of the muscles in relation to the electric current. In some cases reaction of degeneration is complete and prompt; in others the response is normal.

Severity varies greatly.

"Neuritis" has become of late almost as much the mode as "lithemia" was a decade or two ago, but the greatest care should be observed in differentiating at

Caution.

least the pains of recurrent minor angina and the many cases of deltoid "bursitis" both of which, the latter especially, are mistakenly called neuritis in many instances.

Multiple neuritis.

ACUTE FEBRILE POLYNEURITIS.—This is merely a *primary* multiple neuritis due to an unknown virus characterized by an acute febrile onset exactly simulating that of the acute infectious diseases, the temperature being high as a rule.

Manner and extent of involvement.

Subsequently a typical neuritis symptom-complex develops, taking the form usually of an *ascending paralysis* of varying degree, most often commencing in the feet, less often in the arms. *It may be limited to the extremities or affect the whole body even to the involvement of the diaphragm and other respiratory muscles.*

Prognosis.

Death is common in the severe forms, but as a rule the patient recovers after an illness of a month or six weeks. Residual partial paralyses are frequent, but seldom persist for more than a year.

Cases of this acute febrile type occurred with considerable frequency during the Great War just passed and two distinct types of onset were observed.

In one the paralysis was preceded by an initial febrile attack of brief duration, in the other paralysis was the first symptom.

In the former type an interval of from five to ten days elapsed between the initial illness and the onset of the paralysis.

The cortex is spared and the virus expends its virulence upon the spinal ganglia, the cord and the peripheral nerves.

Recurrent Multiple Neuritis.—It would hardly seem that a separate place should at present be given to a polyneuritis distinguished only by a marked tendency to recurrence.

Foot- and wrist-drop.

Neuritis due to Alcohol, Lead or Arsenic.—*In this form the onset is usually gradual and preceded by numbness, tingling and neuralgic pain. "Foot-drop" and "wrist-drop" are common features, giving rise in the former case to the prancing or steppage gait.*

Prancing gait, in peroneal type.

In alcoholic cases mental symptoms may be present, but the history of the case usually points the way to a correct etiologic diagnosis, and it should be remembered that these paralyses chiefly affect the extremities, and that *diphtheria* shows a preference for the muscles of the eye and throat, *lead* for the extensors of the arms, *arsenic* for the legs and especially the peroneal group. The distinctions are, of course, often disregarded.

Peculiarities of localization.

Diagnosis.

The diagnosis, therefore, depends upon the association of mutiple neuritis with edema or marked cardiac disturbance in connection with the known prevalence of the disease, or the past residence, or association of isolated cases.

Dislocation and crutch palsies.

PRESSURE PARALYSIS.—It is hardly necessary to do more than call attention to the various paralyses due to compression of individual nerve trunks. The common *musculo-spiral* paralysis and *circumflex* paralysis, resulting from dislocation of the humerus, are the most common. The former also may result from crutch pressure, or pressure during deep sleep, especially "*in the open,*" or from the position in which a patient is placed during a long operative procedure (see also "Brachial Plexus Paralysis").

Pressure paralyses.

VON RECKLINGHAUSEN'S DISEASE.—This interesting condition is characterized by a generalized neuro-fibromatosis, producing a remarkable variety of pressure symptoms referred to all parts of the body, patchy pigmentation of the skin and marked impairment of intellection.

A curious syndrome.

NEURALGIA.—See "Pain."

HERPES ZOSTER (*Zona*), (*Shingles*).—*This disease is characterized by violent neuralgic pain along the course of the superficial cervical, thoracic, sacral or abdominal nerves, or the branches from the Gasserian ganglion, associated with the appearance of crops of vesicles corresponding to the superficial nerve distribution and has its commonest seat over the lower thoracic of upper abdominal zone.*

Usually thoracic and unilateral.

It is commonly unilateral, but may be bilateral and is supposed to be due to an inflammation of the ganglion cells of the posterior roots.

There is no disease which causes more exquisite pain and suffering, usually of a few days' duration, but sometimes extending over long periods, and furthermore, troublesome ulceration may follow rupture and coalescence of the vesicles. The excessive severity and distinctly neuralgic or neurotic type of the pain and its distribution, together with the typical rash makes the diagnosis clear, but the pain may precede the herpes by many hours or even days and prove misleading, especially in the thoracic form.

Extreme pain.

Delayed herpes.

PERIODIC TRANSIENT PARALYSIS.—This curious ailment is strikingly *hereditary*, often traversing five or six generations, and is characterized by the *rapid onset of paralysis involving the arms and legs, or both, sparing the face*, though sometimes implicating the regions supplied by the vagus and spinal accessory. *There is no fever, a slow pulse, general diminution but rarely a loss of reflex*, deep or superficial, and *marked diminution of faradic excitability. These paralyses are transient*, lasting for only a few hours as a rule, but are strikingly *recurrent and periodic*, diurnal cases being noted. Like migraine the periodic paralysis usually ceases at the age of fifty.*

Recurrent, afebrile, transient paralysis.

Prognosis.

FACIAL HEMIATROPHY.—This incurable and obscure condition, most frequently developed in childhood, rarely in the adult, is a progressive atrophy involving all structures on one side of the face. It is peculiar in that it affects first the skin and superficial tissues, then the bone and lastly the muscles. Loss of the hair and teeth and hemiatrophy of the tongue and palate usually occur, but bilateral atrophy is rare and sensation and the electric response of the muscles is usually unaffected. It is probably not due to disease of the trigeminus and as is generally held should not be confounded with congenital asymmetry of the face. In direct contrast is unilateral facial hypertrophy in which every symptom is reversed.

Peculiar line of march.

* The transitory paresis or paralysis is strikingly like that observed in infectious anterior poliomyelitis in association with the true permanent palsies. These we now believe to be due to the edema present in the affected cord.

In view of the hereditary and neuropathic tendencies of both this ailment and angioneurotic edema, and the fact that the latter is often strikingly familial and may come and go in the same irresponsible and disorderly manner, one might hazard the statement that a connection between these conditions is suggested.

PARALYTIC VERTIGO (*Gerlier's Disease*).—This is a form of transient paralysis occurring only, so far as known, in one Swiss town.

Nuchal pain, clouded vision, paralysis or paresis of ocular nerves, unequal bilateral ptosis, muscular weakness, and vertigo are its chief symptoms.

Hiemal.

Three forms.

RAYNAUD'S DISEASE (*Symmetrical Gangrene, Local Asphyxia*).—This mysterious hiemal vasomotor affection of unknown causation assumes three forms: (1) *local asphyxia*, (2) *local syncope*, (3) *symmetrical gangrene*. Local asphyxia and local syncope in their mildest form may be seen in chilblains.

Exciting causes.

In Raynaud's disease either emotion or trivial exposure to cold, or even gastric disorders are followed by *coldness, pallor of the surface of the fingers or of both fingers and toes*. After a variable period and in irregular order the affected members become *sharply congested and perhaps livid. Stiffness and discomfort or even severe pain may accompany the swelling. Symmetrical gangrene varies greatly in degree, is rarely extensive, and* usually a persistence of the extreme stagnation of the cyanotic stage results in *superficial necrosis* involving small areas or oftentimes *but one* finger or toe. *Sensation*, often diminished in the cyanotic stage, is lost over the gangrenous area which is black, icy cold and covered with blebs. *The area of apparent involvement greatly exceeds that of actual necrosis*. The ear is involved frequently, the tip of the nose occasionally, symmetrical areas on the arms, legs or trunk rarely. Cases of rapid, extensive and fatal gangrene have been reported and occur most often in children. Aside from the local symptoms there are no constant disturbances. Hemoglobinuria, transient loss of consciousness, mental torpor and delirium are occasionally observed.*

Lividity.

Gangrene.

Necrosis limited.

Sensation.

Occasional symptoms.

Malignant cases.

Diagnosis.—*The diagnosis depends upon the occurrence of symmetric localized syncope, cyanosis or gangrene without an assignable organic cause.*

ERYTHROMELALGIA.—This rare condition unlike Raynaud's disease is essentially a summer ailment and is characterized by *superficial congestion, swelling and burning pain* in the feet or hands usually the former and worse at night, the pain being severe and increased by the dependent position. It is chronic and irregularly recurrent, often associated with headache and vertigo, and occasionally with Raynaud's disease.

Purely subjective.

ACROPARESTHESIA.—This symptom-complex is closely allied to angio-neurotic edema and is characterized by itching, burning, prickling and pain in the fingers and toes. Heat, cold and injury are apparently causative factors; constipation and gastric disorders almost constant, and occasionally there is rigidity of the fingers. It is hardly worth separate classification.

Usually trivial.

ANGIO-NEUROTIC EDEMA.—This vasomotor condition seems closely related to urticaria and, like it, is usually associated with digestive dis-

* In one case observed by the author symmetrical gangrene of the ears of the hiemal type had resisted treatment for years, but recovered promptly when the cause was found. The patient was taking large quantities of morphin by the hypodermic method, the skin over large areas of the trunk and limbs being thickened and infiltrated through the use of a dirty syringe. As withdrawal was resisted the patient was told to take the drug by the mouth, since that time there has been no recurrence of the gangrene.

turbances. Exposure to cold and emotion may be associated with it, but it often appears without apparent cause. It *is essentially a sudden and transient swelling, affecting usually the hands, feet, genitalia, or any portion of the face,* rarely the lips, tongue or even the glottis where it may produce dangerous or fatal obstruction. As in Raynaud's disease associated hemoglobinuria has been noted. *It may be periodic but is more often irregularly recurrent.* The *gastrointestinal symptoms may be severe and associated with vomiting and colic,* or, extremely slight. Osler reports a case in which the whole arm was swollen. Aside from the rare involvement of the glottis the disease is as unimportant as it is obscure.* Occasionally serious.

INTERMITTENT JOINT EFFUSIONS.—Closely allied to angio-neurotic edema is this rapidly produced but painless swelling of the joints. It lasts for only a few days, but is liable to recur.

EPILEPSY.—This *psychoneurosis* depends to an extraordinary degree upon heredity, but in predisposed individuals may apparently arise from emotional shock, acute toxemias and sometimes from remote and purely reflex causes. One variety arises from direct irritation due to injury, recent or remote, or forceps injuries in childbirth. Alternatives in heredity.

From the general etiologic viewpoint it may be (*a*) *idiopathic*, arising from no assignable cause; (*b*) *reflex;* (*c*) *toxemic;* (*d*) secondary or symptomatic; (*d*) senile or arteriosclerotic; (*e*) luetic.†

One-half the cases occur in the first five years of life and it is so rarely a primary lesion in the adult after thirty as always to excite a suspicion of syphilis, arteriosclerosis, renal inadequacy and arterial hypertension, brain tumor, or, chronic alcoholism. Age.

Sex.—The incidence is nearly equal in the two sexes. *Of inheritance* it should further be said that *it is not so much direct as it is equivalent* (*see "Alternatives in Heredity"*). Parental intemperance, for example, is an important factor, syphilis and alcoholism common and direct causes. *Attacks very similar to true epilepsy occur in arteriosclerosis,* which condition probably accounts for the cases met with in old people. Sex. Alternatives in heredity. Senile cases.

Divisions.—We speak of "*major epilepsy*" (grand mal), "*minor epilepsy*" (petit mal), "*Jacksonian epilepsy,*" and "*psychical epileptic equivalents.*"

Grand Mal.—*Auræ and various subjective sensations precede an attack.* There may be auditory, visual, gustatory and olfactory paresthesias, discomfort in the epigastrium, intestines or rectum, or peripheral sensations usually referred to the hand. *In other cases the aura is motor,* the patient turning rapidly, running for a short distance or twirling about on the toes. *The actual onset* is sudden, sometimes preceded by the so-called epileptic cry. A fall is likely to produce injury, a matter of importance in differentiating hysteria, and, *the condition is one primarily of tonic spasm,* the patient being livid and usually in a distorted attitude; *after a few seconds* follows the Auræ. Onset. Seizure.

* It strongly suggests an anaphylactic phenomenon.

† Dr. C. A. L. Reed's interesting announcement of the *Bacillus epilepticus* has not yet received confirmation.

stage of *clonic convulsions*, at first slight, but increasing rapidly in violence and affecting the ocular and facial muscles as well as those of the body and extremities. The patient froths at the mouth, is likely to bite the tongue, or pass urine or fecal matter involuntarily. *The duration of this stage is variable*, but it *seldom lasts over two or three minutes* and is followed by coma, succeeded by a deep sleep lasting for several hours if undisturbed, the patient wakening with slight mental confusion or perhaps headache. *The attacks vary greatly in frequency, usually tending to increase with the lapse of time. They may occur only at night* and the patient may be and often is, entirely unconscious of their true nature, a point to be remembered in case taking.* In the so-called "*status epilepticus*" there is fever, rapid respiration and increased pulse rate, accompanying a succession of attacks without *persistent* unconsciousness. The reflexes are sometimes absent, more often increased and accompanied by ankle clonus. *The urine if not passed during the attack* as is usual, is *passed in quantity after it. Post-epileptic paralysis and aphasia* are occasionally seen but are ordinarily transient. *In repeated seizures mental deterioration may be marked and post-convulsive mania may assume a dangerous form and become of medico-legal importance in murder cases.* So also these unfortunates may be arrested for indecent exposure due merely to mental disturbances, characterized by various automatic actions of which the patient has no consciousness or recollection.

Tongue-biting.

Incontinence.

Increasing frequency.

Nocturnal.

Transient polyuria.

Mental changes.

Innocent exhibitionism.

Petit Mal.—*This interesting condition lacks the convulsive features of epilepsy, though in a majority of instances grand mal develops, either wholly replacing or alternating with the minor attacks.* Amongst the various forms may be mentioned *simple incoherency, acts of automatism*, usually of short duration, *sudden falls, with or without transient loss of consciousness, sudden jerkings, tremor or subjective sensations of various sorts, mere interruption of conversation, and what appears to be a peculiar absent-mindedness.* During such attacks the patient is likely to turn suddenly pale, there may be fixation of the eyes and relaxation of the grasp but auræ seldom occur. All of these acts are wholly lost to the patient's recollection.

Convulsions absent.

Curious manifestations.

Jacksonian Epilepsy.—This is the reverse of petit mal, *consciousness being retained early in, or throughout, the attack and convulsive seizures present.* One must admit possible sensory equivalents but direct cerebral irritation is nearly always present. *The spasms are usually orderly and follow motor areas of the cerebrum, but may be limited to one tract such as the face and leg.* Partial epilepsy, moreover, often becomes general with the lapse of years, old injuries, tumor, meningitis, cerebritis, uremia, abscess, hemorrhage and sclerosis are the usual causes and post-hemiplegic epilepsy is of this type.

Petit mal reversed.

Motor tract spasms.

Epileptic Equivalents, Psychoses.—Hallucinations of hearing, narcolepsy (profound sleep), and, possibly, certain cases of somnambulism, seem to replace the convulsive stage of epileptic seizures, and in some instances, certain moral deficiencies are also prominent, often amounting to an entire

Peculiar and important.

* In many such cases there is nothing to mark the attack in the morning but a slight persistent injection of the facial capillaries, the patient feeling perfectly well, with unclouded mind.

change of character or the development of traits or habits abhorrent to the patient in a condition of health.*

Differential Diagnosis.—If an attack of major epilepsy be seen little doubt can be entertained as to its nature, though *only by an examination of the urine can uremic convulsions be positively differentiated. Hysteria* offers many difficulties, but is seldom accompanied by tongue-biting, involuntary passage of urine, and *never by the absolute unconsciousness presented by the epileptic.* Hysterical patients choose a safe place to fall and the movements are usually disorderly and to a certain extent purposeful and struggling rather than jerking; moreover, the duration of the hysterical attacks is longer and often promptly relieved by sharply applied pressure over the ovaries.

Uremia and hysteria.

Jacksonian epilepsy is unmistakable, though its cause may be obscure; petit mal, often beyond the range of absolute diagnosis, its recognition depending upon the coincidence of transient unconsciousness, vertigo, and perhaps automatism.

CHOREA

(*St. Vitus's Dance, Sydenham's Chorea*)

Etiology.—Within the past few years some remarkable discoveries have been made which bear quite directly upon the etiology of Sydenham's chorea.

Etiology.

Now, one can conscientiously abandon the older views and adopt that of infection without binding himself to any microörganism at present vaunted as *fons et origo malorum.*

It would seem that the true agent of infection is more likely to be found through a study of the activities of the bacteria composing the tonsillar, adenoid, and peridental bacterial flora than by attempts to recover the causative agent from tissues which yield little or no evidence of decided or specific anatomic changes.

Certainly, if one may draw inferences from personal experience the author must favor the theory of cryptogenetic focal infection, the most striking results he has encountered having followed the removal of chronically infected adenoids and tonsils.

The belief that it represents the reaction of a delicate, high strung, overworked or undernourished organism to the chronic or recurrent toxemias of this origin is wholly in accord with the author's limited experience, and apparently expresses the views held at the present time by a large number of clinical and laboratory workers.†

* The author finds it difficult to credit the assertion so frequently made that either somnambulism or "night terrors" should be regarded as epileptic equivalents. Certainly a host of children, who pass through phases of night terrors and somnambulism alike, show absolutely no signs of either epilepsy or its "equivalents" in adult life. He also feels that the statement, recently encountered, that sudden attacks of angina-pectoris, asthma, actual syncope and the like may be so classified, merely because no readily demonstrable cause is apparent, is wrong. Nearly all of these are either direct or reflex circulatory phenomena. Etiologically, epilepsy is itself a mystery, and can hardly claim the right to take over phenomena so readily explainable on the basis of known circulatory mechanics.

† D. J. McCarthy in a very scholarly, conservative and convincing article in Osler's "Modern Medicine," defines it as "due to the effect of an infectious agent or its toxin."

Rheumatism.

The clinical relationship of rheumatism, rheumatic endocarditis and pericarditis to chorea is too intimate to be dismissed and the concurrences too common, but they need not force one lightly to as yet accept the *Micrococcus rheumaticus* as the cause and chorea itself as "cerebral rheumatism."

Precocious children.

Unwise parents.

Inherited vulnerability may then prove to be the explanation of the assumed but ill-proven "hereditary tendency." Precocious, delicate, excitable children are peculiarly liable to the disease, especially if, as is so often the case, they are pushed ahead in their studies or encouraged to develop unusual mental traits at an unduly early age.

Secondary factors.

Overforcing and depressed nutrition are the chief predisposing factors in chorea, and the elements of shock, worry, grief, etc., are merely important as tending to increase preëxisting vulnerability.

Seizures due to reflex *irritation* are rarely seen but may occur in connection with various conditions affecting the gastrointestinal tract, sexual organs and yet oftener as a result of *naso-pharyngeal adenoid growths.**

Age and sex.

The disease predominates in females between the ages of five and fifteen, is less often seen in the well-to-do, and bears an interesting relationship to rheumatism and a yet more direct one to heart disease.

Heart disease.

It is certain that about two-thirds of the active cases *show valvular murmurs* and that practically all of the fatal cases show actual organic valvular lesions, chiefly of the mitral type, but it is by no means proven or even rendered probable that either embolism or heart disease is the cause of chorea.

A clean-cut clinical picture.

Symptoms.—*Involuntary, incoördinate, characteristically jerky movements, unilateral or bilateral, localized or general, and associated to a varying degree with excitability and emotional disturbances, characterize chorea.*

Though difficult to describe, it is never forgotten when once seen, nor does it offer any difficulties to the diagnostician save in its rarest forms. Accord-

Varieties.

ing to its severity, we recognize a *mild and severe form,* to which may be added a *third, the chorea,* associated with delirium (maniacal).

Premonitory symptoms take the form of *increased nervous irritability,* perhaps *unusual wilfulness,* and a *general restlessness.* As the irregular

Misunderstood clumsiness and "badness."

movements begin, the apparent clumsiness of a child, associated perhaps with unusual disobedience, may bring about undeserved punishment which may precipitate the attack. *The face and arms are most commonly involved,* but in severe cases the whole body may participate. *Disturbance of speech*

Speech, gait.

occurs in about one-fourth of the cases, varying greatly in degree *and the facial grimaces and jerking, incoördinate and bizarre movements of the extremities involved are absolutely distinctive.* If the legs be affected, a peculiar *jerky hitching gait* appears, and there may be motor weakness in the leg or arm. Occasionally there is crossed chorea, still more rarely one encounters

Rare mental symptoms.

the most severe type, maniacal chorea. *Mental disturbances of marked degree are unusual* yet intervals of delirium or temporary hallucinations sometimes occur. Practically all of the other associated symptoms would seem to be dependent upon rheumatism and endocarditis as concurrent

Heart signs.

factors. *The heart symptoms* are chiefly those of functional or organic mur-

*Probably because of the double factor of chronic irritation and toxemia.

murs, the latter being present in about 50 per cent. of all cases and two-thirds showing a murmur of some sort. The ordinary *hemic murmur* with its maximum at the second pulmonary interspace is common, but of little moment, *and other functional murmurs may be associated with the accelerated heart action almost invariably present. The common murmur is systolic and apical* and the differentiation between the functional and organic lesion must depend upon the application of the usual diagnostic rules. *Evidence of secondary dilatation and the presence of a loud harsh well-conducted murmur indicate an organic mitral lesion.* Arsenical pigmentation may occur,* but fever, purpura, urticaria and joint symptoms have probably no direct relation to the disease itself, but rather to the underlying causative infection and toxemia with which it is so commonly associated. Murmurs. Cutaneous signs.

Course.—Although showing a distinct tendency to recurrence (50 per cent.), the individual attacks seldom last over two or three months, though rarely they may extend over years, particularly the slighter manifestations. The death rate during the attacks is almost negligible.

DIFFERENTIAL DIAGNOSIS.—It should be remembered that *choreiform movements* are increased by excitement or obstruction, *entirely cease during sleep*, whether natural or produced by drugs, *and, that neither sensibility nor, usually, reflex activity is affected.* **Chorea electrica** is merely a variant of chorea proper, characterized by lightning-like movements.

Huntington's chorea *is hereditary, appears usually during the third decade and is characteristically constant and progressive. Mental deterioration is marked and it usually progresses to terminal dementia. The movements themselves are slower, the gait more swaying and the disease should have a single descriptive name.*† Hereditary chorea.

Friedreich's Ataxia.—*Nystagmus, scanning speech, scoliosis, talipes, the slow movements and clear evidence of heredity make the diagnosis clear.*

Hysteria.—*Rarely, cases may exactly simulate chorea, but usually the movements are less jerky and irregular, more rhythmic and there are associated stigmata which make the diagnosis easy. "Rhythmic chorea" of hysterical origin produces orderly movements of the muscle groups involved, for example, the salaam convulsion.*

CONVULSIVE TIC (*Habit Spasm*).—This usually takes the form of localized facial spasm associated with head movements or the raising of a shoulder, though often the spasm affects only the eye or the mouth, or more commonly both. It is a somewhat common, transitory and unimportant condition.

GENERALIZED IMPULSIVE TIC (*Gilles de la Tourette Disease*).—*This is characterized by involuntary facial, brachial or general muscular movements, often violent, in association with marked disturbances of speech and sometimes of mentality. Articulation is explosive even to incoherence, mimicry may be present, usually in the form of repeated utterance of words heard or of actions* Mimicry.

* If that drug is pushed strongly over long periods.

† It is variously described as "megrim," "megrum," "senile," "chronic progressive," "adult hereditary" and "chronic" chorea.

seen, or, the frequent repetition of obscene words may accompany the spasm. The *mental disturbances are similar, taking the form of obsessions or fixed ideas. Every action may be associated with an impulse to count to a certain number or to touch a certain object.*

PANDEMIC CHOREA.—This group of diseases, more closely allied to hysteria than chorea, is chiefly of historic interest, although rare instances are seen at the present day. The tarantella of the composer was suggested by the remarkable religious manifestations epidemic during the middle ages. These were characterized by the wildest excitement, violent gesticulation, and dancing and leaping to the point of exhaustion.

The "tarantella."

SALTATORY SPASM.—This is a transient or chronic condition characterized by *sudden violent contraction of the leg muscles upon any attempt to stand.* Marked mimicry of words and actions may be present, and heredity plays a part.

PARALYSIS AGITANS (*Shaking Palsy, Parkinson's Disease*).—Occurring more frequently in men than in women and seldom under the age of forty this disease possesses no definite etiologic factors, nor has it any characteristic pathology unless it be a premature senility of a cerebrospinal type.

Symptoms.—The chief symptoms are *tremor, rigidity, weakness and a peculiar attitude and gait.* The disease, though insidious, is easily recognized when fully developed. The *tremor* chiefly affects the hands and feet, thumbs and forefingers showing the so-called "pill rolling" motion; the toes are less involved, the chief tremor in the lower extremities being in the ankle-joint, and *head nodding*, if present is usually vertical, more rarely rotatory. Emotion increases tremor; voluntary movement may or may not check it, and it is absent during sleep. *Rigidity* is shown in slow and awkward movements, and weakness may be the earliest symptom but often appears only after exertion. *The face is immovable and expressionless, the eyebrows raised, and the attitude and gait* become characteristic as the disease advances. The *patient stoops with bent head, with the arms* held away from the body and *flexed at the elbow* and the fingers bent. The *voice is high-pitched* and *the speech* curiously hurried though perhaps primarily hesitating. In walking *festination* is observed, the stooping patient apparently trying to overtake his center of gravity (Trousseau) and if thrust backward the same short hurried steps are taken and the patient will fall if not supported.

Bread crumbling tremor.

Rigidity.

The mask-like face.

Attitude, voice and speech.

Festinant gait.

Propulsion and retropulsion.

Diagnosis.—The developed disease is unmistakable and a diagnosis may be made on the basis of rigidity, weakness and attitude alone. It could be confounded only with post-hemiplegic tremor from which the history will usually at once distinguish it.

HYSTERIA.—*The fact that one is dealing with a genuine, deep-seated, though inorganic pathological state should never be forgotten in relation to hysteria, and further, that it is equalled only by syphilis in the protean nature of its manifestations.*

A disease.

It is perhaps unfortunate that a field of medicine which has shown such wonderful results from scientific experiment and painstaking rational inves-

tigation should still retain a problem such as "hysteria" to keep alive in the domain of *etiology* the purely speculative and theoretic method.

The great number of theories, no less than their extreme subtlety, complexity and inconclusiveness, make any attempt to consider or discuss them in a book of this kind wholly futile. Furthermore, as Jeliffe says, "at the present time there are hardly two neurologists agreed as to what shall be considered as stigmata or essential features of the hysterical phenomena."*

"Another feature concerning the revaluation of symptoms and one of the most difficult to adequately present, is *simulation*. Unconscious simulation, if there be such, and aggravation are not included here. These are features of what must be termed the hysterical mentality. Simulation as here restricted is probably rare, yet it is constant and serves to discourage careful study of many deserving patients."

"*Before attempting a systematic presentation of the symptoms of the hysterical reaction a few remarks may be made regarding the value of this motley collection of observations in which real fragments are mingled with those born of credulity and mental laziness. The golden period of hysteria which reached such a high point as a result of the stimulus of the Charcot teachings has been followed by one of analysis in which the careful sifting of the enormous material has become an imperative necessity. One of the first striking facts of this reëxamination is the astonishing frequency of mistakes in diagnosis.*"

"Thus, the tubercle bacillus has shown the real character of many heretofore diagnosed hysterical hemoptyses; gastric chemistry has similarly relegated many intestinal disorders into their proper position; microscopic and cryoscopic methods enable one to determine the essential features of supposed hysterical hematurias; methods of blood examination have reduced the number of hysterical fevers almost to a minimum."

"In the field of the paralyses, hemiplegias, monoplegias, paraplegias, etc., mistakes have been especially frequent. With the newer signs of organic involvement of the nervous system many organic disorders are recognized which, heretofore, were called hysterical, and are even called so at the present time in their early stages. Literally thousands of sick individuals suffer from complaints which a lazy diagnosis dubs hysteria. The falsity would be revealed by a searching and intelligent analysis."

"One knows that for Janet, anesthesias, amnesias, abulias, paralyses and changes of character represent the stigmata, while under the head of accidents, he groups hysterical attacks, somnambulism, subconscious acts, and fixed ideas, whereas Babinski claims that careful examination shows that anesthesia never exists, and between these one finds all possible variations. Some authors lay great stress on restriction of the visual fields, others practically deny its existence."

"Inasmuch as anatomy teaches the absolute interrelation of nervous structures within the entire human body, it is an idle distinction to say that hysteria is a psychosis or a neurosis. Splitting the difference by calling it a

* The attitude of Dr. Jeliffe is so rational and his statements so illumining that further quotation seems justifiable.

psychoneurosis simply implies that the nervous adjustments of the different parts of the body, one to another, as well as the adjustment of the individual as a whole to his environment, show various types of disturbances called hysterical symptoms."

"Hysteria is a general tendency to certain reactive expressions. The difficulty in description is an evidence of the instability of the concept, its width, and its fluctuating outlines. There lies in every person the possibility of the hysterical reaction, and it all depends either on the stimulus or the change in the resistance to bring it out." *

The clinical field of hysteric phenomena is steadily narrowing as accuracy in diagnosis increases.

There can be no doubt that many who are termed "hysterics" are merely grown-up "mother's darlings" and "papa's pets" and, too often, the mother or father is of the same basic temperamental type and hereditary influences must be considered.

Heredity and early training.

One cannot doubt the possibility of inherited emotional instability or lability and in the tainted individuals who are so exquisitely sensitive that any reproof brings floods of tears, whose passionate outbursts seem to be hung on hair-triggers, but who turn quickly from anger to passionate self-reproach and assuage by impassioned bursts of affection the hurt they may causelessly have inflicted, are very likely to be treated as special creations set apart from and above wholesome discipline.

Harmful sympathy.

In many families, mother, father and the more stable and unselfish brothers or sisters are made slaves to the whims, caprices, and childish moods of the tainted family member, and instead of wholesome discipline there is found the constant but futile endeavor to avoid offense and to substitute harmful sympathy for wholesome correction.

Heredity and early training, or rather the lack of it, are extremely important factors in hysteria, for wholesome discipline and wise control go far to counteract congenital faults of temperament. Indulgence of whims, the development of the emotional side by excessive sympathy, the failure to demand of the child or young adult self-control and consideration for others are factors that can seldom fail to create an hysterical temperament, even if the body is not relatively feeble, the intellect precocious and the neuropathic hereditary taint marked.

In such persons psychic instability, intense, yet variable, over-reaction to stimuli, lessened will power, and increased suggestibility usually are evident early.

On the other hand, the condition, when once developed, must not be regarded as one depending upon the unassisted volition of the individual, or wholly subject to his or her control, but rather as an actual acquired psychoneurosis to be ameliorated, and perhaps removed, by suggestion and a complete control which, though kind, is *firm, persistent, insistent and lacking in every element that enters into the upbuilding of the hysterical temperament.*

Proper treatment.

With this must be combined the correction of actual organic disorders and the upbuilding of the patient's nutrition when this is lowered.

* Osler's Modern Medicine, Vol. V, 1915.

Age, Sex and Race.—The disease occurs for the most part between the ages of fifteen and twenty, and almost exclusively in women, cases of hysteria in the male being the rare exception *in this country*. It rarely occurs in children under ten and adults over sixty. Southern races yield many examples as compared with the more Northern.

Young women chiefly.

Relation to Disease.—*It is found in combination with and apparently as a result of actual disease far more frequently than is usually supposed, malnutrition and anemia, lead and arsenic poisoning, unrecognized dyspepsias, gastric or intestinal, reflex irritation, naso-pharyngeal obstruction, or diseases of the pelvic organs, movable kidney, chronic appendicitis and unrecognized gastric ulcer being some of the exciting causes encountered by the author.*

Often associated with organic ailments.

We generally err about as often one way as the other, failing on the one hand to recognize an underlying organic cause in certain cases, and on the other construing pure hysteria as the organic disease which it simulates.

Furthermore, actual malingering or simulation is not a rare cause of mistaken diagnosis.

Sexual excess or sudden deprivation in certain instances is most important, though the hysterical woman is usually sexually indifferent and the genesic sense *may* be abolished through anesthesia of the vagina and clitoris.

Sexual factors.

Symptoms.—These may be present in almost any portion of the body and consequently almost any disease may be simulated. *Motor symptoms* may be paralytic, paretic or spasmodic. *Paraplegia and monoplegia* are more common than *hemiplegia*. *Paralysis is usually* incomplete, muscles may show disuse atrophy, *but the electrical reactions are normal*, the knee-jerks seldom diminished, *usually increased* and equal, and a paralyzed leg is usually dragged limply along. *Urinary retention may be seen*, true *incontinence never*, true ankle clonus is absent, pseudo-clonus sometimes present; *aphonia* is frequent but the paralysis is seldom complete. *Ptosis* is usually double, often with an overactive orbicularis, but the muscles of the eyeball are seldom affected, though conjugate deviation *rarely* occurs. *Both tonic and clonic spasm* may occur, particularly after injuries and both paralysis and spasm may be associated with *anesthesia*. *Pseudo-contractures, persisting during sleep but disappearing under general anesthesia*, and fine *tremor* or regular and irregular muscular contractions and even edema are encountered. *Hystero-epileptic* seizures are common among the Southern races, but their differentiation is seldom difficult, though their various phases (*i.e.*, prodromal, epileptoid, clownism, passional, delirious), may vary greatly in intensity or appear in an isolated form. *Hyperesthesia* is especially marked in the hysteric zones, *i.e., the hypogastrium and lower spinal* and *inframammary regions*, but it may be *visual, auditory or olfactory*. *Anesthesia and more often, analgesia* may be associated with loss of response to heat and cold and impaired muscle sense.

Hysteric stigmata.

Contractions rather than contractures.

Both motor and sensory paralyses affect the left more often than the right side, but sensory symptoms may involve the entire body, including the mucous membranes and in hemianesthesia they are characteristically limited by a sharply defined border representing the median line of the body. The stocking, garter, glove, sleeve, or drawers' leg forms are seen, as are irregular or disseminated

Some form of anesthesia seldom absent.

islets of anesthesia conforming to neither nerve nor segment distribution while they may capriciously shift their seat or boundaries after a seizure or without apparent cause. They may also take the form of *concentrically* contracted visual fields, usually unequal and *sometimes* unilateral, or of *hysterical color vision. The loss or perversion of special senses* and *loss of sensation is actual and not imaginary in all true hysteria*, and the patient is usually unconscious of the latter. Absolute blindness or deafness is rare and monocular diplopia is a rare symptom. Mental symptoms range from mere hysterical laughing and crying, to amnesia, loss of will power, impressionability, somnambulism, and even the simulation of actual insanity. The pharyngeal reflex is lost in 90 per cent. of the cases of hysteria as is frequently the plantar reflex. *Auto-hypnotism* and *catalepsy* are among the rare conditions, the reflexes being lost in the latter condition.

A difficult problem.

Diagnosis.—Most cases are evident to any well-trained clinician, but in those simulating organic disease of the spine or cord some difficulty may arise. The *normal or increased knee-jerk, absence of urinary incontinence and true ankle clonus*, the *normal electrical reactions*, absence of true atrophy, and the *active resistance* of spasmodic contractures, *i.e.*, the evident active resistance offered the examiner's hand, are quite unlike true spastic contraction. *The onset of symptoms is, moreover, generally coincident with some violent emotion, shock, or injury.* The disappearance of contractures during anesthesia or sound sleep is also important and the minor seizures may instantly disappear under sharp ovarian (hypogastric) or supraorbital pressure. Disseminated sclerosis presents oftentimes a strong resemblance, but in that disease tremor is absent or markedly lessened when the patient is at rest, whereas it is continuous in hysteria.

Prognosis.—The prognosis in any case depends upon two factors: the ability to undergo a proper course of treatment and the physician's success in obtaining the coöperation of sensible and intelligent family members.

In many instances surgical or medical treatment directed to the cure of underlying organic disease or the removal of reflex irritation completely removes the symptoms, but, in many more, unwise surgery creates havoc.

TRAUMATIC HYSTERIA.—(The Traumatic Neuroses of Oppenheim) (*"Railroad Spine," "Railroad Brain"*).—No more difficult task presents itself to the physician than that involved in an opinion regarding the effect of injuries as represented by purely subjective or indeterminate objective symptoms and too often involving a large element of unconscious or deliberate deception. To do justice alike to an injured individual and the harassed and much bled corporation is at times well nigh impossible.

Important Features of Traumatic Cases.—Aside from out-and-out malingering one may encounter one of four reasonably distinct *clinical* types, viz.: (1) *Those in whom a pure nervous depression is manifest.* (2) *Individuals markedly and genuinely hysterical.* (3) *Cases exactly resembling the first two groups, but developing after a variable period actual disease of the brain or cord.* (4) *Cases of manifest injury.*

Psychically Depressed Type.—This differs in no particular from ordinary mental depression save that the morbid introspection relates to the accident and its supposed effects. Clinical types.

Hysteric Type.—This is merely a psychasthenia* with hysteric stigmata added, the emotional side being pronounced. Psychasthenic and hysteric.

Symptoms Commonly Presented.—Headache, insomnia, vertigo, subjective weakness, mental and nervous irritability and instability are marked, mental depression is common and may attain actual melancholia. Pain in the back is almost invariable, the reflexes are increased and the impaired digestion and lack of appetite, together with the mental depression and anxious, drawn countenance, often lend a misleading facial element of suffering, emaciation and pallor. The tongue is usually coated and constipation is common. Numbness and tingling may be present in either the hysteric, or, using the word in its literal sense, the psychasthenic types, but in the former may co-exist with either a mere limitation of the visual fields or an achromatopsia. A marked hysteric or emotional tremor is not uncommon. The pupils are often dilated and may be slightly unequal. Physiognomy. Paresthesias. Tremor. Pupils.

Circulatory disturbances are common and tachycardia, marked cardiac irritability, arrhythmia, and vasomotor disturbances often persist for long periods. Subjective sensations of heat and cold, flushing, excessive perspiration or recurrent sweats, variations in the urinary secretion and the like frequently occur. Menorrhagia, amenorrhea, sexual psychasthenia and loss of memory may be added to the long list of symptoms usually representing purely functional disturbances. Heart.

From personal observation the author believes that no line can be drawn between "traumatic neurosis" and traumatic psychic depression or hysteria, for while at times cases arise that fit the syndrome first named, in many others there is a blend of signs that hopelessly blurs any sharply drawn boundaries and the element of deliberate or unconscious exaggeration too often fostered by alarmist opinions and vague theories on the part of the oversympathetic physician pervades and weakens the whole diagnostic structure.

He also doubts the frequency of "traumatic neuroses," so-called, after trivial falls and injuries when not connected with claims for damages, and has many times found them properly attributable to preëxisting organic disease of the heart, blood vessels, kidneys or nervous system. Trivial injury. How explained.

In those predisposed to or actually suffering from physical or psychic instability or hysteria those conditions may readily be excited or intensified by slight trauma or excitement. There are probably but three *actual* conditions, viz.: (1) *Genuine injury to and actual lesions of the brain or cord.* (2) *Pure asthenia with psychic depression or hysteria, and* (3) *pure or adulterated malingering, usually for revenue.* Actual conditions.

The diagnosis must depend upon the discovery of symptoms purely subjective or of the psychasthenic or hysteric type on the one hand, or *definitely* objective upon the other. Lacking objective signs after several months, one is *reasonably certain* that the case will recover after settlement. Only in

*Using the word in its direct and literal sense.

Prognosis.

Evident exaggeration.

Sprains, fractures, etc.

the presence of the signs of organic disease is the physician justified in a lugubrious prognosis on or off the witness stand. In fully 80 per cent. of such cases, examined by the author, in which suit was pending, the element of exaggeration was *evident*. In hardly any of those injured in a similar manner but without liability on the part of a corporation have there been any symptoms save those of direct injury such as bruises and sprains, neither has the pain in these been persistent over long periods *even in the cases associated with great primary mental shock save in exceptional instances.*

Indeed it must plainly appear to anyone experienced in these matters that the after-symptoms in genuine railroad injuries are remarkably out of consonance with the degree of mental shock experienced at the time.

Finally there is the group of cases which are truly surgical or relate to actual visceral lesions and of fracture of the vertebræ, sprains, hemorrhage into or about the cord, myelitis and meningitis, and are directly diagnosticable, though in rare and deplorable instances the *delayed* development of organic disease may result in an injustice to some honest claimant.

MOUNTAIN SICKNESS.—This is an ailment which attacks mountain climbers or travelers in high altitudes whose circulation is weak, who are unhabituated and unacclimated or are generally out of condition.

It represents oxygen shortage in the blood due to diminished oxygen-tension in the pulmonary alveoli and cardiac overstrain and may last for a period varing from a few days to two weeks.

No one who has visited high altitude resorts or indulged in mountain climbing can doubt that cardiac overstrain of itself is largely responsible for many such seizures.

Symptoms.—The chief symptoms are cardiac arrhythmia with or without decided dilatation, dyspnea, cyanosis or cyanotic pallor, hurried respiration, headache, vertigo, syncopal attacks or a tendency to nausea and vomiting.

Death occasionally occurs in coma.

All gradations of such disability may be encountered.

With the rapid increase in red blood cells and hemoglobin characteristic of high altitude acclimatization the symptoms gradually disappear.

AVIATOR'S SYNDROME.—The European war has served to bring prominently into view certain disturbances related largely to rapid transition from low to high altitudes.

The disturbances experienced by balloonists have long been known but are far less frequent and decided because of the relatively gradual ascent and descent and the lack of special demands upon the physical strength and mental alertness of the operator.

The modern aviator, and especially he who plays his part on the battle-fields or in scouting expeditions, is under tremendous strain, mental, physical, and oftentimes, emotional.

It follows that few or none escape symptoms referable to these factors, quite apart from those due to mere demands upon endurance of the effects of cold.

These are chargeable only in part to mere altitude, which in itself may

seriously embarrass the respiration, markedly accelerate the heart or, at extremely high levels, produce syncope, but for the greater part, represent the effect of rapid rise (increased oxygen demand) abrupt descent, and the extraordinary nervous tension inseparable from such flights.

Mechanical interference with the action of the diaphragm by a distended stomach or intestines must be a decided factor in the production of dyspnea.

High altitude flights must involve enormous fatigue due to oxygen shortage and cardiac weakness even under the compensating increased pulmonary ventilation attained through rapid breathing, yet high altitude flights are repeatedly made over long distances.

The commonest of the symptoms experienced after their return by army aviators would seem to be (*a*) profound fatigue, (*b*) drowsiness, succeeded, perhaps, by high nervous tension and insomnia, (*c*) vertigo, (*d*) rise in blood pressure, (*e*) precordial oppression and palpitation, (*f*) headache of the congestive type.

THE INTOXICATIONS

SUNSTROKE

(*Thermic Fever, Insolation, Siriasis*)

Etiology.—Great heat with decided humidity is the chief factor in both heat exhaustion and sunstroke. Mere exposure to the sun does not cause it, the highest degrees of heat being borne in the western deserts of the United States with less liability to sunstroke than would be experienced at a much lower temperature on the seaboard or in a marshy district. Furthermore, it occurs both night and day, indoors and out of doors. Physical exhaustion, lack of food or water, an unsound heart or kidneys, the overuse of alcoholics *and especially actual intoxication at the time of exposure*, are important contributive factors.

"Sunstroke" a misnomer.

Physical condition.

If one might assume that inadequate heat dissipation is associated with excessive heat production due to exhaustion of inhibitory centers and that at autopsy an extensive pulmonary, cerebral, hepatic, and renal congestion is present, these assumptions would suffice to explain the symptoms, but unfortunately the former remain hypothetical and the latter are inconstant findings at the post-mortem section.

Rational explanation.

Symptoms.—There are preliminary vertigo and oppression *with high fever* (105° to 112°F.) and a "burning" skin followed by sudden coma or decided stupor with or without a convulsion sometimes preceded by brief delirium. The facies is that of apoplexy without paralysis, *i.e.*, lividity and cyanosis, stertorous breathing and pupils fixed in contraction until *exitus* approaches.

Coma.

Hyperpyrexia.

Facies.

Death may occur within a few hours or even minutes unless measures of relief are promptly instituted and the mortality probably exceeds 30 per cent. Relapses are not infrequent and the "stroke" may be followed later by meningitis. The after-effects endure for years in the shape of discomfort on

Mortality.

After-effects.

exposure to unusual heat and humidity or actual attacks of heat exhaustion or sunstroke. This acquired vulnerability is recognized by all life insurance companies.

Differential Diagnosis.—The extreme high temperature at or even before the onset excludes even pontine hemorrhage and at once rules out uremia, alcoholism, diabetic coma and opium poisoning.

Convulsions may occur and tetany or actual total paralysis may exist in some cases but the high initial fever and burning skin of siriasis (sunstroke) are sufficiently distinctive.

It must be remembered that albumin and casts are quite commonly found.

Warning Signals.—In countries where sunstroke is prevalent men recognize the not uncommon occurrence of certain premonitory symptoms which may give warning several hours at least before the actual attack.

Among these Manson enumerates—profound general malaise, pains in the arms and legs, vertigo, drowsiness, mental confusion, abnormal thirst, anorexia, photophobia, chromatic visual aberrations, nausea, precordial anxiety, a sense of impending calamity, emotional instability, a hot skin and rapid pulse. An extremely curious and common precursor is excessive irritability of the bladder (Longmore).

It is commonly believed that a sudden checking or marked diminution of perspiration is a warning signal.

SUN TRAUMATISM.—The cases grouped by Manson under this head seem to be due probably to modified true sunstroke on the one hand, and pre-existing ailments of various kinds and degrees upon the other. They would seem to cover cases of sudden death under exposure to a hot sun in relation to which, diagnostically, a Scotch verdict must be rendered.

Unlike sunstroke.

HEAT EXHAUSTION.—This condition which is essentially a state of more or less complete collapse though entire unconsciousness may or may not be present, is characterized by a cold skin, profound pallor, a subnormal temperature, sweating, and is wholly unlike sunstroke.

ALCOHOLISM

Acute.

ACUTE AND CHRONIC ALCOHOLISM.—The well-known effects of large potations need not be described, yet many mistakes arise in connection with the unconscious or stuporous stage. It should be remembered that in acute alcoholism the pupils are usually dilated, the individual can be partially roused by pinching the inner side of the upper arm or thigh, that the temperature is normal or more frequently subnormal, the breathing deep, slow, but rarely stertorous. The fact that the breath is alcoholic is important, but far from conclusive evidence.

Sneak drinkers.

The symptoms of this condition may be patent to any layman, or entirely absent. The face of the sot requires no description here, but quite as great destruction may be going on in the tissues of one of his fellows whose habits may be wholly unsuspected, even, as happens in rare instances, by his family members. A certain spree drinker of the author's acquaintance has for years

left home at regular intervals, gone to a hotel, quietly passed into an alcoholic stupor only to arise in the late forenoon of the following day, take a Turkish bath and proceed with the business of life. Such is the sneak drinker. Others drink openly and boisterously, yet others, quietly, continuously and in a routine way without themselves suspecting the formation of any habit or the creation of secondary diseases. The kind of stimulant taken is important: pure beer and good wines are the least injurious; absinthe, brandy, whiskey, and gin the most harmful. The poisonous sophisticated liquor so generally sold in low groggeries is peculiarly pernicious in its effects, and the drinking of strong or mixed liquor on an empty stomach so common in this country is an unmixed curse, fostered by the "treating" habit.

Unconscious drunkards.

Sophisticated liquors.

A vicious custom.

Visceral Alterations.—*Digestive System.*—Chronic gastric catarrh frequently associated with hepatic cirrhosis is a common sequence. Gastric dilatation in beer drinkers, and the coated tongue and foul breath of the heavy drinker of stronger liquors is well known.

Cirrhosis.

In the kidneys the chief change is found in simple transient congestion, though interstitial nephritis is undoubtedly promoted by the drinking habit.

Renal lesions.

The lungs seem to be rendered more vulnerable to bronchitis and tuberculous processes, and the circulatory system shows a tendency to myocardial degeneration and arteriosclerosis.

In the nervous system both general and local changes may occur, the morning depression, mental irritability, impaired concentration and quickness of perception, failure of memory, and a decided change in the moral character, being common and well-known manifestations. Recurrent or persistent tremor, especially marked in the morning hours, and even transient or persistent delusions may occur (Korsakoff's psychosis).

May simulate "paresis."

Korsakoff's Psychosis.—This represents a curious combination of alcoholic polyneuritis and memory defect.

The latter takes the form of an almost total failure to record and retain recent events, together with a sort of dream memory of past events which takes the form of pseudo-reminiscences often of a most bizarre character.

Hallucinations may occur and the usual course is one of progressive mental defect.

If recovery occurs the individual's true personality usually is lost or submerged even though outwardly and in casual relationships no change is perceptible.

Alcoholic neuritis is described elsewhere. Alcoholic epilepsy is a curable form occasionally encountered and the so-called "wet brain" and its symptoms have been described under "alcoholic meningitis." One of the unfortunate results of the early morning malaise, impairment of digestion and mental depression is the fact that it largely or wholly disappears if a brisk stimulant is taken and thus the vicious circle is maintained.

Delirium Tremens (*Mania a potu*).—This condition is invariably associated with *chronic alcoholism;* never with the spree of an habitually temperate person, however prolonged the *lapsus*. In the chronic drinker it may result either from some excess or from sudden withdrawal, and is frequent

in such persons following an accident, surgical operation, or some great emotional shock. Its association with acute disease and especially with pneumonia *of hard drinkers* is well known to clinicians.

Tremor, insomnia, depression.

Symptoms.—One of the first evidences is tremor associated with a marked depression, restlessness, and insomnia, which leads to increased potations.

Delirium.

Within twenty-four or forty-eight hours or even less an active voluble delirium appears, and sooner or later the well-known hallucinations known vulgarly as "the horrors." Friends become enemies seeking to deprive him of life

Hallucinations.

and liberty; the commonest noises are misinterpreted; imaginary snakes, rats, or other animals surround him, and he is in a constant and pitiable

Restraint required.

state of terror. Such patients need to be watched and usually restrained, though seldom dangerous save to the furniture and themselves. The tremor of the tongue and hands is extremely marked, there may be a fever of mild degree, insomnia is constant and the disease subsides by lysis in three or four days unless death occurs from exhaustion and heart failure.

Mode of death.

The frequent existence of cardiac dilatation, Bright's disease, or some serious internal injury and the tendency to the development of pneumonia in these

A common error.

cases should never be forgotten. This is particularly true of those seen in public service, who have been brought in by the police.

Indeed apex pneumonias are not infrequently associated with a delirium closely simulating that of alcoholism. The fact that 10 per cent. or more in such services die, emphasizes the importance of the ailment and the frequency

Recurrence.

of complications alike. Recurrence follows continued excessive drinking and usually shows an increased severity and higher mortality.

Morphin and Cocain Habits.—These topics have been briefly discussed under "Case-taking."

CHRONIC LEAD-POISONING

(*Plumbism, Saturnism*)

Certain Etiologic Factors.—The relation of occupation to lead poisoning has already been discussed.

Accidental contamination of drinking water by lead is common through faulty plumbing, the use of cosmetics and hair dyes and the adulteration or faulty preparation of foods and beverages. It is claimed that females are more suspectible than males, and adults more than children. Oddly enough a seasonal influence is noted. Not only is more water ingested during the summer and early autumn, but, if, as is usually the case, the community supply comes from peaty districts, the acid condition of the water tends to increase the degree of its contamination.

Symptomatology.—The chief lesions represent peripheral degenerative neuritis and, aside from rare cases of anterior cornua degeneration, the brain and spinal cord are spared, though marked cerebral symptoms are sometimes observed. Peculiar features of peripheral lead paralysis are the *preservation of sensation* and the extraordinary frequency of *musculo-spiral paralysis*. The kidney and gastrointestinal tract are also frequently affected.

The most characteristic symptoms are: (*a*) *The "blue line"* on and within the margin of the gums unaffected by the toothbrush and representing a black sulphid of lead deposited in the papillæ. It may be faint, stippled, and *blue-black*, and when present is almost pathognomic. *The inner surface of the gums as well as the outer should be examined in all cases.* The blue line.

(*b*) *Colic.*—A violent diffuse spasmodic abdominal pain unassociated with tenderness and relieved by pressure is a common and characteristic symptom. There may be associated diarrhea and persistent dull pain between the paroxysms. A misleading symptom.

High-tension pulse and vomiting are sometimes observed and should suggest uremia from interstitial nephritis or lead encephalopathy.*

(*c*) *Paralysis.*—If paralysis ensues, the arms are most often affected, usually bilaterally, occasionally unilaterally and the nerves of both arms and legs or of the whole body may be involved, *but the tendency is to limitation to muscle groups.* Hence we distinguish a **musculospiral** *variety* associated with "wrist-drop" in which the supinator longus is usually spared, a *peroneal type* (10 to 15 per cent. of cases) involving the common extensor of the toes and that of the big toe and associated with foot-drop and the steppage gait, *and a brachial type* bilateral, and involve the brachialis anticus, supinator, tongue, biceps, deltoid, and occasionally the pectoral muscles. *An Aran-Duchenne* type is interesting because of its resemblance to the first stage of "progressive muscular atrophy," the paralysis involving the small muscles of the hand and producing wasting in the thenar and hypothenar regions. *The laryngeal form* is rare and consists of an adductor paralysis. *Various combined palsies* may be noted and general paralysis may be either of a gradual or rapid development, in rare instances simulating Landry's disease. Paralysis. Types, musculospiral and peroneal. Aran-Duchenne. Laryngeal.

It should be remembered that sensation is usually preserved, a certain amount of early pain may be present, atrophy is marked, the reaction of degeneration present and, in the forearm, tremor is commonly encountered. Motor chiefly.

(*d*) *Cerebral Symptoms.*—Hysterical manifestations, coma, convulsions, transient delirium, and temporary or permanent insanity are occasionally encountered usually associated with arterial hypertension.

(*e*) *Anemia* is usually present in the form of a secondary anemia of varying degree and basophilic degeneration of the red cells is constant in, though not peculiar to, lead poisoning. In no other disease, however, do they appear in such large numbers. Wright's staining method best shows these peculiar cells. Anemia and basophilia.

(*f*) *Arteriosclerosis* and *interstitial nephritis* are common accompaniments of chronic lead poisoning and the examination of the urine for lead is not only important, but indispensable in many cases for the differentiation of the disease. This is especially true of those varieties which exactly simulate an anterior poliomyelitis. Renal and vascular signs.

CHRONIC ARSENICAL POISONING.—The symptoms of acute poisoning are essentially the same as those of cholera morbus with excessive abdominal pain and are described elsewhere. Often unrecognized.

* See also discussion under "blood pressure."

Chronic poisoning possesses a complex symptomatology most baffling and misleading unless the case history *suggests* poisoning by arsenic.

Gastric signs and malnutrition.

Anorexia, nausea, intermittent diarrhea, attacks of colic or abdominal discomfort are the usual gastrointestinal phenomena.

Chronic bronchitis may occur, as may weakness, emaciation, and joint swelling. The nervous symptoms range from those of pronounced "neurasthenia" to neuralgia or actual multiple neuritis. The only symptom in any way characteristic is the yellowish-brown pigmentation of the skin, observed in advanced cases, and puffiness of the eyelids.

Sallowness and puffy eyes:

Arsenical paralysis differs from lead palsy only in its tendency to involve the legs oftener than the arms.

PTOMAIN POISONING AND FOOD POISONING.—It is evident that one must distinguish between the poison inherent in the food itself, whether affecting all or only certain persons, and poisons introduced by accidental contamination in manufacturing processes, and finally, the poisons due to putrefactive alkaloids (ptomains). Under the first class we find a large number of articles which are inherently and generally injurious, such as that caused by certain fish in tropical countries and the mussel poisoning, which, however, is due to a known ptomain, *mytilotoxin*, which produces rapid pulse, dilated pupils, numbness, and a cold surface. Death results from collapse in about 25 per cent. of the cases.

Poisonous fish and mussels.

BOTULISM.—This type of food poisoning has assumed great prominence of late years and is due to a specific bacterial toxin derived probably from one of two strains (A and B) of the *Bacillus botulinus*. This microörganism was reported in 1896 by Van Ermengem, who described it as a large, spore-bearing, anærobic bacillus. It measures from 2 to 6 microns in length and from 0.5 to 1.2 microns in width. The usual arrangement is in pairs, end to end, but in some instances it forms chains. The ends of the individual bacilli are rounded and during spore formation they may be either racket-shaped or spindle-shaped, according to the position of the large oval spore at the end or in the middle of the bacillus. They are slightly motile, sparsely flagellated, Gram positive, and readily take the ordinary stains.

The bacillus is readily cultivated in ordinary media and antitoxins have been prepared and are in use which are thought to be of value in treatment.

Symptoms.—In botulinus poisoning the symptoms appear usually in from 16 to 48 hours after the poisonous food is ingested, though there are instances in which the first effects have been manifest within a much shorter period, this being in some cases reduced to 3 or 4 hours.

The cases which develop early usually show profound gastrointestinal symptoms, namely, nausea, vomiting and purging; the last condition, however is likely to be followed by an obstinate constipation, and this may be the primary condition so far as the intestinal tract is concerned.

If prodromal symptoms are present they are usually merely those of headache, vertigo, and a certain amount of gastric uneasiness or actual distress associated with more or less profound lassitude.

Eye Symptoms.—The eye symptoms are of peculiar interest in that they may play a very important part in the symptomatology. Fundamentally they seem to depend upon an impairment of accommodation and an early attack upon the third cranial nerve. In some instances there is a complete paralysis of all the extrinsic eye muscles; vertigo, nystagmus and photophobia may be marked, and marked pupillary changes may be associated with ptosis of the lids.

Throat Symptoms.—Constriction of the throat is a relatively early symptom and difficulties in speech may result both from impaired immobility of the tongue and a partial paralysis of the laryngeal muscles and of those of the pharynx. Difficulty in swallowing may be marked, and complete aphonia may ensue.

General *muscular weakness* is usually profound but true paralysis does not occur. Sensation is usually unaffected throughout and the mind is usually clear though the patient may become apathetic and ultimately stuporous, or, in fatal cases, comatose.

All *secretions* are usually diminished. The mouth is dry, little or no fluid can be ingested during the severer stages of the seizures, and the urinary output is usually diminished markedly.

The *temperature* is seldom raised above the normal unless a bronchopneumonia supervenes, and as might be anticipated, sub-normal readings are the rule.

Respiratory symptoms are usually absent until a fatal termination is near, when there may be a very marked dyspnea or even Cheyne-Stokes breathing.

Mortality.—It is impossible to give accurate figures with relation to mortality in cases of botulism as it seems to vary widely in different epidemics, in some instances reaching 50 per cent. or more.

Note.—Within the past few years several important instances of botulism have occurred in the United States and the mortality figures have been high. One of the most recent instances was that which occurred at a country club near Canton, Ohio. Of 200 people attending a banquet there were 14 cases of botulism due to the eating of infected ripe olives, and of these 14 cases, 7 proved fatal.

A still greater mortality ratio was reported by Sisco in 1920 as occurring in an Italian family in the Bronx, New York City. In this instance seven of eight family members ate the ripe olives and all died.

ERGOTISM is a well-known form due to the fungus, claviceps purpurea, which produces extensive epidemics due to the use of contaminated grain, the symptoms being either tonic, cramping, spasms, enduring hours or days and characterized by flexion of the arms, extension of the legs. Marked mental symptoms amounting to actual insanity and epilepsy may terminate the convulsive attacks. A second form is characterized by gangrene of the toes, fingers or, more rarely, nose and ears, preceded by muscular spasm of lesser degree, anesthesia, paresthesia, and pain.

PELLAGRA.—This disease of the country rather than the city has been recognized for more than two centuries in Southern Europe and occurs also

An ancient disease.

in Egypt, Algiers, and Mexico. It is probably an infection, though formerly held to be caused by a poison contained in decomposed or fermented corn (maize) whether used as a food or in the form of a stimulant of which it is the basis. Both the nature of the infective or toxic agent and its exact mode of origin and development are as yet unknown.

Cause unknown.

Of late the disease has been steadily extending its area of incidence and many thousands of cases have been reported from the southeastern portion of the United States* together with lesser groups of isolated cases in widely separated localities.

Recently it has been estimated that between the years 1902 and 1920 500,000 cases with 50,000 deaths have occurred in the United States.

Adults between the ages of twenty-one and forty-five are chiefly affected and negroes are especially susceptible.

Symptoms.—The primary symptoms comprise general malaise, constant cerebro-spinal pain, mental dulness, pallor, a peculiar staring facies, progressive emaciation, irritability and insomnia, together with a patchy and varying erythema lasting for a few weeks only to disappear and leave behind a rough skin stained light sepia. The other symptoms last through the winter, while the spring is associated with the full development.

This takes the form of marked dyspepsia, both gastric and intestinal, more or less severe diarrhea or actual dysentery and a peculiar remitting but progressive erythematous rash affecting the exposed surfaces of the body and followed by desquamation. This dark red rash is particularly well marked on the neck and hands and blanches under pressure.

The palms of the hands are usually free and as the period of desquamation approaches the deep red becomes gradually a livid blue.

The skin finally becomes thinned and lifeless and the course of the eruption may be varied by vesiculation, suppuration, fissures, or the formation of crusts, or take on a macular form.

Summer brings a remission, the following spring or, in our own country, the fall, a relapse, and finally a cachexia appears, associated with disorders of the special senses and of the mind, spastic paralysis of the legs associated with atrophy and contracture, normal superficial reflexes and increased knee-jerks.

Cerebrospinal pain is intense, associated with troublesome paresthesia and maddening hyperesthesia. Anemia and emaciation are profound.

Diagnosis.—The combination of the skin lesions, gastrointestinal disturbances and seasonal variations make a striking combination. An acute form is rarely observed which may simulate typhoid fever or meningitis.

Prognosis.—The disease lasts for a decade or more and furnishes a considerable proportion of the cases of insanity encountered in Italy. As might be expected the autopsy often shows chronic exudative meningitis, and occasionally, hemorrhagic sclerosis of the posterior and postero-lateral columns, and degeneration of the peripheral nerves.

* Alabama, Mississippi, South Carolina, Tennessee and Texas have shown a heavy death rate.

BERI-BERI.—This peculiar endemic epidemic neuritis causes a large mortality in Asiatic countries, is especially prevalent in the Malay Archipelago and a few important cases are encountered in this country usually from South American and West Indian ports or among fishermen on the Newfoundland banks. The specific cause is unknown, but Baron Takaki, Japanese Surgeon-General, found that a marked diminution in the incidence of the disease followed the withdrawal of raw fish from the diet, and the substitution of unshelled for shelled rice.*

Baron Takaki's work.

The Vitamin Doctrine.—A vitamin is a substance belonging to a chemical group of unknown composition "present in small quanities in certain foods and essential to normal metabolism."

In rice, for example, in the process of removing the husk and polishing the grains the pericarp is removed and with it a part of the alurone layer. It would seem to be established that this process involves the loss of a vitamin, indispensable to the entire prevention of Beri-beri in a population depending largely upon rice for food.

Symptoms.—The symptoms are chiefly *those of neuritis* of greater or less severity. Edema is an important symptom varying from that of the slightest degree to a general anasarca. Palpitation and dyspnea are also marked symptoms, varying greatly in severity. A somewhat arbitrary classification is based upon the factors above named, there being a "*mild form*," in which all symptoms are slight, a "*wet or dropsical*" variety in which edema is the predominant symptom, a "*dry form*" characterized by predominant paralysis and relatively slight edema and a "*pernicious form*" in which extreme cardiac decompensatory symptoms predominate.

"Mild", "wet," "dry" and pernicious forms.

MALINGERING

Until medicine becomes an exact science the physician will do well to give the benefit of any doubt to the supposed malingerer and avoid measures savoring of harshness or cruelty, or the giving of injurious testimony in a court of law, save when the case is clear and the imposture of a particularly barefaced or injurious nature.

The chief sufferers from feigned diseases are railway, street car, and accident insurance companies, but the evil extends to the army and navy pension bureaus, and even to the home, and every physician must meet with numerous cases whatever may be his line of practice.

No one class of diseases has any monopoly of this form of deception, though the nervous system lends itself most readily to the needs of the impostor, while, on the other hand, presenting more traps and pitfalls and, in certain directions, more difficulty in simulation than general diseases.

The basis of the most serious types of malingering or imposture is the dollar and the lengths to which people will go in their attempts to defraud

* Shortly after the Russo-Japanese war Baron Takaki stated to the author that the mortality and incidence of this disease in the fighting forces was thus reduced to an almost negligible factor.

Malingering extraordinary.

accident insurance companies especially is almost incredible. For many years these companies paid a large indemnity for the loss of the left hand and the number of claims incurred was so great as to lead to a reduction of the indemnity by one-half, following which the claims of this class fell off over 80 per cent.

Men will deliberately shoot themselves or place a hand or foot under the wheels of a moving train for the sake of the small amount of money represented by accident indemnity and similar examples of self-mutilation are found in the countries where military service is compulsory or in the case of soldiers tired of service and desiring a discharge; the common form being formerly the mutilation of the trigger finger as furnishing the readiest and slightest injury incapacitating for active service.

The minor frauds in this connection are consequently encountered by every physician and the unscrupulous man with an unrecognized but readily sprained flat-foot or the still more fortunate possessor of a shoulder or hip that he can dislocate at will is reasonably sure of realizing something from his infirmity.

Furthermore, as is now well known, certain outlaw attorneys and equally unscrupulous physicians of the large cities form a criminal class known as "ambulance chasers." Such scoundrels will not only follow up cases of injury and suggest lawsuits, but will furnish witnesses ready made, sworn physicians' statements of any desired character, and any other bit of criminal machinery needed in the given case.

A Curious Case of Simulated Injury.—Among the many instances of professional malingering one will suffice for illustration. A certain individual, following the usual custom of the expert criminal, took one special line as his own and the street car companies as the objects of his attack. His procedure was simplicity itself; he would enter a street-car carrying a cane which bore on its tip a screwdriver and while sitting in the car would quietly elevate a floor screw of the seat in front of him. Thereupon he would rise hastily to leave the car, ostensibly trip over the screw-head, and in falling receive an apparently severe and painful injury. He would then summon a carriage and be driven rapidly home where he would take a tack-hammer and after covering the knee, elbow or ankle-joint with a few layers of cloth would pound the point selected until he had produced the necessary appearance of a severe joint injury, after which he summoned a physician. It is said that this went on for years and that the man received in damages from different corporations a very decent fortune. As regards deliberate mutilations of the more serious type, it should be remembered that the victims are usually urgent in their demands for amputation, frequently suggesting it and often insisting upon it. Such are usually holders of recently issued accident policies, and the mutilation is ordinarily just sufficient to come within the terms of the contract and seldom involves the most useful member; the *left* hand or foot being usually selected.

Suggestive facts.

Classification.—*Disease or injury may be (a) wholly spurious and non-existent, or (b) actual, but either self-produced, factitious or deliberately exagger-*

ated or aggravated, (c) the deception may be an innocent one due to a genuine belief in its serious nature on the part of the patient, which opinion is usually fostered by friends and family and too often by an oversympathetic physician or a shyster lawyer.

SOME OF THE COMMONER FEIGNED STATES

Anemia.—By taking a nauseating substance pallor is readily achieved, but the absence of anemia is readily demonstrated by testing the blood.

Angina pectoris can be successfully simulated by one familiar with the subjective symptoms by taking drugs which produce a disturbed heart rhythm, swallowing tobacco to produce pallor and clammy skin, and then simulating the paroxysmal seizures. It requires, however, a good actor to deceive one who has seen the genuine attacks. The tobacco or other drugs may be taken by the rectum and palpitation may be caused by extreme compression of the abdomen; indeed, Herold states that hypertrophy may thus be induced, which statement we would beg leave to doubt. A normal or but slightly raised blood pressure would at once arouse *suspicion* of simulation. In nearly every case of true *major* angina pectoris so far tested by the author the arterial pressure has been *very* high.

Most frauds of this type throw themselves about and produce a clinical picture which is the exact antithesis of the genuine one.

Aphonia.—Here the one reliable test is the volubility of the patient during the stage of excitement or the after stage of recovery from an anesthetic. No condition is more readily simulated.

Asthma of the spasmodic type cannot be successfully simulated nor can the severer forms of dyspnea because of the absence of persistent cyanosis and the characteristic physical signs.

Atrophy of the Extremities.—A certain amount of atrophy may be induced by long periods of splinting or disuse and increased by the use of tight bandages.

Blindness.—Amaurotic blindness is easily feigned by instilling atropia and is difficult to detect unless the patient can be thrown off his guard and carefully observed for a considerable period. Simulation of blindness, however, usually involves one eye and is oftentimes a mere exaggeration of an existing though trivial defect in refraction. A pupil reacting normally to light excludes most forms of blindness, but in all, the testing of the visual field will often reveal deliberate deception and a simple test consists in holding a pencil close to the sound eye and asking the patient to say when its tip is lost to his vision; he may still see it after it is passed well beyond the nose or deny seeing it while it is manifestly in the range of the normal eye. If asked to look at an object held in his own fingers a malingerer feigning total blindness will often pretend that he cannot get the direction of the object, while those actually blind will look at it, judging its position without hesitation. If a pencil is held between a book and the eye and double vision exists it does not interfere with the reading, but if only one eye is competent reading is impossible as certain words will be cut out. This is an excellent test because the

pencil can be thus used without attracting the attention of the malingerer. It must of course be held close to the eye and motionless. Lenses make still more difficult any fraud in connection with unilateral blindness. If, for example, a strong prism is placed in front of a sound eye, base up, and brought gradually in front of the eye from below upward, double vision will occur before the base reaches the pupillary center, one image directly transmitted, the other refracted. This being admitted by the patient, the prism is gradually pushed upward so as to obscure the pupil, producing a single image for that eye, but if both eyes are competent a double image because of the different levels. If, therefore, a double image is still admitted, as is almost invariably the case, it proves vision in both eyes. Another test consists in using a 6 D convex glass upon the sound eye, thus reducing the range of vision and making reading possible only at 17 cm. or less. The patient is asked to read and the book gradually removed, when it will be found ordinarily that the range is greatly exceeded or variable. Still another test consists in introducing a red glass before one eye and a green one in front of the other; the patient is then asked to look at Snellen's colored test types. Only the colored letters that correspond to the color of the glass in front of the eye can be read by that eye, and if one is blind unilaterally he will not even suspect the existence of letters other than those read by the supposedly sound eye. This test may also be made by using an ordinary pencil and a red pencil to make the written characters of the two colors.*

Cancer.—By severe irritation of an existing sore in certain regions, such as the breast or lip, or by attaching to the skin a section of the spleen a passable imitation of cancer may be attained, but the success of such impostures seems incredible if any examination be made and the patient under proper observation.

Catalepsy.—This is imperfectly simulated, as the maintenance of the forced postures characteristic of the true state is impossible, the arm or leg if extended soon tiring and dropping to the normal position.

Cerebral Concussion.—This condition is often feigned, but it is only necessary to remember that the genuine lesion is associated with superficial respiration, pallor and moist skin and usually with nausea or vomiting.

Chorea is in itself difficult to simulate and readily detected if the subject can be observed when awakened from a sound sleep, in which case he will for a moment forget his rôle.

Consumption.—Only the stethoscope can unmask the skilful malingerer who chooses this ailment, and if, as frequently happens, there is actual bronchitis the exposure depends upon the absence of physical signs of infiltration or cavity formation and of fever or bacilli in contrast to the patient's apparent condition, which is usually pitiable. The common method employed consists of feigned cough, bloody sputum, obtained by pricking the gums or when opportunity offers (as in hospitals) mixing ordinary sputum with blood or pus, and emaciation, produced by abstinence from food, drink-

* These tests are for the most part derived from Fuch's "Text Book of Ophthalmoscopy."

ing quantities of vinegar or chewing and swallowing large quantities of tobacco.

Contractures.—Joint diseases with contracture or ankylosis offer an inviting field for the malingerer, but the joint in such cases is normal or becomes so if the patient is kept under observation and lacks implements and opportunity for producing fictitious lesions. Pain is frequently associated with the lesion and is often of such a nature as to at once expose the attempted deception. Muscular atrophy save that of disuse is lacking, the patient resists by evident muscular contraction attempts to move the joint and an anesthestic reveals motility and exposes the fraud. One simple method is often effective with this class, viz., having the patient stand and then suddenly pushing him off his balance when the supposed crippled leg will usually be extended for support. Another method is to place the patient, standing on one leg, upon some high object, as a stepladder, the sound limb being slightly flexed; muscular fatigue will often force the patient to bring down the affected leg for support. Yet another consists in suspending a heavy weight over the contracted limb, and it soon yields to the steady pull. Another old procedure involves the use of an Esmarch's bandage, which, being tightly applied, prevents the muscular action which maintains the false contracture.

Convulsions.—It is physically impossible to maintain for a long period the more violent convulsive movements and difficult to simulate correctly and in proper sequence those of the well-known types. Nevertheless, even epilepsy may be exactly simulated by one having a thorough knowledge of the true disease and willing to go to the extent of biting the tongue and passing urine during the paroxysm.

Cutaneous Lesions.—These are readily simulated in many of their forms A simple mustard plaster will produce erythema and may be cut in any form desired; pustules may be caused by croton oil; blisters by cantharides. Urticaria may be deliberately produced by eating certain substances known to produce them in the given individual (shell fish, strawberries, etc.). Pruritus offers no difficulties to the malingerer. Dermatitis and ulcer may be produced by acids or caustics; gangrene, or the appearance of it, by constricting bands, and indeed nearly every skin disease, including alopecia, bromidrosis and chromidrosis, may be accurately feigned, but are of little consequence. Detection of most of them is easy if a fixed plaster-of-Paris dressing can be applied, the patient being then unable to keep up the necessary irritation, and in the case of simulated gangrene, stripping the patient will disclose the cause and close confinement and observation will result in a disappearance of the lesion.

Diarrhea and dysentery both may be simulated, most commonly by mixing the fecal discharges with urine and adding blood from the finger or gums. Soap may also be employed and iron and bismuth taken by the mouth may add to the apparent abnormality. Such impostures ought never to be successful if a patient can be watched, as the evacuations can be received in a pan and directly inspected.

Dropsy, Edema, Ascites.—Edema may be produced in an extremity by the use of a concealed ligature which, however, will leave its mark. It is even said that water has been injected directly into the peritoneal cavity.

Dyspepsia.—No disease is more easily simulated, because of the predominance of subjective symptoms in the real disease. The introduction of a stomach tube after a test meal, if accompanied by a positive statement as to the certainty of its findings, may reveal the imposture, unless the simulator knows much of the vagaries of the disorders of digestion. Another successful method is the buttermilk cure with absolute rest in bed, and without means of diversion.

Dyspnea on exertion can be clumsily simulated, but with normal heart, blood, lungs, and kidneys is disproved.

Calculi and gravel are frequently simulated, most commonly by urinary sediments, which are usually readily proven to be street gravel, brick dust, cinders, or bone. In the female these substances are not infrequently actually introduced into the bladder or stored away in the vagina and used as occasion offers.

Epilepsy.—As before stated, this disease can be, but is with difficulty simulated. It is employed by professional thieves and beggars, as it draws a crowd, diverts attention and excites sympathy. The use of a pinch of snuff often suffices to expose the fraud, as will the injection of apomorphia and occasionally the actual cautery or a threatened operation. Marked improvement under the administration of sodium chloride, the patient thinking that he is taking sodium bromide, may expose the supposed victim of chronic seizures.

The Eye.—Diseases of the eye, aside from the blindness already mentioned, are readily simulated, a great number of irritants being ready to hand. In these cases the rapid onset of inflammation and its prompt subsidence after the withdrawal of the irritant exposes the fraud. Chronic inflammation as induced by beggars may be extremely difficult to detect and is often carried to the point of ulceration of tissue.

Feigned Sleep.—It is said that in genuine sleep if the eye be suddenly opened, even in the presence of a bright light, there is transient primary dilatation of the pupil.

Fever.—Save in actual hysteria, the simulation of fever is unsuccessful in the light afforded by clinical thermometers, but nothing is commoner than the use of the hot-water bag or a hot drink for the purpose of raising the temperature. Fever readings of any height are easily produced thus or by friction applied to the thermometer.* Chill is sometimes feigned, but the malingerer is found hot and sweating under his blankets from the warmth and from his own exertions.

Fistula.—Anal fistula is readily and often successfully simulated by making a cut and treating it with some irritant, such as carbolic or one of the

* In a recently observed private case a much-desired winter trip to the South was obtained by this means.

strong mineral acids, but usually it is clumsily done and the imposture detected.

Fictitious Wounds.—Aside from mutilation under accident policies, fictitious wounds are not uncommon in connection with false charges of assault or in the case of those who seek to disguise their own theft by the pretense of attack and injury at the hands of burglars. They are also employed as a means of establishing self-defense in cases of actual or attempted homicide. The most significant factor in their detection is in the comparatively trivial nature of the injury, the fact that knife wounds are inflicted in the region most accessible to the hand commonly used by the individual, or, as is often the case, such cuts and gunshot holes are made in the clothing to intensify the appearance of a struggle, as should manifestly have involved the skin or body but do not. Attempted suicide by throat cutting with the razor or knife may be denied or concealed, but the characteristic incision obliquely downward and forward from the angle of the jaw on the side opposite the arm commonly in use is usually sufficiently characteristic.

Fractures.—Simulated fractures can impose only upon ignorance and are absolutely exposed by the X-ray.

Headache.—Feigned headache can hardly be detected with certainty, though close observation by the physician or nurse and the readiness with which sleep is produced may in some cases be important.

Heart.—Save in the rare instances of inhibition of the heart action, cardiac ailments are simulated only by producing palpitation and changes in the pulse rate. Sly compression of an axillary artery or the use of a concealed ligature may produce unilateral weak pulse. Digitalis in full doses may produce an apparent bradycardia, or, if pushed still further, an irregular and tumultuous heart action. Cardiac dilatation or hypertrophy, valvular murmurs and abnormally accentuated heart so unds are not readily simulated.

Hemorrhages.—The commonest are those simulating *hemoptysis*, hematemesis, and hemorrhoids. The saliva may be clumsily covered with dyes, brick dust, etc., or, more commonly, mixed with genuine blood obtained by pricking the gums or fauces. If the symptoms of tuberculosis are absent, both as to fever and physical signs, a solution is relatively easy.

Hematemesis may be simulated by pricking the gums and swallowing the blood which is afterward vomited, and constitutes one of the most difficult of all impostures for the medical examiner, particularly if associated with feigned pain by one having a knowledge of gastric ailments. The sources of anal hemorrhage are readily detected by local examination, further they result from cutting or pricking the parts or from the introduction of blood, the lesion being evident upon removal of the blood which shows no possible source for the hemorrhage. In some instances substances, such as inflated fish bladder, have been introduced to simulate hemorrhoids. They are readily detected by local examination.

Hernia.—Inflation of the cellular tissue of the scrotum and in some instances voluntary retraction of one testicle have been employed, but the fraud

is readily detected by the behavior of the supposed intestinal coil under manipulation or the absence of one testicle.

Hydrocele.—Attempts to simulate this lesion by injecting water into the tissues of the scrotum are clumsy and unsuccessful and in any event readily detected by drawing off a portion of the fluid.

Hydrocephalus.—Pseudo-hydrocephalus has been produced by the injection of air into the cellular tissue of the scalp. The crackling on palpation and peculiar fascial distribution should at once excite attention.

Hydrophobia is frequently simulated, but is usually greatly overdone and readily detected.

Hysteria may of course be readily simulated, but is an absurdity.

Incontinence of Urine.—This is oftentimes extremely difficult to detect, but it is frequently possible to make the circumstances surrounding it so embarrassing as to expose the deception. The *steady drip* of overflow incontinence cannot be simulated if the patient is closely observed.

Insanity.—The following points may be of service in this frequently feigned disorder: *Melancholia.* A sudden onset with loss of memory and dementia at once proves the ailment spurious. *Mania.* A sudden onset lacking all relation between the surrounding and the patient's ideas, the relatively easily induced fatigue and natural sleep proves the falsity of the ailment. *Maniacal Frenzy.* Absence of a preliminary period of depression and loss of memory of events just prior to the attack are the two chief signs of fraud. *Paranoia.* The feigned disease is indicated by the obtrusiveness of the assumed delusions, the usual evidence of a capacity to weigh and judge the effect of statements made and the lack of elaborately systematized delusions. *Paretic Dementia.* A sudden onset, the absence of eye symptoms, the lack of true speech defects of the well-known type are sufficient to establish the deception. *Loss of Memory.* The radical error of impostors is their failure to observe the fact that in all forms of dementia the loss of memory is of the senile type affecting recent events, but not necessarily remote ones, often seeming to intensify those dating back to childhood and youth. In the less extreme form of genuine amnesia the memory remains for striking events, the very ones in fact that a criminal seeking to escape punishment will forget.

Insanity is, moreover, extremely difficult to simulate and demands a knowledge of the various types, possessed by few. Not only is failure of memory usually exaggerated and often intermittent and inconsistent, but the facial expression is difficult to assume and the demands upon the simulator for deviations consistent in speech and action are seldom met. The person feigning dementia almost invariably pretends to have forgotten all events.

Jaundice cannot be successfully simulated because of the impossibility of maintaining the yellow discoloration of the conjunctiva when supplies are cut off. Turmeric has been used to color the skin, HCl added to the stools to make them clay-colored, rhubarb ingested to give the urinary color, and sometimes the eyes are deliberately inflamed to hide the deficiency in that region.

Joints.—See "Contractures," "Rheumatism" and "Limping."

Limping.—Careful observation of a person using a cane or crutch may establish the fact that it is not being made a genuine support. Pretended fixation of joints is revealed by the use of an anesthetic and the absence of all inflammatory signs, joint crepitation, etc., will usually suffice to detect fraud.

Lumbago may be successfully simulated and is the favorite form; fortunately the treatment ordinarily pursued is sufficiently heroic to make it of short duration.

Ozena is said to have been feigned by the introduction of ripened, highly odorous cheese into the nostrils.

Pain and Tenderness.—These are extremely difficult symptoms to detect the falsity of, being wholly subjective, yet if the patient be under close observation it is seldom difficult to expose the fraud. Few can carry on a conversation while the physician is examining the body and still be prompt to respond to pressure made with one hand over the supposed tender area while a more vigorous and obtrusive procedure is being carried on elsewhere with the other hand. Furthermore, the administration of morphia may suggest fraud, either by the relief of pain afforded by a small dose or a spurious one or by the assertion of persistence of pain or tenderness after the administration of full doses. Nearly all cases of this kind are overdone and the pain is referred to vague, indeterminate, shifting, or unusual locations. If, moreover such a patient can be examined during sleep, such as that produced by sulphonal or some other pure narcotic in moderate dose, it will be found that the tender areas do not exist and that the pain which should prevent sleep does not affect it.

Exaggeration is usually so great as to reveal the imposture.

Paralysis.—This condition can hardly be feigned with success if the examination be thorough and in competent hands. Its description would involve a rehearsal of the signs and symptoms of the paralyses resulting from the lesions of individual nerves or nerve groups and the different areas of the brain and cord, as well as the phenomena of hysteric paralysis. It may be well to remind the reader that in the last-named disturbance loss of sensation in the extremities stops sharply at the junction of the limb with the body, and if unilateral and of the trunk, is sharply limited by the median line and is likely to affect the entire side, including the face and scalp. Furthermore, the reflexes in this condition are likely to be bilaterally increased even though the paralysis is unilateral. Feigned anesthesia is best detected by the unexpected application of the electric brush and it is impossible for a malingering patient to maintain the fixed boundary line of a true anesthetic zone, as first defined by a skin pencil according to his own responses, repeated examinations revealing marked variations. So also partial general anesthesia will almost invariably unmask feigned paralysis.

A very simple device often successful when unilateral involvement of the extremities is asserted consists in pricking first one limb and then the other demanding that a prompt "yes" or "no" be given with respect to sensation.

The unwary malingerer will answer "no" when the supposedly anesthetic limb is touched forgetting that he can feel nothing on that side.

Peritonitis is often simulated, but seldom successfully; one of the most common lapses being a *restlessness* on the part of the patient, sharply in contrast with the fixed position assumed in true peritonitis; the true facies and pulse cannot well be simulated.

Rheumatism can only be successfully feigned in its acute form by pounding the joints, and this is seldom carried to the point of producing any misleading resemblance. The fever and peculiar sweating are beyond the patient's resource if he is under observation, and the lesions rapidly fade.

Retention of Urine.—This cannot be simulated if the patient is carefully watched.

Sciatica.—This frequently feigned disorder is oftentimes readily detected by the clumsily described referred pain and points of tenderness. Few genuine cases lack maximum tenderness midway between the sciatic notch and the knee, even though the other points are insensitive. In the genuine disease, moreover, if the extended leg is flexed upon the pelvis, the pain is severe though the opposite movement causes little distress. In spurious cases the patient may walk upright though limping and feigning great pain and distress, whereas in the true sciatica the thigh is always more or less flexed and the body inclined forward.

Scoliosis.—Feigned scoliosis is almost invariably dorso-lumbar, the spinal axis is not changed as in the true ailment, the skin is more markedly involved and the secondary compensatory curve is absent.

Scurvy cannot be simulated except by deliberately taking mercury to the point of excessive salivation, and swelling of the gums otherwise seldom or never will be encountered.

Unconsciousness.—Aside from ovarian and supraorbital pressure in the hysterical cases, the application of a sharp electric current, and similar drastic measures, the best procedure by far is that of administering ether, when the patient will invariably struggle and probably talk during the stage of excitement, unless the unconsciousness is real, when there will be no response.

Venereal Diseases.—Such simulation is not infrequent in connection with blackmail. Artificial eruptions are as readily produced on the genitalia as elsewhere and a fair imitation of gonorrhea can be produced by the use of caustics or other irritants, whether in the female or in the male, but the absence of the gonococcus is readily established and the peculiar characteristics of a primary sore are not easily simulated.

Vertigo.—It may be impossible to detect spurious vertigo, though here as elsewhere close observation may result in catching the patient off guard.

Vomiting.—This may be a very difficult form of malingering to detect, but is usually so causeless or so directly and immediately related to the taking of bland substances as to expose its real nature. It may often be detected by making the circumstances under which it would occur particularly embarrassing to the impostor. In a case of persistent habital vomiting observed

by the author a cure was effected by forcing the patient to go to the theatre for an evening.

Wry Neck.—This is usually readily detected by the muscular rigidity which follows any attempt on the part of the physician to correct the deformity. Furthermore, the associated unilateral atrophy cannot be simulated and the condition disappears during sleep or anesthesia.

Comment.—*In conclusion one may say that in all simulated diseases detection depends upon (a) the incongruity and lack of proper sequence in the symptoms presented; (b) the presence of a motive for simulation; (c) the patient's actions and the course of his ailment when under close observation and control or twhen exposed to shock, surprise or suggestion; (d) the use of drugs and anesthetics; (e) the absence of physical signs of the disease or symptom simulated.*

By far the most difficult cases are those occurring in the domestic circle where the physician must use great tact and judgment and is usually obliged to make the patient expose himself. It may be added that the ruse so often suggested of allowing patients to overhear conversations calculated to produce absolutely spurious symptoms is successful only in the case of stupid and ignorant patients, the brainy impostor being ordinarily prepared for such maneuvers. On the other hand, in many instances the simplest of measures suffice for exposure if the patient is under control, among the commonest being the mere threat of operation, the administration of nauseating drugs, or the establishment of an attenuated or unpleasant dietary and irksome restraint. Confinement to bed, a diet of milk or buttermilk and a mixture of nux vomica and asafetida t.i.d. proved of sovereign virtue in a certain public service infested by clumsy malingerers suffering, actually, from antipathy to honest labor.

CONDITIONS SIMULATING DEATH.—Asphyxia.—In drowning especially, death is often only apparent and resuscitation possible after long periods of submersion (one hour or even more). The same is true of infants apparently born dead. Efforts to restore life in such cases should therefore be carried out for a long period before hope is abandoned.

Catalepsy.—In this condition the simulation of death is not ordinarily sufficient to lead to error, although both respiration and circulation may be but faintly indicated, superficial reflexes lost, analgesia and anesthesia present, and the temperature decidedly low. In the "*trance state*" the simulation of death is much more perfect, the limbs being flaccid or showing a rigidity simulating rigor mortis, the face pale, and the pupils often fixed in dilatation. In these cases the question of voluntary temporary but decided partial inhibition of the heart action and respiration is interesting, and there can be little doubt that certain of the East Indian fakirs possess this power. In the case of Colonel Townsend, as described by Cheyne, the mirror test and stethoscope failed to reveal any sign of respiration or heart action for fully half an hour.

Syncope (Fainting).—As this represents a temporary failure of respiration and heart sounds and is associated with complete unconsciousness and pallor, it perfectly simulates death, but lasts ordinarily but a few seconds.

Signs of Life in Persons Apparently Dead.—This title seems to the author more appropriate than the usual one, viz., "Signs of Death." In the trance state or in those who can voluntarily inhibit heart action the pulse may be absent and the heart sounds inaudible, but the following signs will be present if the patient is living.

1. *A deep red or purple color in the finger tips will become evident gradually if a firm ligature be applied to the digit.* If applied to the wrist, prominence of the veins on the dorsum of the hand indicates life. The ligature must not be so tight as to completely cut off the circulation.

2. *Several hours after a supposed death blood will flow persistently from a cut artery.* A small artery should be chosen, not mere wet cupping or haphazard puncture.

3. *If a needle thrust into the tissues and left for a time becomes oxidized, life is present.*

4. *If any cloud repeatedly appears upon an ice-cold mirror held close to the mouth, there is respiration, but its absence does not alone suffice to prove death.*

5. *If a powerful vesicant produces redness or blisters, there is life.*

6. *If a body fails to take approximately the temperature of its environment forty-eight hours after apparent death there is life.*

7. *Pupillary response to light shows life, its absence does not prove death. Several hours after death it is affected neither by atropin nor eserin.*

8. *Persistence of the red in, and visibility of the arteries of the optic disc* are signs of life, as is also *persistent clearness of the media,* six to eight hours after apparent death.

9. *A sensitive cornea is a sign of life, absence of the corneal reflex is not a sign of death.*

10. *Presence of electric excitability in all muscles twenty-four hours after apparent death indicates life.* (Usually lost in from three to six hours, but retained for from ten to fifteen in certain cases.)

A BRIEF SUMMARY OF THE SYMPTOMS AND TREATMENT OF ACUTE POISONING

ACIDS, MINERAL.—(HNO_3, H_2SO_4, HCl.)

Symptoms.—Burning pain in the mouth, esophagus and stomach, vomiting of grumous liquid containing shreds of tissue; mind clear, intense thirst, dysphagia and usually dyspnea or actual suffocation, scanty or suppressed urine, constipation and profound shock.

Acid Stains on Mucous Membranes. Sulphuric: White turning to dark brown or black. *Nitric:* White, then orange, then brownish-red. *Hydrochloric:* White or gray. *Duration of fatal period:* Several hours or *months,* depending upon amount taken, etc.

Treatment.—Cardiac stimulants, morphia to relieve pain, baking powder or bicarbonate of soda in milk, egg albumin, carbonate of magnesia, calcined magnesia, mucilaginous liquids, sweet oil, olive oil, chalk, whiting, soap and

water; *the stomach tube or pump being barred.* All treatment fails if pure acid has been swallowed in quantity.

ACONITE.—(*Aconitum Napellus, monkshood, wolfsbane.* Fatal dose, 80 minims of the tincture; maximum medicinal dose, 1 to 5 minims.)

Symptoms.—(Usually come on in a few minutes or may be delayed one hour or more.) Pallor, dryness and tingling of lips, tongue, pharynx, then of extremities and finally of whole body, nausea, perhaps vomiting, pharyngeal constriction (subjective); slow, weak, later rapid and irregular pulse, subnormal temperature, slowed, shallow and irregular respirations, vertigo, dimness of vision, tinnitus aurium, clammy anesthetic livid skin, dilated pupils if convulsions are present, profound prostration and death. *Fatal period* from thirty minutes to five hours.

Treatment.—*Absolute rest.* Evacuation of stomach by tube or stomach pump *if condition permits*, elevation of foot of bed, external heat, cardiac stimulants, artificial respiration, hypodermoclysis, hot rectal enemata. Tannin and charcoal may be given, but are probably of little use. Large doses of digitalis seem effective (m xx–℥i).

ARSENIC.—(Fatal dose, 1 to 2½ grams, retained.)

Symptoms (After half an hour or more).—Thirst, epigastric pain and tenderness, nausea, vomiting, purging with tenesmus, muscle cramps, rapid labored respiration, incessant and violent retching, pulse weak and rapid, urine scant or suppressed, cold wet cyanotic skin, perhaps paralysis, convulsions, and coma.

Variations.—Cerebral symptoms may predominate, pain and vomiting may be absent or gastro-enteric symptoms be exceptionally violent. *The usual picture is that of cholera morbus. Average duration* in fatal cases between eight and seventy-two hours, shortest seventeen minutes.

Treatment.—Evacuation of stomach, bland and mucilaginous drinks, ferric hydroxide (ferric chloride precipitated by any alkali) in milk, barley water, etc., followed by castor oil.

ATROPIN.—(Belladonna. Fatal dose of atropin ½ grain.)

Symptoms (After one-half to two hours).—Dry hot throat, dysphagia, vertigo, dilated pupils, blurred vision, flushed face, brilliant eyes, rapid pulse and respiration, active or even maniacal delirium, hallucinations, nausea, vomiting, scant, bloody, or suppressed urine, sometimes strangury and priapism; scarlatiniform rash on face and neck, sometimes general; stupor and death. It should be remembered that 10 to 15 drops of the urine in such a case will dilate the pupil of any animal except birds and monkeys.

Treatment.—Evacuation of the stomach and bladder, morphin, pilocarpin, tannic acid, cardiac stimulants.

CANTHARIDES.—(Fatal dose, 24 grains or 1 ounce of tincture.)

Symptoms.—Abdominal pain, vomiting, dysenteric purging, dysphagia, loin pain, dysuria, strangury, and frequently priapism in the male, hot and swollen labia in the female, or actual frenzied eroticism.

Treatment.—Evacuation of stomach, cathartics and bowel flushing, opium, cardiac stimulation, demulcent drinks but no oils, chloroform inhalations.

CARBOLIC ACID.—(Fatal dose usually ℥ to ℥ i, but less would probably suffice, depends largely on dilution and stage of digestion.)

Symptoms.—Odor of acid on breath, whitened mucous membranes from contact. Burning pain throughout affected tract, vomiting, retching, contracted pupils, insensitive cornea, dyspnea, respirations rapid and shallow, stertor, collapse, coma and perhaps convulsions. Urine scant or suppressed, smoky, sometimes black or bottle-green. *Fatal period*, few minutes to several hours.

Treatment.—*Dilute alcohol*, whiskey, brandy in quantity, *then evacuation of stomach; epsom salts*, saccharated lime, soap and water may be used and demulcent drinks.

CAUSTIC ALKALIES.—(Sodium and potassium hydrate, ammonia.)

Symptoms.—Same as corrosive acids except that there is dysenteric purging.

Treatment.—Oils, dilute vinegar, lemon juice, cardiac stimulants, *morphia*, milk, mucilaginous drinks. *Stomach tube contraindicated.*

CHLORAL HYDRATE.—("Knock-out drops," etc.)

Symptoms.—Stupor or coma, pallor, either slow or rapid *feeble* pulse, slow respiration, pupils first contracted, later dilated, marked muscular relaxation, cardiac failure.

Treatment.—Cardiac stimulants followed by hot packs and brisk friction, artificial respiration, hypodermoclysis.

COCAIN.

Symptoms.—Nausea, vomiting, mental excitement, delirium, rapid pulse and respiration, dilated pupils, *fever*, convulsions, stupor, coma.

Treatment.—Cardiac stimulants, inhalations of chloroform to control convulsions, evacuation of stomach, morphia.

COLCHICUM.—(Fatal dose, ½ ounce of wine or less.)

Symptoms.—Those of a violent gastrointestinal irritation with dilated pupils, cramps, perhaps delirium or convulsions, collapse, urinary suppression, partial or complete. *Fatal period*, a few hours to several days.

Treatment.—Cardiac stimulants, hypodermoclysis, morphia.

CROTON OIL.—(Fatal dose, 30 minims.) Typical choleriform symptoms, collapse.

Treatment.—Symptomatic.

CASTOR OIL.—Same symptoms.

Treatment.—Symptomatic.

CORROSIVE SUBLIMATE.—(*Mercuric chloride.* Fatal dose, 3 grains.)

Symptoms.—Immediate choking, burning pain in epigastrium and esophagus, metallic taste, nausea, vomiting, persistent retching, dysenteric purging, distended tender abdomen, shock, dyspnea, muscle cramps, syncope, stupor, convulsions. Salivation not immediate and sometimes absent. Poison has an acrid coppery taste as compared with arsenic and, in the case of the latter, symptoms are delayed and the stools are less often bloody.

Treatment.—Evacuate stomach, morphia, cardiac stimulants. Give white of egg in milk in quantity or flour paste.

FORMALDEHYDE.—This produces intense abdominal pain, *lachrymation and nasal irritation* (fumes), cyanosis and cardiac failure. The odor of the breath is characteristic.

Treatment.—Evacuation by stomach pump if apomorphia fails, ammonia by inhalation and the same drug (dilute) by the mouth if possible; morphin, cardiac stimulation.

GELSEMIUM.—(Fatal dose, ℥i of fluid extract or ℥iv. of tincture.)

Symptoms.—Extreme muscular weakness. *Oculo-motor paralyses* chiefly ptosis, *blurred vision*, fixed and dilated pupils, jaw-drop, face pallid and congested, livid cold sweat, impaired speech, dyspnea (respiration slow), partial paralysis of extremities, marked cardiac depression, occasionally pharyngeal spasm or general convulsions. *Fatal period*, one to several hours.

Treatment.—Evacuation of stomach, *morphin freely*, cardiac stimulants, hypodermoclysis.

HYDROCYANIC ACID.—(Prussic acid. Fatal dose, about ℥i of official preparation or 1 grain of anhydrous acid.)

Symptoms.—*Immediate in large doses*, vertigo, muscular relaxation (patient falls), protruding eyes, locked jaws, slow gasping respiration, dilated pupils, insensitive conjunctiva, pulse feeble, extremities cold, convulsions may occur, the face is livid and bloated, the lips foamy, the breath carries the distinctive odor of bitter almonds and death occurs in coma with *stertorous breathing* or convulsions.

Fatal Period.—*A few minutes*. *Death* is not instantaneous as is commonly believed.

Treatment is usually futile, impossible or unnecessary, the patient being dying, dead, or out of danger in a few moments.

LEAD ACETATE.—(Maximum fatal dose, about 1 ounce.)

Symptoms.—Sweet metallic taste, violent vomiting and a particularly intense abdominal colic with a rigid retracted abdomen, intense thirst and obstinate constipation, or, more rarely, a diarrhea with *black stools*, vertigo, convulsions and stupor or coma. The blue line may be present even in acute poisoning. *Fatal period*, a few hours to several days.

LOBELIA.—(Indian tobacco. Fatal dose, ℥i of leaves.)

Symptoms.—Nausea, vomiting, perhaps purging, cold sweats, contracted pupils, collapse, coma.

Treatment.—Evacuation of stomach. External heat, cardiac stimulants, morphia, tannic acid.

MUSHROOM POISONING.—(Usually *Agaricus muscarius or vernus.*)

Symptoms.—Violent abdominal pain, vomiting, purging, mental and nervous excitement, dyspnea, stertor, collapse.

Treatment.—*Large doses of atropia*,* hypodermoclysis, external heat, cardiac stimulants. Empty stomach if necessary.

*This treatment was first successfully applied by Dr. Jacob E. Schadle, of St. Paul, who in a personal communication states that the dose should be 1/100 grain repeated as necessary and that ½ grain may be required during twenty-four hours.

OXALIC ACID.—(Frequently taken for epsom salts.) *Fatal dose,* ℥iv.

Symptoms. (Immediate).—Violent vomiting often bloody, or acute pain in head, neck, back and limbs with tingling or numbness, tetanic spasms or convulsions, aphonia and marked cardiac weakness or collapse. Usually fatal within one hour, perhaps in a few minutes.

Treatment.—*Never give potassium or sodium salts,* but rather chalk and magnesia in milk, oils, lime-water, morphin and cardiac stimulants.

OPIUM.—(Fatal dose 4 to 5 grains; laudanum ℥i, or their equivalents. In children trifling doses have caused death. Habitues may use several hundred grains daily.)

Symptoms.—(Usually within from three to thirty minutes.) Somnolence, vertigo, stupor, coma, according to dose, the *pupils are contracted* ("pin-point") and almost invariably *equal,* pulse slow and full, later weak and compressible. Respiration slow and stertorous, skin at first flushed and warm, later clammy, with diminishing respiratory rate. *Pupils may dilate* as death approaches. The fact that contracted pupils may occur in pontine hemorrhages and uremic poisoning (Reese) must not be forgotten.

Duration of Fatal Period.—*Seven to twelve hours.*

Treatment.—Evacuate stomach (apomorphia 1 to 10 grains, stomach pump, etc.). Hypodermic injections of atropin, administer strong coffee; exercise mild cases and dash cold water over chest to excite respiration. *Use artificial respiration* and cardiac stimulants. *Administer* 10 *grains of potassium permanganate in* 6 *to* 8 *ounces of water. Empty bladder, use hypodermoclysis.* In doubtful cases note odor of breath (laudanum) and search for hypodermic punctures, old or recent. Cocain and adrenalin have been recommended and repeated lavage is valuable. Death occurs from respiratory failure indicated by increasing lividity and irregularity and slowness of respiration.

PHOSPHORUS.—(Fatal dose, 1 to 10 grains have killed.)

Symptoms.—(Usually delayed several hours.) "Garlicky" breath, nausea, violent vomiting, abdominal pain, either diarrhea or constipation, dilated pupils, cardiac depression. Vomited matter is green and is luminous in the dark; in protracted cases jaundice and hemorrhages from mucous membranes (especially hematemesis), or under skin. Urine scant and albuminous or suppressed.

Treatment.—*Ordinary oils must not be used.* Evacuate stomach by use of copper sulphate, purge freely and repeatedly; give *old* oil of turpentine in emulsion, albuminous and mucilaginous drinks (barley water, milk, flaxseed tea, egg albumin, etc.).*

POTASSIUM NITRATE.—(Niter, saltpeter.)

Symptoms.—Same as caustic alkalies, but less marked, with tremor and perhaps convulsion and delirium.

Treatment.—*Evacuate stomach* and give milk, barley water, flaxseed tea, cardiac stimulants, and morphia.

*H. C. Wood states that American oil of turpentine is valueless in this condition. Crude French oil is undoubtedly best.

POTASSIUM CHLORATE.

Symptoms.—Vomiting, purging, asthenia, delirium, spasm or convulsions, acute nephritis.

Treatment.—Same as preceding.

STRAMONIUM AND HYOSCYAMUS.—Same as belladonna.

STRYCHNIN (Nux vomica).—Fatal dose, ½ grain.

Symptoms (After fifteen to thirty minutes).—*Constriction of the throat or subjective dyspnea*, muscular twitching of the face, followed by jerking of the head and extremities, and violent *tetanic* spasm (usually opisthotonos) associated with lividity. Contraction of the facial muscles produces the "risus sardonicus," there may or may not be locking of the jaw (if so, *it comes on last and relaxes first in the spasms*), and, as in hydrophobia, spasm may be induced by attempts to administer water. Relaxation follows in from one-half to five minutes and there is marked exhaustion and profuse perspiration. After a few minutes the spasm recurs and they increase in frequency and severity until death ensues from exhaustion or asphyxia. The special senses are exquisitely acute and a noise, draught of air, or sudden bright light may induce spasm; vomiting is usually absent. It should be noted as separating this condition from hysteria, that *the eyes are opened and the mind* clear until the last or until the supervention of asphyxia if the case thus terminates. *Unlike trismus* proper, the *jaw muscles are involved late* and the convulsions are intermittent with periods of *complete* relaxation. *Fatal period*, five minutes to several hours.

Treatment.—Absolute quiet in a darkened room, evacuation of stomach *after chloroform inhalation has been started*, bromides and chloral, atropin.

TARTAR EMETIC.—(Fatal dose, ½ to 40 grains.)

Symptoms.—Almost exactly as in arsenic poisoning with profound cardiac depression.

Treatment.—Same as arsenic, save that tannic acid, not iron, is the antidote (strong green tea, etc.). *Morphia* and cardiac stimulants are indicated.

TARTARIC ACID.—(One ounce has proved fatal.)

Symptoms.—Violent gastrointestinal irritation.

Treatment.—Sodium bicarbonate, magnesia, chalk, best administered through stomach tube which permits emptying stomach of contents and generated gas.

TABLE OF (APPROXIMATE) METRIC EQUIVALENTS

Grains or Minims	Grams or (c.c.)	Grains or Minims	Grams or (c.c.)
1/200	=0.0003	10	= 0.6
1/100	=0.0006	12	= 0.8
1/64	=0.001	15	= 1.0
1/32	=0.002	20	= 1.2
1/16	=0.004	30	= 2.0
1/12	=0.005	60	= 4.0
1/8	=0.008	120	= 8.0
1/6	=0.01	240	= 15.0
1/4	=0.015	480	= 30.0
1/3	=0.02	Ounces	
1	=0.06	2	= 60.0
2	=0.12	4	=115.0
3	=0.2	6	=170.0
4	=0.25	8	=230.0
5	=0.3	10	=280.0
6	=0.4	15	=420.0
8	=0.5	20	=568.0

CENTIGRADE AND FAHRENHEIT SCALES

To convert Fahrenheit into Centigrade, subtract 32, multiply the remainder by 5, and divide the result by 9.

To convert Centigrade into Fahrenheit, multiply by 9, divide by 5, and add 32.

The following table shows the relation of degrees Fahrenheit to Centigrade.

Centigrade	Fahrenheit	Centigrade	Fahrenheit	Centigrade	Fahrenheit	Centigrade	Fahrenheit
110	230	45.0	113.0	37.0	98.6	15	59.0
100	212	44.0	111.2	36.5	97.7	10	50.0
95	203	43.0	109.4	36.0	96.8	+ 5	41.0
90	194	42.0	107.6	35.5	95.9	0	32.0
85	185	41.0	105.8	35.0	95.0	− 5	23.0
80	176	40.5	104.9	34.0	93.2	−10	14.0
75	167	40.0	104.0	33.0	91.4	−15	+5.0
70	158	39.5	103.1	32.0	89.6	−20	−4.0
65	149	39.0	102.2	31.0	87.8	−17.8	
60	140	38.5	101.3	30.0	86.0		
55	131	38.0	100.4	25.0	77.0		
50	122	37.5	99.5	20.0	68.0		

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W

Weakness. (*See* Exhaustion.)
Word blindness. (*See* Blindness.)

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